Atlas of Head Neck and Skull-base Surgery

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ISBN 978-981-99-6131-3 ISBN 978-981-99-6132-0 (eBook)
https://doi.org/10.1007/978-981-99-6132-0

To my wife Anu who has been by my side throughout our life journey, whose love, endless support and encouragement have made it all possible
To my son Rishabh, pillar of my strength, for his unconditional love and support
To my teachers and colleagues, for their faith and encouragement
To our patients and their families, for their trust at all times
NMN

To my parents and brother Hitesh for their unending love
To my husband, Harshal, and kids, Harshit and Rupansh, for their immense support and understanding
To my teachers, patients and God Almighty, for constant blessings
RM

To my parents, Mr. Tejveer Singh and Mrs. Gayatri Singh, and brothers Durgesh Singh and Lokesh Jadon, whose constant support and guidance have been invaluable to me
To my loving wife, Deepa Singh, and son Reyansh Singh, who have been my source of inspiration and motivation throughout this journey
To my teachers, patients and God Almighty, for constant blessings
AS

To my pillar of strength, my loving wife, Sumedha
My source of inspiration, Appa and Amma
My supporting brother Kaushik
My ever-encouraging father-in-law and mother-in-law
KNR

To my ever-loving wife, Mona Yadav, and daughter, Nitara, for their endless love and encouragement
To my parents, Shrirang Dange and Vedavathi K.L., who gave me everything and always seem to find ways to give more; to my brother Preetham for his support and to my father-in-law and mother-in-law, Jaswant Singh and Dinesh Yadav, for their unfaltering encouragement
PSD

Foreword

The head and neck region is one of the most complex anatomical structures in the human body. Being located between the heart and the brain, and incorporating the upper aerodigestive tracts, it is also the origin of various pathological processes of neoplastic, infectious or congenital origin. The head and skull base were also among the last areas to be explored surgically. Throughout the past decade, various treatments were developed for the management of head and neck pathologies. The welcome infusion of new tools including minimally invasive and robotic techniques has enabled safer and les morbid surgical approaches to this region. To improve the outcome of patients, microvascular techniques were developed which significantly improved the quality of life of patients. Hence, surgeons need a comprehensive easy-to-use atlas that serves as a manual for the management of such patients.

Head and neck surgery is an intricate and demanding medical speciality that requires a comprehensive understanding of human anatomy and physiology, as well as the technical skills and dexterity necessary to perform complex procedures with precision and care. As surgical techniques continue to advance, it is crucial for practitioners to stay up to date with the latest developments and procedures.

Atlas of Head Neck and Skull Base Surgery uses a case-based approach to highlight the commonly encountered cases to complex cases, making it an easy and practical resource. It includes various sections, each focusing on a different aspect of surgery. Each section covers general principles, including patient evaluation, relevant preoperative investigations, surgical techniques and post-operative care. The various sections discuss surgery of the cutaneous malignancies of the head and neck, thyroid gland, parathyroid gland, salivary glands, larynx and airway, oral cavity, nose and paranasal sinuses, skull base lesions and neurogenic tumours of the neck.

This Atlas serves as an intraoperative step-by-step guide of the various surgical approaches to the head and neck, without requiring prior knowledge in the field. It is meant to be accessible to residents, fellows and specialists in various disciplines, including otolaryngology, head and neck surgery, neurosurgery, plastic and reconstructive surgery, maxillofacial surgery and oncology. Throughout, the emphasis is on the vision of interdisciplinary education including the preoperative workup, surgical technique, post-operative complications and follow-up.

The Atlas is organized by anatomical region, with each section covering a range of procedures commonly performed in that area. The procedures are

presented in a self-explained format, with detailed descriptions of each step accompanied by high-quality photographs to help readers visualize the surgical anatomy and techniques involved.

The Atlas is written by one of the renowned head and neck surgeon professors (Dr.) Nitin M Nagarkar and his efficient team of surgeons. Professor (Dr.) Nitin M Nagarkar is among the most experienced head and neck surgeons in the world, and through this Atlas, he aims to provide a comprehensive resource material for head and neck surgical education and practice. The authors also present and discuss the cases of varying complexities throughout, introducing all the major ideas that a physician involved in treating these patients would be expected to know. This book contains not only the genesis and pathogenesis of head and neck diseases, but also what all surgeons need to bring about successful resolution of disease, with the return of normal function.

I trust that this Atlas will serve as a valuable reference for those seeking to enter or expand their knowledge and skills in surgical practice.

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Preface

It is with great pleasure that we present to you our book "Atlas of Head Neck and Skull base Surgery: A Case-Based Approach". This project has been a long-standing dream of ours, and we are excited to share it with you.

The field of head, neck and skull base surgery is constantly evolving, and it is essential for surgeons to keep abreast with the latest evidence and techniques. This Atlas is designed to provide a comprehensive overview of the various surgical procedures used in this speciality, including both ablative and reconstructive approaches.

Our goal in creating this Atlas was to provide a resource that is both informative and accessible, which can help learners at all levels to gain a deeper understanding of this subject.

The book is divided into several sections, each focusing on a different aspect of head, neck and skull base surgery. Each section covers general principles, including patient evaluation, surgical technique and post-operative care. The sections discuss surgery of the cutaneous lesions of the head and neck, thyroid gland, parathyroid glands, salivary glands, larynx and airway, oral cavity, nose and paranasal sinuses, skull base lesions and neurogenic tumours of the neck. The final section covers the reconstructive approaches to head and neck defects, principles of adjuvant therapy and transoral robotic surgery.

With a focus on multidisciplinary treatment, our Atlas showcases the latest evidence-based practices and patient management techniques that are critical for achieving the best possible outcomes. Pertinent high-quality references have been cited in the manuscript. Each case is presented in a clear, concise manner, with detailed descriptions of the surgical techniques employed and the rationale behind them. Additionally, the book features state-of-the-art management and follow-up strategies that are designed to optimize patient outcomes and improve quality of life.

Our Atlas is a culmination of years of clinical experience and research. It includes exhaustive illustrations, diagrams and photographs to aid our readers to visualize and understand the complex nature of head and neck.

We hope that this Atlas will be useful for residents, fellows and practitioners in the field of head, neck and skull base surgery. It is our goal to educate and inspire the next generation of head and neck surgeons to continue pushing the boundaries of what is possible in this exciting and rapidly evolving speciality.

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Acknowledgements

Dear all,

I would like to take this opportunity to express my gratitude and appreciation to all those who have helped in envisioning the book "Atlas of Head Neck and Skull Base Surgery: A Case-Based Approach". Your invaluable support, guidance and insights have been instrumental in making this book a reality.

I would like to express my appreciation for Dr. Amit Kumar Chowhan and Dr. Nighat Hussain, who have been instrumental in providing an in-depth understanding of the various pathological conditions encountered in head, neck and skull base surgeries.

I would also like to extend my heartfelt thanks to Dr. Jiten Kumar Mishra and Dr. Shamendra Sahu for their expertise in reconstructing complex head and neck surgical defects. Their exceptional knowledge of the applied anatomy and attention to detail, with a focus on functional outcome, have been invaluable in understanding the reconstructive aspects of head, neck and skull base surgeries.

I would like to extend my sincere thanks to Dr. Mudalsha Ravina, whose expertise and insights have been necessary for understanding the nuances of nuclear medicine imaging and its role in managing head, neck and skull base surgical cases.

I would also like to thank Dr. Siddhartha Nanda, whose inputs have been instrumental in understanding the role of radiotherapy in head and neck malignancies, especially the latest techniques.

I am extremely grateful to Dr. Amit Agarwal for his valuable inputs related to medical oncology.

Dr. Nandkishore Agarwal's support from the Department of Anaesthesiology and Critical Care has been very vital for the anaesthetic and ICU management of head-neck and skull base surgeries.

I would also like to extend my thanks to Dr. Narendra Kuber Bodhey and Dr. Saroj Kumar Pati for their essential inputs to help us understand the radiological aspects of head-neck and skull base surgery cases.

The enthusiastic approach of Dr. Anil Kumar from the Department of Neurosurgery has been very useful in understanding the nuances of neurosurgery in skull base surgeries.

Dr. Nitin Kashyap's expertise has been crucial for understanding the intricate anatomical details related to cardiothoracic and vascular surgeries in relation to our speciality.

I am greatly indebted to Dr. Anu N. Nagarkar for her continuous encouragement and meticulous proofreading of the document.

I would also like to express my gratitude to all our faculty members in the Department of ENT and Head-Neck Surgery: Dr. Ripudaman Arora, Dr. Satish Satpute and Dr. Sharmistha Chakravarty, as also the senior and junior residents of the team and the OT and OPD nursing and ancillary staff at AIIMS Raipur, without whose support and dedication, this book would not have been possible.

I am deeply indebted to all those who have supported us in this endeavour.

Sincerely,

Nitin M Nagarkar

Contents

Introduction to Head and Neck Cancers

Nitin M Nagarkar, Rupa Mehta, and Prajwal S Dange

1 Introduction

'Head and neck cancer' is the term used to describe several different malignant tumours that arise from the mucosa of the upper aerodigestive tract, including the oral cavity, pharynx, larynx, nasal cavity and sinuses. It also includes neoplasms originating from the salivary glands, thyroid parathyroid glands, soft tissue, bone and skin in the head and neck region.

It encompasses the surgery of head and neck cancers involving the spectrum of variable pathology, ranging from a verrucous lesion, which is clinically indolent, to the most sinister neoplasm, like anaplastic thyroid cancer.

The most common malignant neoplasms of the head and neck are squamous cell carcinoma in the mucosal surface of the aerodigestive tract and papillary thyroid carcinoma in the thyroid gland. Salivary gland tumours, soft tissue and bone sarcomas are relatively infrequent.

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Risk factors include tobacco, various forms of tobacco, including smokeless tobacco and betel-quid chewing, alcohol, poor oral hygiene, nutrient-deficient diet and sustained viral infections, i.e. human papillomavirus (HPV). Chronic exposure of the upper aerodigestive tract to these carcinogenic factors can result in dysplastic or premalignant lesions in the mucosa and ultimately progress to cancer. The relative prevalence of these risk factors contributes to the variations in the observed distribution of head and neck cancers in different areas of the world.

Surgery has been the mainstay of therapy in head and neck neoplasms for more than a century. With the introduction of radiation in the latter half of the twentieth century, it became an important modality used independently or in combination with surgery. Although initially chemotherapy was used primarily with palliative intent, it is now used as part of curative treatment approaches when combined with radiation. Similarly, biological or targeted agents are evolving to become part of the standard therapy. Accordingly, understanding and implementing multidisciplinary management strategies are cornerstones for achieving optimal therapeutic outcomes.

1.1 History of Head and Neck Cancers and Neck Dissection

- Some of the earliest attempts at head and neck surgery can be credited to Egyptian physicians who attempted ablative and reconstructive

N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_1

procedures of the oral cavity and lip. The *Edwin Smith Papyrus*, the origins of which are dated to approximately 3000 BC, contains some of the first descriptions of surgical management of mandibular and nasal fractures and lip tumours.

- The first documented efforts of reconstructive head and neck surgery are found in the Sanskrit texts of ancient India written in *Sushruta Samhita* by Sushruta approximately 2600 years ago.
- Sushruta, regarded as the 'father of Indian surgery', described various surgical techniques for reconstructing head and neck defects. Considerable controversy exists over the period of his contributions, with dates ranging from 600 BC to 1000 AD. He contributed to many fields of medicine, but he is said to have laid the foundations for a variety of pedicled and rotation flaps, and he was the pioneer of reconstructive nasal surgery and has described more than 15 methods of nasal reconstruction, which are similar to many of the techniques utilized in the nineteenth and twentieth centuries.
- Aulus Cornelius Celsus, considered to be the greatest of the Roman medical authors and surgeons, also described a variety of techniques similar to those practised in India in his medical text of the first century, *De Medicina*, and is credited with one of the first head and neck cancer procedures describing excision of a lip malignancy [1].
- There was a significant decline in all surgical advances in the middle ages.
- The period of Renaissance in the fourteenth century signalled a rebirth of science, medicine and the world of surgery.
- Tagliacozzi, in the latter half of the sixteenth century, described and refined the use of distant pedicled flaps for various head and neck reconstructions [2].
- The modern era was heralded by the development of the achromatic microscope, which allowed pathologists to view tissues first under magnification.
- In 1835, Mirault [3] and Langenbeck described wedge excision of the tongue with ligation of the lingual artery to control bleeding, a significant advance in reducing the bleeding associated with these procedures.
- The procedure for access to the oral cavity was first described in 1839 by Roux. He was the first to describe lip-splitting incisions combined with a mandibular osteotomy.
- The mid-nineteenth century was dominated by developments in the pathologic description of tumours, including those by the father of modern oncologic pathology, Virchow.
- Gordon Buck from New York was the first to describe the laryngofissure approach to remove laryngeal tumours in 1851.
- The famous Viennese surgeon, Theodor Billroth, introduced techniques of bilateral mandibular osteotomy for oral access in 1862 and described the first total laryngectomy in 1873.
- In the latter part of the century, Kocher described the technique of lateral mandibular osteotomy along with the first description of the importance of neck node management in mucosal tumours of the head and neck [4].
- The early twentieth century was dominated by developing knowledge of the head and neck lymphatics and improvements in surgical technique.
- Polya, in 1902, described the lymphatic drainage of the oral cavity, demonstrating that 50% of the lymphatics traversed the mandibular periosteum leading to an interest in en-bloc resections and the foundations of the original composite resection for oral cancer.
- In 1906, the American surgeon George Crile described his approach to head and neck tumours, becoming the foundation of the present-day radical and functional techniques for neck dissection [5].
- In 1913, Gluck and Soerensen described improved approaches to creating tracheostome and repairing the pharynx in laryngectomy. The approaches by Gluck and Soerensen arguably became the foundation of modern laryngopharyngeal ablative and reconstructive surgeries [6].
- In 1932, GL Semken described radical neck dissection and en-bloc resection of the tongue and the associated lymphatic structures.
- In 1932, Ward performed and described the first composite resection.

- A significant innovation for head and neck malignancy management was described in 1934 by Hayes Martin and Ellis as the use of fine needle aspiration cytology as a diagnostic tool; this dramatically altered the treatment of head and neck malignancy and thyroid disease [7].
- The 1930s and 1940s were dominated by attempts at treating head and neck tumours with radiotherapy. A renaissance of interest in surgical approaches to head and neck diseases occurred in the late 1940s and 1950s as the early and late effects of this primitive form of radiation became evident.
- The 1940s and 1950s were dominated by the development of surgical techniques and an increasing interest in organ-preserving surgical procedures.
- Gluck and Portmann described the technique of vertical hemilaryngectomy to extirpate small-volume laryngeal lesions and reported a large series of patients with successful outcomes [8].
- In 1951, Alonso from Uruguay described vertical and horizontal supraglottic laryngectomy techniques [9].
- In 1951, Hayes Martin published his seminal work on head and neck surgery, describing his techniques and outcomes for patients over the previous three decades at Memorial Sloan Kettering [10].
- In 1952, Conley and Pack extended concepts of vascular surgery to neck tumours, describing approaches to vascular tumours and malignancies involving the carotid [11].
- Management of neck disease evolved in the 1960s and 1970s following Soares' descriptions of selective neck dissection in South America and Bocca in Italy [12, 13].
- The current approaches to neck dissection arose from refinements of techniques popularised by these authors and provided an evidence basis from work done by Shah, Byers, Medina and others.
- The 1960s, 1970s and 1980s were dominated academically by giants in the field who changed the face of head and neck surgery through their enormous contributions to the evidence base of head and neck surgery. Their fellowship training programmes extended their skills to other countries and provided the academic foundation for the next generation of surgical leaders. A list of individuals of this era would include, but not be limited to, Dr. Hayes Martin (USA), Dr. John Conley (USA), Philip Stell (UK), Arnold Maran (UK), Joseph Ogura (USA), Dr. Douglas Bryce (Canada), Dr. M Lederman (UK), Sir Donald Harrison (UK) and Dr. Richard Jesse (USA).
- The primary surgical innovations of this era were the increasing interest in minimally invasive surgery of the larynx, endoscopic endonasal and skull base surgery. Jako and colleagues from the United States and Kleinsasser from Germany influenced the developments in minimally invasive surgery of the larynx.
- Most notable among these was Dr. Wolfgang Steiner [14] and his colleagues from Germany, whose systematic approach to the endoscopic laser excision of laryngeal tumours has changed the management approach to early and advanced laryngeal malignancy.
- In the early 1980s, Messerklinger, Stammberger [15] and colleagues from Austria introduced the concept of functional endoscopic sinus surgery, providing the technical foundation for minimally invasive nasal surgery development.
- Several groups worldwide, most notably Kassam, Carrau and Snyderman [16] from the United States, have extended these concepts and techniques, developing transnasal approaches to managing skull base tumours.
- The last two decades have seen an expanded interest in multimodality therapy, combining surgery and radiotherapy or chemotherapy and radiation, with surgery reserved for salvage. This approach has evolved from surgeons becoming increasingly involved in clinical trials and their interest in developing an evidentiary basis for the treatments they offer.
- The most prominent of these trials have been the laryngeal organ preservation trials in the United States [17] and the evolution of clinical trials evaluating the role of chemotherapy, radiation therapy, molecular targeted therapy and surgery in the United States and Europe.

1.2 Sites of Head and Neck Cancers

- Lip and oral cavity cancer
- Oropharyngeal cancer
- Nasopharyngeal cancer
- Laryngeal cancer
- Hypopharyngeal cancer
- Thyroid and parathyroid cancer
- Paranasal sinus and nasal cavity cancer
- Salivary gland cancer
- Soft tissue sarcoma

1.3 The Burden of Head and Neck Cancers

Worldwide, head and neck cancers account for approximately 900,000 cases and over 400,000 deaths annually [18].

Males are affected significantly more than females, with a ratio ranging from 2:1 to 4:1. The incidence rate in males exceeds 20 per 100,000 in regions of France, Hong Kong, the Indian subcontinent, Central and Eastern Europe, Spain, Italy, Brazil and among African Americans in the United States. Buccal mucosa/gingivolabial sulcus and tongue cancers are more common in the Indian subcontinent, nasopharyngeal cancer is more common in Hong Kong and pharyngeal and/or laryngeal cancers are more common in other populations; these factors contribute disproportionately to the overall cancer burden in these Asian countries [19, 20].

According to the GLOBOCAN 2020 data, the number of new cases of head and neck cancer in 2020 was 1,518,133, and Table 1 shows the distribution of all the cases according to various sites. Figure 1 shows estimated age-standardized incidence rates (world) in 2020 for both sexes and all ages [18].

Table 1 Incidence of various head and neck cancers around the world (source: https://gco.iarc.fr/today/) [18]

Cancer	Number of new cases	Incidence/100,000
Thyroid	586,202	6.6
Lip, oral cavity	377,713	4.1
Larynx	184,615	2
Nasopharynx	133,354	1.5
Oropharynx	98,412	1.1
Hypopharynx	84,254	0.91
Salivary glands	53,583	0.57

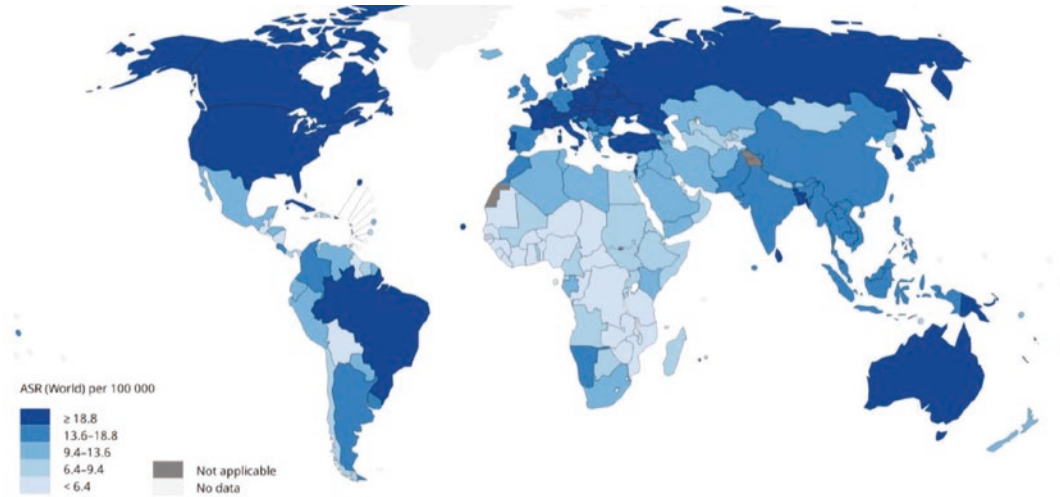

Fig. 1 Estimated age-standardized incidence rates (world) in 2020 for both sexes and all ages (source: https://gco.iarc.fr/today/)

According to the GLOBOCAN 2020 data, the estimated age-standardized incidence rate of all head and neck cancer in the Indian subcontinent is 13.6–18.8 per 100,000 [18].

The total number of new cases of cancer detected in India during 2020 is 1,324,431. Among them, 135,929 (10.3%) were lip and oral cavity cancers in both sexes and all ages (Fig. 2), making lip and oral cavity the second most common cancer in India, next to breast cancer. Among males, lip and oral cancers are the most common contributing up to 16.2% of all cancers (Fig. 3), and in females, lip and oral cancers are the fourth most common contributing up to 4.6% of all cancer cases (Fig. 4) [18].

Globally, oral cancer is the sixth most common type of cancer. India contributes almost one-third of the total burden and the second-highest number of oral cancer cases [21].

According to the National Cancer Registry Programme (NCRP) reports based on 27 Population-Based Cancer Registry (PBCR) [22] in 2014, the incidence of oral cancer in India is as follows.

Tongue cancer—The age-adjusted rate (AAR) of tongue cancer in males is highest in the East

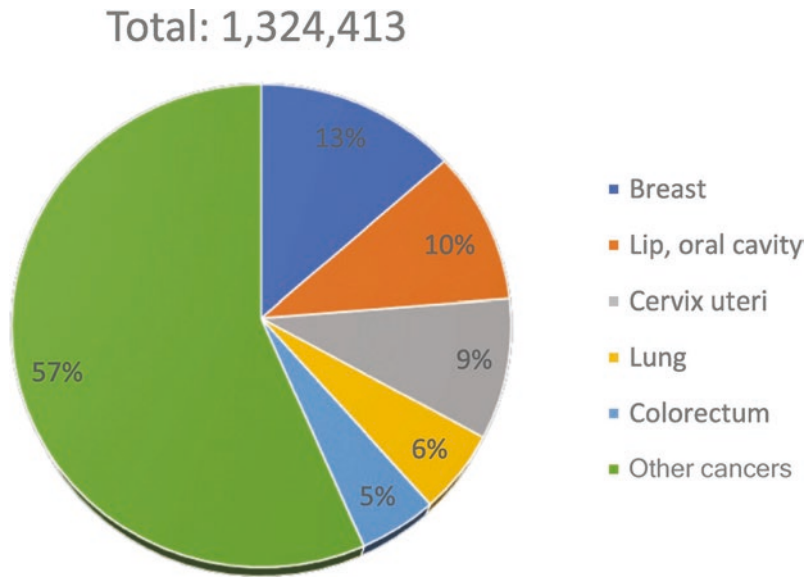

Fig. 2 Number of new cases in India in 2020, both sexes, all ages (source: https://gco.iarc.fr)

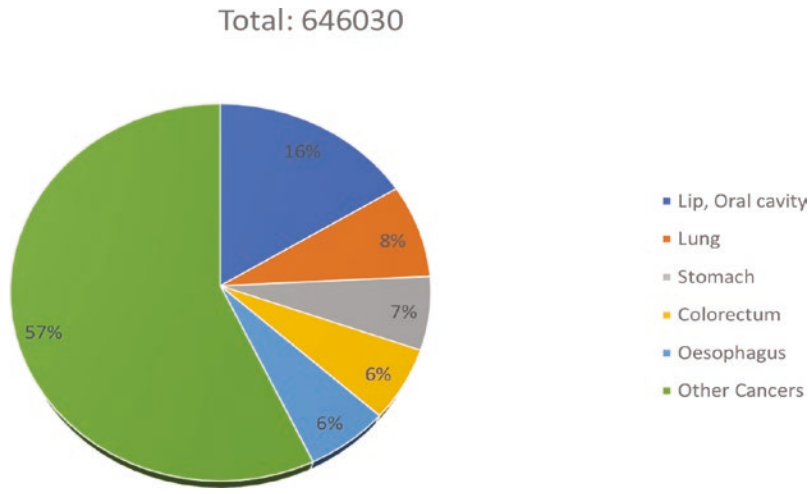

Fig. 3 Number of new cases in India in 2020, males of all ages (source: https://gco.iarc.fr)

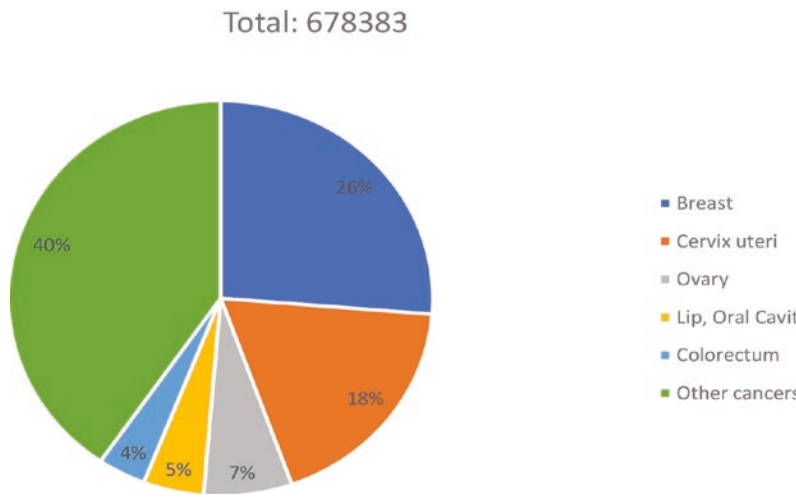

Fig. 4 Number of new cases in India in 2020, females of all ages (source: https://gco.iarc.fr)

Khasi hills district from Meghalaya, which is 11.7 per 100,000 population and the second highest in Ahmedabad, which is 10.4 per 100,000. In females, tongue cancer has the highest incidence in Bhopal, 3.7 per 100,000.

Mouth cancer—AAR of mouth cancer in males is highest in Ahmedabad, 18.7 per 100,000 population, and second highest in Bhopal—14.3 per 100,000. In females, mouth cancer incidence is highest in the East Khasi hills district from Meghalaya—at 9.1 per 100,000.

Tobacco consumption has been the predominant factor causing oral cancer. The continual use of tobacco in various forms, such as gutka, zarda, mawa, kharra, khaini, cigarettes, bidi, hookah, etc., is a significant cause of tumour development in the oral cavity in both young as well as adult Indian populations [23].

Epidemiologically, Kerala has the lowest incidence of oral cancer in India, while West Bengal reports the highest [21].

In India in 2020, lip and oral cavity cancers were accountable for 75,290 cancer-related deaths, up to 8.8% of all deaths related to cancer [18].

1.4 Strategy for Prevention

Among the head and neck cancers, oral cancer is one of the most preventable cancers, and screening is possible as it is an easily accessible site for early detection. The ever-increasing numbers due to continued tobacco consumption, varied disease patterns and lack of awareness bring the patient to the medical facility at an advanced stage.

Oral cancers were the most common head and neck cancers in patients below 40 years of age. In contrast, carcinoma in the oropharynx and larynx were more common in patients above 40 years of age group. Also, there have been studies correlating tobacco chewing (smokeless) to mouth cancers (40%); in smokers, the most common sites were the larynx and hypopharynx.

According to the NCI, the 5-year survival rates for oral cavity and pharynx cancers are as follows:

- 83% for localized cancer
- 64% for cancer that has spread to nearby lymph nodes
- Overall, 60% of all people with oral cancer will survive for 5 years or more. The earlier the stage at diagnosis, the higher the chance of survival after treatment. The 5-year overall survival rate in those with stage 1 and 2 oral cancers is typically 70 to 90%.

This makes timely diagnosis and treatment all the more important; understanding the landscape of head and neck cancer will empower head and neck oncosurgeons to play a critical role among patients and societal education regarding the

importance of addressing modifiable risk factors and continuing to play an essential role in the diagnosis and management of head and neck cancer.

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Pathology of Head and Neck Cancers

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1 Head and Neck Pathology

1.1 Introduction

The head and neck area is complex, with the skin, soft tissue, upper airways and other organs making up its anatomy. The pathology of this area is diverse, requiring clinicopathological correlation for diagnosis. Sometimes, a diagnosis can be made through a patient's history and exam, but pathology is the gold standard, especially for malignant diseases. Tumour grade, type and stage are prognostic indicators and may determine whether adjuvant therapy is needed after surgery. Molecular pathology has enabled a more individualized treatment.

1.2 Pathological Evaluation

There are several methods for acquiring samples for pathological analysis.

The following list includes some of the usual methods.

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1.2.1 Fine-Needle Aspiration Biopsy

Fine-needle aspiration biopsy (FNAB)/cytology is the primary study for patients who report cervical lymphadenopathy or a lesion in the major salivary glands or thyroid gland [1].

Advantages

- Simple method
- Inexpensive
- Highly accurate in distinguishing between benign and malignant lesions

A negative FNAB test does not rule out illness, and a 'triple evaluation' consisting of clinical, imaging and cytopathology is necessary to diagnose correctly.

The Most Frequently Used Stains in FNAB

- Haematoxylin and eosin (H&E)
- Papanicolaou
- Romanowsky
- May-Grunwald-Giemsa (MGG)

1.2.2 Core Biopsy

A core biopsy is similar to FNAC because the biopsy needle used during a core biopsy is larger; it will produce a cylinder of tissue that may be subjected to histological investigation. The tissue volume removed and the maintained tissue architecture improve the diagnostic value. There is also a chance that the procedure will produce enough tissue for histochemistry.

N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_2

Benefits of a Core Biopsy

- Simple and affordable
- Greater diagnostic yield than FNAC and histological examination of the sample
- Higher likelihood of lymphoma subclassification

1.2.3 Incisional Biopsy

It involves removing a wedge-shaped sample typical of the lesion for histological investigation. This method is employed for more easily accessible tumours affecting the larynx, pharynx, oral cavity and hypopharynx.

Incisional Biopsy Benefits

- Able to deliver a conclusive histopathological diagnosis

1.2.4 Excisional Biopsy

It involves the whole lesion being removed, offering a firm histological diagnosis. This might include anything from a major en-bloc resection to the excision of the vocal cord polyp.

Excisional Biopsy Benefits

- Can provide definitive histological diagnosis
- Can be the definitive treatment for the tumour

1.2.5 Sentinel Lymph Node Biopsy

Patients with clinically T1/T2 N0 oral cavity squamous cell carcinoma (SCC) are advised to use this approach [2]. A radioactively tagged colloid tracer is injected into four places around oral cancer before surgery. The first echelon of lymph nodes is where the tracer drains after entering the lymphatic system. The imaging may then be used to map the radioactive lymph nodes. The sentinel lymph nodes are harvested by a tiny incision in the neck after the surgeon utilizes gamma radiation detecting equipment to confirm their location. There might be as many as four sentinel nodes. After that, each 'hot node' is processed in the pathology lab using a technique that samples every lymph node at intervals of 125 μm.

To increase the test's sensitivity, cytokeratins are stained using immunohistochemistry (IHC).

Elective neck dissection can be avoided by monitoring the neck after a negative sentinel lymph node biopsy, which has a high negative predictive value per cent.

A neck dissection must be completed in a subsequent procedure if a lymph node is positive.

1.2.6 Histochemistry

The following are the processes involved in tissue preparation before a histopathological investigation:

- In the operating room, tissue samples are treated with 10% formalin.
- Macroscopically describe the specimen, dissect it, and send a sample tissue slice for analysis.
- A tissue slice goes through cycles of dehydration where alcohol replaces the tissue's aqueous fixative, xylene is used for clearing instead of alcohol during infiltration, and molten paraffin is used for clearing during embedding to prepare for microtomy.
- Slices of 3–5 μm are produced using a microtome.
- Usually, the slices can be stained with H&E. Haematoxylin colours tissue glycoproteins light blue and nuclei dark blue. Collagen and the cytoplasm are stained pink by eosin.

Special stains used are:

- Deep purple periodic acid-Schiff (PAS) stain is used to highlight carbohydrates.
- Simple carbohydrate moieties are first removed by the enzyme diastase, leaving mucins present in glandular secretions and fungal hyphae.
- To understand the architecture of the tissue, particularly the blood vessels, use the elastin van Gieson stain; elastin fibres stain black and collagen red.
- Ziehl-Neelsen stain—acid-fast bacilli in tuberculosis.

1.2.7 Immunohistochemistry

Immunohistochemistry (IHC) is a technique used to detect the presence of a specific antigen or epitope by having it attached to a particular antibody. This antibody is then coupled to a label, such as a fluorescent dye or enzy-

matic substrate, that can be seen under a microscope. Monoclonal antibodies are typically preferred because they are more specific than polyclonal antibodies; however, even in the case of monoclonal antibodies, no single antibody is 100% sensitive and specific. Therefore, immunophenotyping is best done with a panel of multiple antibodies, both positive and negative, to ensure the accuracy of the results. Furthermore, using multiple antibodies allows for a more comprehensive analysis, as it is possible to identify several different antigens or epitopes that may be present in a tissue sample.

Antibodies directed against:

- Intermediate filaments like cytokeratins, neurofilament, desmin, and vimentin
- Epithelial markers—BerEP4, epithelial membrane antigen
- Structural proteins—calponin
- Storage granules/products—calcitonin, synaptophysin, chromogranin, thyroglobulin
- Hormone receptors—progesterone, oestrogen
- Nuclear epitopes—p16, thyroid transcription factor-1
- Lympho-reticular epitopes—CD3, CD20, CD79a, etc.
- Cell proliferation—Ki67

1.2.8 In Situ Hybridization

Using the in situ hybridization, a particular nucleic acid segment may be precisely localized inside a histologic slice. A complementary strand of nucleic acid with a reporter molecule attached is used to accomplish this.

ISH probes:

- Double-stranded DNA (dsDNA) probes
- Single-stranded DNA (ssDNA) probes
- RNA probes (riboprobes)
- Synthetic oligonucleotides (PNA, LNA)

It is used in head and neck oncology:

- RNA ISH is used to detect EBV.
- RNA ISH is used to detect kappa and lambda light chains in B lymphocytes.
- DNA ISH can be used to identify low-risk and high-risk human papillomavirus infections.
- Chromosomal abnormalities, such as amplifications, translocations and deletions, can be identified by fluorescence ISH.

1.2.9 Molecular Testing

To create a high-resolution genetic profile of tumours, molecular testing is employed.

The following are some applications of molecular testing in head and neck oncology:

- Molecular tests for early detection of malignancy:
  - Proteomic and serum multiplexed cytokine profiling
  - Saliva genomic and proteomic analysis for early oral SCC detection
- Molecular tests for prognosis in head and neck malignancy:
  - HPV detection as a good prognostic indicator [3].
  - Epidermal growth factor receptor (EGRF) genetic abnormality is a negative prognostic indicator [4].
  - Genomic profiling for genomic profiling for molecular characterization.
  - Epstein-Barr virus detection as a predictive biomarker.
- Molecular tests for prediction of treatment response in squamous cell carcinoma:
  - Expression of ERCC1 as a cisplatin response predictor [5].
  - A sign of radiation sensitivity is hypoxia.
  - Prediction of EGRF inhibitor response.
  - Anti-angiogenic agent reaction forecast [6].

1.2.10 Subsites in the Head and Neck

The head and neck regions are divided into several subsites as outlined in Table 1.

1.3 Tumours of the Head and Neck

According to the origin (histogenesis) and/or differentiation route of the tumour cell, benign and

Table 1 Head and neck cancer sites and subsites

Head and neck site	Subsite
Oral cavity	• Lip – External upper lip (vermilion border) – External lower lip (vermilion border) – Commissures • Floor of mouth • Cheek mucosa • Retromolar trigone • Lower alveolus and gingiva • Anterior 2/3 of the tongue • Mucosa of the upper and lower lips • Upper alveolus and gingiva • Bucco-alveolar sulci, upper and lower • Hard palate
Oropharynx	• Tonsillar fossa • Lateral wall • Tonsil • Vallecula • Posterior wall • Uvula • Inferior surface of soft palate • Tonsillar pillar • Base of tongue
Hypopharynx	• Posterior pharyngeal wall • Postcricoid region • Pyriform fossa/sinus
Larynx	• Supraglottis – Arytenoid – Ventricular bands (false cords) – Infrahyoid epiglottis – Aryepiglottic fold, laryngeal aspect – Suprahyoid epiglottis (including tip, lingual [anterior], and laryngeal surfaces) • Glottis – Anterior commissure – Posterior commissure – Vocal cords • Subglottis
Nasopharynx	• Superior wall • Floor: superior surface of the soft palate • Lateral wall: including the fossa of Rosenmuller • Posterior wall: from the level of the junction of the hard and soft palates to the superior wall
Ear	• External auditory canal • Middle ear • Inner ear
Nose and paranasal sinuses	• Nasal cavity – Septum – Floor – Lateral wall – Vestibule • Maxillary sinus • Ethmoid sinus
Salivary gland	• Parotid gland • Submandibular gland • Sublingual gland • Minor salivary glands
Thyroid gland	
Parathyroid glands	
Neck	• Level I, submental IA and submandibular IB nodes. • Level II, upper jugular nodes divided by spinal accessory nerve into IIA and IIB • Level III, middle jugular nodes • Level IV, lower jugular nodes • Level V, posterior triangle nodes divided by spinal accessory nerve into VA and VB • Level VI, anterior compartment
Skin	

malignant tumours can be distinguished. The benign and malignant tumours that are more frequently seen in clinical practice will be covered in this section.

1.3.1 Benign Tumours

Benign Epithelial Tumours

Squamous Cell Papilloma

A localized benign exophytic growth of the squamous epithelium is known as a squamous papilloma. It can affect the nasopharynx, larynx, sinuses and oral cavity. The proliferation of stratified keratinizing squamous epithelium supported by cores of the fibrovascular connective tissue characterizes its typical microscopic appearance.

Depending on the place and subtype, there may be a single lesion or many:

- Oral squamous papilloma:
  - Squamous cell papilloma or verruca vulgaris
  - Condyloma acuminatum
  - Multifocal epithelial hyperplasia
- Laryngeal papilloma
- Sinonasal papilloma:
  - Exophytic sinonasal papilloma
  - Inverted sinonasal papilloma
  - Oncocytic sinonasal papilloma

Benign Salivary Gland Tumours

Benign epithelial salivary gland tumours encompass a range of different types as per the 2017 WHO Classification of Head and Neck Tumours [7].

These include pleomorphic adenoma, basal cell adenoma, oncocytoma, Warthin's tumour, canalicular adenoma, cystadenoma, myoepithelioma, ductal papillomas, sialadenoma papilliferum, lymphadenoma and sebaceous adenoma.

Pleomorphic Adenoma

The most frequent tumour affecting the salivary glands is a pleomorphic salivary adenoma, often known as a benign mixed tumour or mixed tumour. According to reports, the frequency is between 2.4 and 3.05 per 100,000. About 50% of all salivary gland tumours, 60% of parotid tumours, and 40% of intra-oral minor salivary gland tumours are made up of it [8].

With a peak age of occurrence in the fourth to fifth decade, it is more prevalent in females.

Gross Findings

- A well-defined rounded to oval and irregularly enclosed bulk
- The cut surface that is white-tan, glossy or transparent

Microscopic Findings

- Epithelial glands and ducts
- Chondromyxoid matrix with myoepithelial cells and ductal components incorporated in it
- Spindle, plasmacytoid, epithelioid, stellate or basaloid morphologies of myoepithelial cells
- Myxoid, mucochondroid, hyalinized, osseous and/or fatty mesenchymal stroma

Pathologic Differential Diagnosis

- Benign: basal cell adenoma, myoepithelioma
- Malignant: polymorphous adenocarcinoma, adenoid cystic carcinoma

Warthin's Tumour

Adenolymphoma and papillary cystadenoma lymphomatosum are other names for Warthin's tumour. It is the second most frequent salivary gland tumour [9]. The biphasic tumour is made up of papillary fronds and bilayered oncocytic cells that form cysts and are embedded in a thick, lymph node-like stroma. It is believed to develop from the salivary tissue that has been stuck in intraparotid or periparotid lymph nodes.

With a peak incidence in the sixth to seventh decade, it is more prevalent among white males who have smoked in the past.

Gross Findings

- Brown, yellow or red mass that is circumscribed and firm to cystic

Microscopic Findings

- Oncocytic epithelium comprises two layers, having columnar luminal cells above the cuboidal basal cells
- Many papillary infoldings and cysts
- Dense stroma resembling lymph nodes that may have reactive germinal centres

Immunohistochemical Findings

- The epithelial component reacts with keratin.
- B- and T-cell markers react with the lymphoid component.

Pathologic Differential Diagnosis

- Cystadenoma, sebaceous lymphadenoma and lymph node metastasis

Benign Mesenchymal Tumours

Benign mesenchymal tumours are a group of non-malignant, non-epithelial cell tumours that

arise from mesenchymal cells, which form the connective tissue in the body. Mesenchymal cells are typically located in the bone, cartilage, fat, muscle, fibrous tissue and certain other connective tissues. These tumours behave in a non-aggressive manner and do not usually spread to other parts of the body.

Schwannoma

Neurilemmomas are another name for schwannomas, benign, well-encapsulated tumours from Schwann cells in the sheath surrounding peripheral nerves, from the sympathetic chain; cervical or cranial nerves V, VII, and XII; brachial plexus; or head and neck region.

Gross Findings

- Usually enclosed, whereas those that involve the temporal bone frequently are not.
- The tumour is often connected to a distinguishable nerve and is firm, rubbery and brown to yellow in colour.

Microscopic Findings

- Verocay bodies and tightly backed bundles of spindled cells can be seen in cellular Antoni A regions.
- Areas of Antoni B with hypocellularity and microcystic degeneration.
- Fusiform cells having cytoplasm that is fibrillar.
- Wavy/buckled, pointed and spindly nuclei (often palisaded).
- Hyalinized, medium-sized dilated vessels.

Immunohistochemical Findings

- S100 protein, SOX10 and vimentin responses that are widespread and powerful.
- In degraded regions, there are sometimes positive CD34 fibroblasts for glial fibrillary acidic protein and neuron-specific enolase.

Genetic Studies

- Ninety percent of the mutations in type 2 neurofibromatosis Merlin coded it at 22q12.

Pathologic Differential Diagnosis

- Malignant peripheral nerve sheath tumours, meningiomas, neurofibromas, solitary fibrous tumours, paragangliomas and neurofibromas.

1.3.2 Malignant Tumours

Squamous cell carcinomas, which develop from the epithelial lining of the upper aerodigestive tract, account for the majority (90%) of head and neck malignancies.

Tumour Staging

A technique for expressing the disease's relative severity or extent is used in the staging of head and neck cancers.

The main objectives of staging are:

- To support the doctor in making treatment plans
- To provide some prognostic information
- To aid in assessing the effectiveness of the treatment
- To make it easier for treatment facilities to communicate with one another
- In order to aid in the ongoing study of human cancer

The TNM staging system, created and maintained by AJCC and UICC, is the most commonly used and is kept under review by international expert committees. AJCC and UICC published the eighth edition in 2017 [10].

The TNM staging system's fundamental tenets are:

- Clinical—Based on clinical data, TNM or cTNM (physical examination and imaging).
- Pathological—Based on the cTNM, the pTNM is modified by additional data from the postoperative pathological examination.

Autopsy—When there is no sign of cancer before death and the malignancy is only identified based on the findings of the post-mortem examination, aTNM is utilized.

There may also be other TNM descriptors employed on occasion, such as:

- The 'y' prefix—when classification is done during or after the first multimodality therapy, ycTNM or ypTNM
- The 'm' suffix—when there are multiple primary at a single site, pT(m)NM
- The 'y' suffix—when there is only one primary at a single site, pT(m)NM

Typically, tumours are histologically subtyped based on how differentiated they are components:

- Gx—grade cannot be assessed.
- G1—well-differentiated.
- G2—moderately differentiated.
- G3—poorly differentiated.

The presence or absence of residual tumour is classified as:

- Rx—the presence of residual tumour cannot be assessed.
- R0—no residual tumour is present.
- R1—microscopic residual disease.
- R2—macroscopic residual tumour.

The TNM approach is built on three elements to describe the anatomical extent of the illness:

- T—the initial tumour's size
- N—regional lymph node metastases: the presence, absence and extent
- M—whether there are any distant metastases or not

Method of Staging

Regional Lymph Nodes

Prognostic factors for head and neck cancers include the condition of local lymph nodes. As shown in Table 2, lymph nodes are now classified into seven tiers and distinct anatomical places.

For the majority of head and neck locations (oral cavity, p16-negative pharyngeal carcinoma, larynx carcinoma, sinus carcinoma and salivary gland cancer), the criteria of the N categories are the same and are as stated in Table 3.

Table 2 Nomenclature of anatomical site of lymph node

Nomenclature of anatomical site of the lymph node	
Level I	Consists of the submental and submandibular triangles, separated by the digastric muscle's posterior belly, the hyoid bone and the mandibular body, respectively
Level II	Extends from the level of the hyoid bone inferiorly to the base of the skull superiorly, containing the upper jugular lymph nodes
Level III	Contains the middle jugular lymph nodes that run inferiorly from the cricothyroid membrane to the hyoid bone in the superior position
Level IV	Consists of the lymph nodes in the lower jugular region, which run from the clavicle to the superior cricothyroid membrane
Level V	Contains the lymph nodes of the posterior triangle, which are bordered by the clavicle inferiorly, the anterior border of the trapezius and the posterior border of the sternocleidomastoid muscle
Level VI	Contains the anterior compartment lymph nodes from the suprasternal notch inferior to the hyoid bone superior. The lateral boundary is formed on each side by the carotid sheath's medial border
Level VII	Contains the lymph nodes in the upper mediastinum below the suprasternal notch

As seen in Tables 4 and 5, the most recent version has also added two distinct staging systems (cTNM and pTNM) for neck metastases of human papillomavirus (p16-positive) oropharyngeal carcinoma.

The influence of treatment response on prognosis varies, which justifies a separate N categorization for thyroid and well-differentiated nasopharynx tumours. According to Tables 6 and 7, it is as stated.

Primary Tumour

Lip and Oral Cavity

The oral cavity extends from the line of the circumvallate papillae below anteriorly to the junction of the hard and soft palate above and to the junction of the skin-vermilion of the lips posteriorly. Table 1 lists the anatomical locations and subsites. Table 8 provides the TNM clinical classification for the lip and oral cavity and the T—

Table 3 Clinical N staging of regional neck nodes in head and neck cancers

Clinical N stage of regional neck lymph nodes (LN)	
NX	Regional LN cannot be assessed
N0	No regional LN metastasis
N1	Single ipsilateral LN metastasis, ≤3 cm in the greatest dimension without extra-nodal extension (ENE)
N2	N2a—Single ipsilateral LN metastasis, > 3 cm and ≤6 cm in the greatest dimension without ENE N2b—Multiple ipsilateral LN metastasis, none >6 cm in the greatest dimension, without ENE N2c—Bilateral or contralateral lymph nodes metastasis, none >6 cm in the greatest dimension, without ENE
N3a	LN metastasis >6 cm in the greatest dimension without ENE
N3b	LN metastasis in single or multiple LN with clinical ENE

Table 4 Clinical N stage for HPV-related (p16-positive) oropharyngeal cancer

Clinical N stage for HPV-related (p16-positive) oropharyngeal cancer	
Nx	Regional LN cannot be assessed
N0	No regional LN metastases
N1	One or more ipsilateral LN, none >6 cm
N2	Contralateral or B/L LN, none >6 cm
N3	Lymph node(s) > 6 cm

Table 5 Pathological N stage for HPV-related (p16-positive) oropharyngeal cancer

Pathologic N stage for HPV-related (p16-positive) oropharyngeal cancer	
Nx	Regional LN cannot be assessed
pN0	No regional LN metastases
pN1	Metastasis in four or fewer LN
pN2	Metastasis in more than four LN

primary tumour clinical classification which considers the greatest tumour dimension along with depth of invasion (DOI).

Pharynx

There are three distinct parts to the eighth edition of the AJCC staging manual:

Table 6 N staging for the nasopharynx

N staging for the nasopharynx	
NX	Regional LN cannot be assessed
N0	No regional LN metastasis
N1	Unilateral cervical lymph node(s) metastasis and/or unilateral or bilateral metastasis in retropharyngeal LN, ≤6 cm in the greatest dimension, above the caudal border of the cricoid cartilage
N2	Bilateral cervical lymph node(s) metastasis, ≤ 6 cm in the greatest dimension, above the caudal border of cricoid cartilage
N3	Cervical lymph node(s) metastasis, > 6 cm in dimension and/or extension below the caudal border of cricoid cartilage

Table 7 N staging for thyroid carcinoma

N staging for thyroid carcinoma	
NX	Regional LN cannot be assessed
N0	No regional LN metastasis
N1	Regional LN metastasis
N1a	Level VI (pretracheal, prelaryngeal, paralaryngeal) LN metastasis or upper/superior mediastinal LN
N1b	Unilateral, bilateral or contralateral cervical (levels I–V) or retropharyngeal LN metastasis

- Nasopharynx
- HPV-associated (p16-positive) oropharyngeal cancer
- Hypopharynx and non-HPV-associated (p16-negative) oropharyngeal carcinoma

To more accurately represent the range of pharyngeal illnesses [10].

Oropharynx

The oropharynx reaches from the hyoid bone's superior surface to the plane of the soft palate's superior surface. The oropharynx's subsites are listed in Table 1.

With two exceptions, T categories were identical in p16-positive, HPV-associated oropharyngeal cancer and p16-negative, non-HPV-associated oropharyngeal carcinoma. Because of the non-aggressive pattern of invasion of p16-positive oropharyngeal carcinoma and the absence of a distinct basement membrane in the epithelium of Waldeyer's ring, there

Table 8 T—primary tumour clinical classification for the lip and oral cavity

T—primary tumour clinical classification for the lip and oral cavity	
T1	≤ 2 cm in the greatest dimension and ≤5 mm DOI
T2	≤ 2 cm in the greatest dimension >5 mm but ≤10 mm DOI or > 2 cm but ≤4 cm in the greatest dimension and DOI ≤ 10 mm
T3	> 4 cm in the greatest dimension or >10 mm depth of invasion
T4a	Lip: tumour invades through the skin (chin or nose), floor of mouth inferior, alveolar nerve or cortical bone Oral cavity: tumour invades through the skin of the face and maxillary sinus or into deep/extrinsic muscle of the tongue and cortical bone
T4b	Lip or oral cavity: tumour invades the pterygoid plates, masticator space or skull base or encases the internal carotid artery

is no carcinoma in situ (Tis) in the p16-positive classification, and the T4b category has been eliminated from p16-positive oropharyngeal carcinoma (OPC) (because of no difference in the survival curves of the T4a and T4b) [11]. Tables 9 and 10 summarize these changes.

Nasopharynx

The nasopharynx starts anteriorly from the posterior choana and continues along the airway's plane to the level of the soft palate's free border. Table 1 provides a description of the subsites in the nasopharynx. Table 11 provides the clinical categorization for T—primary tumours.

Hypopharynx

The hypopharynx extends from the plane corresponding to the lower border of the cricoid cartilage to the plane of the superior border of the hyoid bone (or floor of the vallecula). It contains the:

- The lateral and posterior pharyngeal walls
- Piriform sinuses
- The postcricoid area

Table 9 T—primary tumour clinical classification of the oropharynx: p16-negative cancers or p16 immunohistochemistry not performed

Oropharynx: p16-negative cancers or p16 immunohistochemistry not performed	
T1	≤2 cm in the greatest dimension
T2	>2 cm but ≤4 cm in the greatest dimension
T3	>4 cm in the greatest dimension or extension to the lingual surface of the epiglottis
T4a	Invading any of the following: larynx, deep extrinsic muscles of the tongue (genioglossus, hyoglossus, palatoglossus and styloglossus), medial pterygoid, mandible and hard palate
T4b	Invading any of the following: lateral pterygoid muscle, pterygoid plates, lateral nasopharynx and skull base or encases the carotid artery

Table 10 T—primary tumour clinical classification of the oropharynx: p16-positive cancers

Oropharynx: p16-positive cancers	
T1	≤ 2 cm in the greatest dimension
T2	> 2 cm but ≤4 cm in the greatest dimension
T3	> 4 cm in the greatest dimension or extension to the lingual surface of the epiglottis
T4	Tumour invades any of the following: larynx, deep extrinsic muscles of the tongue (genioglossus, hyoglossus, palatoglossus and styloglossus), medial pterygoid, mandible and hard palate, lateral pterygoid muscle, pterygoid plates, lateral nasopharynx and skull base or encases the carotid artery

Table 11 T—primary tumour clinical classification of the nasopharynx

Nasopharynx	
T1	Tumour confined to the nasopharynx or extends to the oropharynx and/or nasal cavity without parapharyngeal involvement
T2	Tumour with extension to parapharyngeal space and/or infiltration of the medial pterygoid, lateral pterygoid and/or prevertebral muscles
T3	Tumour invades bony structures of skull base cervical vertebra, pterygoid structures and/or paranasal sinuses
T4	Tumour with intracranial extension and/or involvement of cranial nerves, hypopharynx, orbit, parotid gland and/or infiltration beyond the lateral surface of the lateral pterygoid muscle

Table 12 provides a review of the hypopharynx's current stage.

Larynx

Table 1 provides a description of the larynx's anatomical locations and subsites. Tables 13, 14 and 15 list the detailed T stages of the various laryngeal carcinoma locations.

Table 12 T—primary tumour clinical classification of the hypopharynx

Hypopharynx	
T1	Limited to one subsite of the hypopharynx and ≤2 cm in the greatest dimension
T2	Invades >1 subsite of the hypopharynx or an adjacent site or measures 2–4 cm in the greatest dimension, without fixation of the hemilarynx
T3	>4 cm in the greatest dimension or with fixation of the hemilarynx or extension to the oesophagus
T4a	Invades any of the following: thyroid/cricoid cartilage, hyoid bone, thyroid gland, oesophagus, central compartment soft tissue
T4b	Invades the prevertebral fascia, encases the carotid artery or invades the mediastinal structures

Table 13 T—primary tumour clinical classification of the supraglottis

Supraglottis	
T1	Limited to one subsite of supraglottis with normal vocal cord mobility
T2	Invades the mucosa of more than one adjacent subsite of the supraglottis or glottis or he region outside the supraglottis (e.g. mucosa of the base of the tongue, vallecula, medial wall of the pyriform sinus) without fixation of the larynx
T3	Limited to the larynx with vocal cord fixation and/or invades any of the following: postcricoid area, preepiglottic tissues, paraglottic space and/or with minor thyroid cartilage erosion (e.g. inner cortex)
T4a	Invades through the thyroid cartilage and/or invades tissues beyond the larynx, e.g. trachea, soft tissues of the neck, including deep/extrinsic muscle of the tongue (genioglossus, hyoglossus, palatoglossus and styloglossus), strap muscles, thyroid and oesophagus
T4b	Invades the prevertebral space and mediastinal structures or encases the carotid artery

Table 14 T—primary tumour clinical classification of the glottis

Glottis	
T1	Limited to the vocal cord(s) (may involve anterior or posterior commissure) with normal mobility T1a—tumour limited to one vocal cord T1b—tumour involved both vocal cords
T2	Extends to the supraglottis and/or subglottis and/or with impaired vocal cord mobility
T3	Limited to the larynx with vocal cord fixation and/or invades the paraglottic space and/or with minor thyroid cartilage erosion (inner cortex)
T4a	Invades through the thyroid cartilage or invades tissues beyond the larynx, e.g. trachea, soft tissues of the neck including the deep/extrinsic muscle of the tongue (genioglossus, hyoglossus, palatoglossus and styloglossus), strap muscles, thyroid and oesophagus
T4b	Invades the prevertebral space and mediastinal structures or encases the carotid artery

Table 15 T—primary tumour clinical classification of the subglottis

Subglottis	
T1	Limited to the subglottis
T2	Extends to the vocal cord(s) with normal or impaired mobility
T3	Limited to the larynx with vocal cord fixation
T4a	Invades through the cricoid or thyroid cartilage and/or invades tissues beyond the larynx, e.g. trachea, soft tissues of the neck including the deep/extrinsic muscle of the tongue (genioglossus, hyoglossus, palatoglossus and styloglossus), strap muscles, thyroid and oesophagus
T4b	Invades the prevertebral space and mediastinal structures or encases the carotid artery

Nose and Paranasal Sinuses

Table 1 lists the anatomical locations and sublocations of the nose and paranasal sinuses. Tables 16 and 17 provide descriptions of the T—primary tumour clinical categorization of cancer.

Salivary Gland

The major salivary glands are the only ones included by categorization.

Table 16 T—primary tumour clinical classification of the maxillary sinus

Maxillary sinus	
T1	Tumour limited to the antral mucosa with no erosion or destruction of the bone
T2	Tumour causing bone erosion or destruction, including extension into the hard palate and/or middle nasal meatus, except extension to the posterior wall of the maxillary sinus and pterygoid plates
T3	Tumour invades any of the following: bone of the posterior wall of the maxillary sinus, subcutaneous tissues, floor or medial wall of orbit, pterygoid fossa or ethmoid sinuses
T4a	Tumour invades any of the following: anterior orbital contents, skin of the cheek, pterygoid plates, infratemporal fossa, cribriform plate and sphenoid or frontal sinus
T4b	Tumour invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve, nasopharynx, clivus

Table 17 T—primary tumour clinical classification of the nasal cavity and ethmoidal sinus

Nasal cavity and ethmoid sinus	
T1	Tumour restricted to one subsite of the nasal cavity or ethmoid sinus without bone erosion
T2	Tumour involves two subsites or extends to involve an adjacent site within the nasoethmoidal complex, with or without bony invasion
T3	Tumour extends to invade the medial wall or floor of the orbit, maxillary sinus, palate or cribriform plate
T4a	Tumour invades any of the the following: anterior orbital contents, skin of nose or cheek, minimal extension to the anterior cranial fossa, pterygoid plates, sphenoid or frontal sinuses
T4b	Tumour invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve, nasopharynx, clivus

The primary salivary glands are

- Parotid glands
- Submandibular glands
- Sublingual glands

Table 18 provides descriptions of the T—primary tumour clinical categorization of salivary gland cancer.

Table 18 T—Primary tumour clinical classification of the salivary gland

Salivary gland	
T1	Tumour 2 cm or less in the greatest dimension without extraparenchymal extension*
T2	Tumour more than 2 cm but no more than 4 cm in the greatest dimension without extraparenchymal extension*
T3	Tumour more than 4 cm and/or tumour with extraparenchymal extension
T4a	Tumour invades the skin, mandible, ear canal or facial nerve
T4b	Tumour invades the base of the skull and pterygoid plates or encases the carotid artery

Except for the tissues specified under T4a and T4b, extraparenchymal extension is clinical or macroscopic evidence of invasion of the skin, soft tissues or nerve. For categorization purposes, microscopic data by itself does not support extraparenchymal extension.

Thyroid Gland

The four major histopathologic types are:

- Papillary carcinoma (including those with follicular foci)
- Follicular carcinoma (including Hürthle cell carcinoma)
- Medullary carcinoma
- Undifferentiated (anaplastic) carcinoma

The current staging system is summarized in Table 19.

Skin Carcinoma of the Head and Neck

The eighth edition of the UICC/AJCC handbook now presents this in a distinct chapter [10]. The categorization includes malignant melanoma, Merkel cell carcinoma and cutaneous carcinomas of the head and neck that do not include the eyelid (as an anatomical location). Table 20 shows the T stage, and the N stage is shown after nodal metastases from various head and neck locations.

The following sites are recognized:

- Lip
- External ear

Table 19 T—primary tumour clinical classification of the thyroid gland

Thyroid gland	
T1	Tumour 2 cm or less in the greatest dimension, limited to the thyroid T1a—tumour 1 cm or less in the greatest dimension, limited to the thyroid T1b—tumour more than 1 cm but not more than 2 cm in the greatest dimension, limited to the thyroid
T2	Tumour more than 2 cm but not more than 4 cm in the greatest dimension, limited to the thyroid
T3	Tumour more than 4 cm in the greatest dimension, limited to the thyroid or any tumour with gross extrathyroid extension involving only the strap muscles (e.g. sternohyoid, sternothyroid or omohyoid muscles) T3a—tumour more than 4 cm in the greatest dimension, limited to the thyroid T3b—tumour of any size with gross extrathyroidal extension invading the strap muscles (sternohyoid, sternothyroid or omohyoid muscles)
T4a	Tumour extends beyond the thyroid capsule and invades any of the following: subcutaneous soft tissues, larynx, trachea, oesophagus, recurrent laryngeal nerve
T4b	Tumour invades the prevertebral fascia and mediastinal vessels or encases the carotid artery
T4a[a]	Anaplastic carcinoma only—tumour (any size) limited to the thyroid
T4b[a]	Anaplastic carcinoma only—tumour (any size) extends beyond the thyroid capsule

[a]All anaplastic carcinomas are considered T4

Table 20 T—primary tumour clinical classification of head and neck skin cancer

Head and neck skin cancer	
T1	Tumour 2 cm or less in the greatest dimension
T2	Tumour >2 cm and <4 cm in the greatest dimension
T3	Tumour >4 cm in the greatest dimension or minor bone erosion or perineural invasion or deep invasion*
T4a	Tumour with gross cortical bone/marrow extension
T4b	Tumour with skull base or axial skeleton invasion including foraminal involvement and/or vertebral foramen involvement to the epidural space

*Deep invasion is defined as invasion beyond the subcutaneous fat or > 6 mm (as measured from the granular layer of adjacent normal epidermis to the base of the tumour), perineural invasion for T3 classification is defined as clinical or radiographic involvement of named nerves without foramen or skull base invasion or transgression.

Table 21 T—primary tumour clinical classification of malignant melanoma of the upper aerodigestive tract

Malignant melanoma of the upper aerodigestive tract	
TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
T3	Tumour limited to the epithelium and/or submucosa (mucosal disease)
T4a	Tumour invades the deep soft tissue, cartilage, bone or overlying skin
T4b	Tumour invades any of the following: brain, dura, skull base, lower cranial nerves (IX, X, XI, XII), masticator space, carotid artery, prevertebral space, mediastinal structures

- Other and unspecified parts of the face
- Scalp and neck

Malignant Melanoma of the Upper Aerodigestive Track

Only head and neck mucosal malignant melanomas are subject to the categorization in Table 21. Stages I and II as well as T1 and T2 are skipped due to the aggressive nature of malignant melanoma tumours.

Squamous Cell Carcinoma

With the exception of the thyroid and major salivary glands, squamous cell carcinoma (SCC) is the most prevalent histological form of malignancy in the head and neck area, accounting for about 95% of cases. The precursor lesion of SCC is known as squamous epithelial dysplasia, which is described as an accumulation of cytological and architectural abnormalities. Due to 'field cancerization', these dysplastic regions might be confined or widespread. The malignant transformation rate for oral dysplastic lesions is 12%, and the mean time to transformation is 4.3 years, according to a meta-analysis [12]. This suggests that the development into invasive malignancy is not always the case. Clinically, these lesions look irregular, confined, white (leucoplakia), red (erythroplakia) or variegated (erythroleukoplakia). Defined as a dynamic process with a continuous morphological range, dysplasia is so.

Pathogenesis

The histological evolution of head and neck squamous cell carcinoma (HNSCC) from the normal epithelium to hyperplasia, dysplasia and, ultimately, invasive carcinoma is influenced by a

number of variables, including genetic alterations that cause genetic instability leading to cellular transformation [13]. The inactivation of tumour suppressor genes and the activation of proto-oncogenes, or both, are examples of genetic alterations. Tobacco-related carcinogens cause p53 mutations that are linked to HNSCC. A number of genetic abnormalities, including the loss of heterozygosity (LOH) of 9p21, which is present in 70–80% of HNSCC, are responsible for the molecular changes. The viral oncoproteins E6 and E7 in HPV16 infection render the tumour suppressor molecules pRb and p53 inactive. Cell growth becomes uncontrolled when pRb is lost. Genomic stability derives from p53-mediated response to DNA damage loss. Additionally, telomerase is reactivated, which promotes cell immortality [13, 14].

Macroscopic Appearance

HNSCC can emerge as an exophytic or ulcerative lesion, among other things.

Microscopic Appearance

- Evidence keratinization and intercellular prickles are often seen in well-differentiated and moderately differentiated SCC (desmosomes). SCC with poor differentiation lacks keratinization signs.
- Some characteristics of malignant squamous cells include:
- Coarse and clumped chromatin
- Nuclear hyperchromatism
- Disordered cell polarity
- Prominent nucleoli
- Increased and abnormal mitosis
- Loss of keratin production
- Increased nucleus-cytoplasmic ratio
- Nuclear pleomorphism
- Disorganized growth
- Premature keratinization (dyskeratosis)

The distinguishing characteristic of SCC is the rupture of the sub-epithelial basement membrane, which enables the malignant cells to invade the healthy connective tissue beneath.

An SCC is described microscopically as follows:

- Histological subtype
- Histological grade (well-, moderate-, poorly differentiated)
- Invasive front (cohesive or non-cohesive)
- Vascular invasion
- Neural invasion

Immunohistochemistry

IHC utilized for reliable diagnosis since the weakly differentiated SCC lacks keratinization and is difficult to recognize histologically. The nuclear proteins p40 and p63, as well as the cytokeratins 5, 6 and 14, are utilized to support the diagnosis of poorly differentiated SCC. HPV testing is carried out for oropharyngeal SCC since it has prognostic importance. It is found using p16 IHC and high-risk HPV DNA ISH. More than 70% of malignant cells exhibit strong and widespread nuclear and cytoplasmic staining. Oncogenic HPV infection is known to be represented by the surrogate marker p16; the test is extremely sensitive but unspecific. The nasopharynx is often where EBV-positive SCCs are found.

Head and Neck Squamous Cell Carcinoma Variants

The morphological variants of SCC include:

- Verrucous carcinoma
- Spindle cell carcinoma (sarcomatoid carcinoma)
- Papillary SCC
- Adenosquamous carcinoma
- Basaloid SCC

Nasopharyngeal Carcinoma

The distinct kind of SCC known as nasopharyngeal carcinoma (NPC) develops in the nasopharynx. The age-adjusted incidence for both sexes of this neoplasm is fewer than one per 100,000 people worldwide, with southern China, northern Africa and Alaska having substantially higher rates of the illness. Inuit people from Alaska and ethnic Chinese residents of the province of Guangdong are particularly vulnerable to the illness [15]. In Hong Kong, which is geographically close to Guangdong

province, the reported incidence of nasopharyngeal cancer is 20–30 per 100,000 and 15–20 per 100,000, respectively [16]. Immigrants from China to Southeast Asia or North America continue to have a high risk of nasopharyngeal cancer [17]. According to this result, environmental, racial and genetic variables may all have a part in the development of the disease. EBV and dietary variables, particularly a high consumption of salted fish and preserved vegetable items, are additional contributors.

The tumour might appear clinically as a bulging, exophytic, lobulated or ulcerative mass. Occasionally, a blind biopsy of the nasopharynx is necessary when there is no visible tumour.

According to the WHO classification system, NPC is categorized microscopically based on the presence or lack of keratinization.

Nasopharyngeal cancer is histologically categorized by the WHO [18]:

- Keratinizing squamous cell carcinoma (WHO type I)

This kind of nasopharyngeal carcinoma is characterized by the presence of squamous differentiation with intercellular bridges and/or keratinization across the majority of its range.

- Non-keratinizing carcinoma

These tumours typically have greater connections with the Epstein-Barr virus and are more radiosensitive than SCC:

1. Differentiated non-keratinizing carcinoma (WHO type II)

   The tumour cells differentiate in a way that causes them to mature in a way that prevents squamous differentiation from being seen under a light microscope.
2. Undifferentiated carcinoma (WHO type III)

   The nuclei of the tumour cells are spherical or oval with large nucleoli. The tumour seems syncytial rather than pavemented, and the cell boundaries are indistinct.

Malignant Salivary Gland Tumours

According to GLOBOCAN 2020 statistics, malignant salivary gland tumours are rare, accounting for 5% of all head and neck cancers, 5% of all malignancies and having an annual incidence of 0.57 per 100,000 people worldwide [19]. Despite having a low prevalence, there are over 20 distinct types of designated malignant salivary gland neoplasm. Malignant epithelial salivary gland tumours encompass a diverse array of types as per the 2017 WHO Classification of Head and Neck Tumours [7]. These include adenoid cystic carcinoma, basal cell adenocarcinoma, myoepithelial carcinoma, carcinoma ex-pleomorphic adenoma, clear cell carcinoma, adenocarcinoma NOS (not otherwise specified), oncocytic carcinoma, squamous cell carcinoma, large cell neuroendocrine carcinoma, sialoblastoma, epithelial/myoepithelial carcinoma, secretory carcinoma, carcinosarcoma, mucoepidermoid carcinoma, undifferentiated carcinoma, intraductal carcinoma, sebaceous adenocarcinoma, salivary duct carcinoma, lymphoepithelial carcinoma, polymorphous adenocarcinoma, small cell neuroendocrine carcinoma, poorly differentiated carcinoma and acinic cell carcinoma.

Mucoepidermoid Carcinoma

The most frequent malignant salivary gland neoplasm, accounting for 12–29% of all salivary gland malignancies, is mucoepidermoid carcinoma [7]. The parotid gland is the salivary gland that is most frequently affected. The peak age group in the fifth decade is when females are more likely to develop mucoepidermoid carcinoma.

Gross Findings

- Infiltrative or varyingly encapsulated.
- The solid portion, which ranges in colour from tan-white to pink, is more noticeable.
- There are often cysts that contain a viscous, dark fluid.

Microscopic Findings

- Contains cells that are mucous, intermediate, and epidermoid.

- Clear cells, columnar cells, and oncocytes are further types of cells.
- Cystic and solid regions appear in various amounts along with other patterns in tumours.
- Characteristic of reactive tumour-associated lymphoproliferation.
- Fibrosis and inflammation are frequent.
- There are three classes of tumours: low, moderate and high grades.

Immunohistochemical Findings

- Cytokeratin, p63, p40 and commonly EMA are immunoreactive in intermediate and epidermoid cells.

Molecular Findings

- Independent of grade, MAML2 rearrangement is seen in 78% to 85% of patients.

Fine-Needle Aspiration

- Cellular stains with a mucinous material background.
- Sheets of cells and cells flowing in the mucus can be found in cohesive epithelial clusters.
- When intermediate and epidermoid cells are present, mucocytes aid in the diagnosis.

Mucoepidermoid carcinoma exhibits a wide range of behaviours, and several methods have been put forth to try and grade tumours and, as a result, predict outcomes. The grading system for mucoepidermoid carcinoma [20] incorporates various parameters and assigns point values to each. These parameters include the percentage of intracystic component (<20%), the presence of neural invasion, the presence of necrosis, the number of mitoses (>4 per 10 high-power fields) and the presence of anaplasia. Each parameter is assigned a specific point value, with intracystic component and neural invasion carrying 2 points each, necrosis and mitoses carrying 3 points each and anaplasia carrying 4 points. The total score is calculated by summing the points for each parameter. Based on the total score, the grade of the carcinoma is determined. A total score of 0–4 corresponds to a low-grade carcinoma, with an estimated overall survival rate of 90% at 10 years. A total score of 5–6 indicates an intermediate-grade carcinoma, with an estimated overall survival rate of 70% at 10 years. A total score of 7–14 signifies a high-grade carcinoma, with an estimated overall survival rate of 25% at 10 years. This grading system provides valuable information for assessing the prognosis and guiding the treatment of mucoepidermoid carcinoma.

Acinic Cell Carcinoma

Approximately 7–17.5% of malignant salivary gland tumours are acinic cell carcinomas.

Distribution of acinic cell carcinoma according to occurrence in various salivary glands is as follows:

- 80% of acinic cell carcinomas are found in the parotid.
- 20% are found in the submandibular and minor salivary glands.
- 1% is found in the sublingual gland.

Males predominate, and they begin to appear around the third decade of life [7].

Gross Findings

- Usually less than 3 cm, rubbery or hard tumours
- Typically limited (occasionally irregular)
- May exhibit bleeding or a cystic transformation

Microscopic Findings

The various growth patterns of acinic cell carcinoma are mentioned in Table 22.

Table 22 Growth patterns of acinic cell carcinoma [7]

Growth patterns of acinic cell carcinoma	
Growth pattern	Features
Solid	Lobules or nodules are formed when cells group together into sheets
Microcystic	The microcysts present give the tumour a sieve-like appearance
Papillary—cystic	Significant cysts with intraluminal papillary projections are seen in the tumour
Follicular	Include many cysts of various sizes that somewhat resemble thyroid parenchyma

- Serous acinar cells with basophilic zymogen granules in the cytoplasm are the distinctive cell type (usually accentuated at the lumen).
- Vacuolated and transparent cells are among the other types of cells.
- Follicular and solid growth patterns.
- TALP/infiltrate (tumour-associated lymphoid proliferation) is occasionally evident.

Ancillary Studies

- Zymogen granules are diastase-resistant and PAS-positive.
- Acinic cells may exhibit amylase, transferrin, lactoferrin, DOG1 and SOX10-positive staining.
- Ten percent or so demonstrate some S100-positive protein (usually weaker than adjacent nerves).

Fine-Needle Aspiration

- Clear cellular stains on a backdrop.
- Cohesive, compact and close clusters.
- There is a lot of granular to vacuolated cytoplasm around the spherical, regular nuclei. The chromatin is coarse (lymphocyte-like nuclei).

Adenoid Cystic Carcinoma

- Ten to 12% of malignant salivary gland tumours are adenoid cystic carcinomas (ACC).
- Submandibular gland cancer is the most prevalent kind.
- It accounts for 30–50% of small salivary gland neoplasms and around 5% of parotid neoplasms.
- No gender preference.
- Most prevalent between the fourth and sixth decades, infrequent in people under the age of 20 [7].

Gross Findings

- Typically solid, frequently well-circumscribed, but not encased

Microscopic Findings

- Including modified myoepithelial cells and duct lining cells.
- In a decreasing order of occurrence, the primary patterns are cribriform, tubular and solid; mixed patterns are frequent.
- Pseudocysts contain hyaline, eosinophilic or basophilic mucoid substance (reduplicated basement membrane material).
- Perineural invasion occurs often and is usually severe.
- Small cells have a large nuclear-to-cytoplasmic ratio.
- Nuclei have a dense distribution of nuclear chromatin and can range in form from carrot-shaped to peg-shaped.
- In ACC with high-grade transformation, stroma is desmoplastic and hyalinized in typical ACC.

According to the prevalent development pattern, ACC may be rated [7]:

- Grade I—Most of which are tubular, and some of which are cribriform.
- Grade II—Cribriform/tubular having less than 30% solid component or fully cribriform.
- Grade III—Any tumour that has solid growth that is higher than 30%.

Ancillary Studies

- Pseudocysts are positive for type IV collagen, laminin, PAS and Alcian blue.
- Low-molecular-weight keratins, EMA and CD117-positive epithelial cells.

Fine-Needle Aspiration

- Cellular smears that are often cohesive sheets of monotonous, sporadic epithelial cells.
- Hyaline globules, which are best observed on air-dried preparations and are reduplicated basement membrane material that resembles pink ‘gum balls’, are surrounded by cells (bright magenta on Romanowsky stains).
- High nuclear-to-cytoplasmic ratio cells contain hyperchromatic nuclei that are peg-shaped or cuboidal.

Carcinoma Ex-Pleomorphic Salivary Adenoma

A carcinoma ex-pleomorphic adenoma develops within or at the location of an earlier pleomorphic adenoma, roughly 6.2% of all pleomorphic

adenomas and 12% of all salivary gland tumours [7, 21].

Risk factors for a pleomorphic adenoma's development into cancer include [7, 21]:

- Older age of the patient (mean, 61 years)
- Male gender
- Prolonged duration of tumour
- Multiple recurrences

The sublingual gland and the parotid gland are less frequently affected.

Gross Findings
- Larger than its benign equivalent.
- Tan-white, firm, poorly delimited, heavily infiltrative masses.
- Sometimes they are well-circumscribed and seem contained.
- The malignant component, assuming it is, tends to radiate centrifugally from this nidus.

Microscopic Findings
- High-grade adenocarcinoma and salivary duct carcinoma, frequently with squamoid differentiation, make up the malignant component in most cases.
- Salivary carcinoma also occurs in other particular subgroups, some of which are monotypic rather than mixed.
- Pleomorphic adenoma, carcinoma and sarcoma (also known as carcinosarcoma, a truly malignant mixed tumour) are extremely uncommon together.

The prognostic importance of the tumour invasion into the capsule of the initial lesion has been described as [7]:

1. Intracapsular carcinoma
2. Minimally invasive carcinoma (less than 4–6 mm invasion)
3. Widely invasive carcinoma

In principle, [1] and [2] have a favourable prognosis compared to [3].

Lymphomas

The second most frequent primary cancer that affects the head and neck is lymphoma. The head and neck region is where around 25% of extranodal lymphomas are found.

An interested reader is directed to the detailed WHO classification of tumours of haematopoietic and lymphoid tissue after reading this general review of lymphoma (fourth edition, 2008) [22].

Non-Hodgkin lymphoma (NHL) and Hodgkin lymphoma (HL) are the two main types of lymphomas (NHL).

Hodgkin Lymphoma (HL)
- Characterized by Reed-Sternberg cells
- Subdivided into:
  - Classical HL:
    - Nodular sclerosing
    - Mixed cellularity
    - Lymphocyte-rich
    - Lymphocyte-depleted
  - Nodular lymphocyte predominant HL

These traditional HL subtypes are divided based on the location of involvement, clinical characteristics, growth pattern, cellular background composition, presence of fibrosis, number and/or degree of atypia of the tumour cells and frequency of EBV infection. Neoplastic cells in all of these subtypes share the same immunophenotype.

Non-Hodgkin Lymphoma (NHL)
- The majority of NHLs—about 85%—are B-cell lymphomas.
- Among aggressive NHLs, diffuse large B-cell lymphoma is the most prevalent.
- Aggressive NHLs that can occasionally be seen in the head and neck area include Burkitt lymphoma and mantle cell lymphoma.
- Rarely observed in the head and neck area are plasma cell neoplasms, most frequently plasma cell myeloma and extramedullary plasmacytoma.
- The head and neck skin may be affected by cutaneous T-cell NHLs; however mucosal areas are seldom affected.
- Strongly linked to EBV infection, extra-nodal NK/T-cell lymphoma prefers the upper aerodigestive tract, often the nasal cavity. It is angiocentric and angio-destructive in nature, mimicking the histological characteristics of polyangiitis in granulomatosis.

Malignant Mesenchymal Tumours

In the head and neck area, malignant mesenchymal tumours (sarcomas) include:

- Liposarcoma
- Rhabdomyosarcoma
- Malignant fibrous histiocytoma
- Osteosarcoma
- Kaposi sarcoma
- Chondrosarcoma
- Angiosarcoma
- Synovial sarcoma
- Mesenchymal chondrosarcoma
- Leiomyosarcoma
- Malignant peripheral nerve sheath tumour

Neuroectodermal Tumours

Mucosal Malignant Melanoma

An uncommon kind of tumour called mucosal malignant melanoma develops from melanocytes that are generated from the neuroectoderm [23].

Epidemiology

- Roughly 15–20% of cases occur in the head and neck, and more than 80% have cutaneous or upper aerodigestive tract origins.
- Between 0.5 and 3% of malignant melanomas across all sites are mucosal [24].
- The sinuses and oral cavity, particularly the palate, are frequent locations for mucosal malignant melanoma.
- More typical in males (findings are not consistent).
- A broad age span (20 to 80).

Gross Findings

- Depending on the quantity of melanin produced, they are often pigmented and range in colour from a light tan to black.

Microscopic Findings

- Often made up of spindle and/or epithelioid cells.
- The cells have a distinct pleomorphic appearance and might be pigmented.
- Intra-nuclear inclusions are common, and nucleoli are obvious.
- Necrosis and inflammatory infiltrates might also be found nearby.
- Melanocytic atypia or melanoma in situ may be seen in the background mucosa.
- For the S100 protein, cells exhibit nuclear and cytoplasmic positivity.
- Typically, MelanA, HMB-45 and SOX10 are positive.
- Epithelial markers are negative.
- All these indicators may not be present in the desmoplastic form of melanoma.

Olfactory Neuroblastoma

Uncommon sinonasal malignant tumour originating from the superior turbinate, cribriform plate and superior third of the nasal septum affects the specialized olfactory mucosa.

There is no sex- or race-specific preference, and the reported incidence is four instances per million with a bi-modal distribution in the second and sixth decades [7].

Gross Findings

- Soft, highly vascular, polypoidal, mucosa-covered tumour.

Microscopic Findings

- The chromatin of the malignant cells is finely stippled (like salt and pepper); has tiny, spherical nuclei and little cytoplasm; and is homogenous.

Table 23 lists the four grades of olfactory neuroblastoma according to Hyams' categorization scheme [25].

Table 23 Hyams' grading classification of olfactory neuroblastoma [25]

Hyams' grading classification of olfactory neuroblastoma				
Histological feature	Grade I	Grade II	Grade III	Grade IV
Architecture	Lobular	Lobular	May be lobular	May be lobular
Mitoses	Absent	Present	Prominent	Marked
Nuclear pleomorphism	Absent	Present	Prominent	Marked
Neurofibrillary matrix	Prominent	Present	May be present	Absent
Rosettes	Homer-Wright rosettes	Homer-Wright rosettes	Flexner-Wintersteiner rosettes	Flexner-Wintersteiner rosettes
Necrosis	Absent	Absent	Present	Prominent

However, there is not a single distinct positive indication.

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Cutaneous Malignancies of Head and Neck

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1 Introduction

The skin is the largest organ in the body and can be prone to various malignancies due to its size and diverse appendages. Basal cell carcinoma and squamous cell carcinoma (SCC) are the most common types of skin cancer, and malignant skin lesions can vary from locally destructive basal cell carcinoma (BCC) to rapidly metastatic melanoma. It is difficult to determine the exact incidence of these types of cancer.

1.1 Principles of Evaluation and Treatment

- Majority of the cutaneous malignancies present as surface lesions. Certain adnexal tumours may present as cutaneous bulges and subepithelial lesions.
- A thorough clinical examination by gentle palpation of the lesion and surrounding tissue to determine the extent of the tumour and draining of the lymph node basin.
- Bilateral parotid glands and submental and submandibular regions must be thoroughly examined for lymph nodal metastasis.
- Radiologic evaluation is directed towards assessing the local extension of disease in the dermis, subcutaneous plane and satellite nodules, along with bone erosion, which can be defined by contrast-enhanced computerized tomography (CECT) or magnetic resonance imaging (MRI).
- Several cutaneous malignancies can be neurotropic and manifest perineural extensions.
- Depth of invasion (DOI) assessment is also critical in the evaluation and treatment.

1.2 Operative Techniques

- After following all standard protocols and under strict aseptic precautions, all patients were taken under general anaesthesia.
  - A wide local excision (WLE) of the cutaneous lesion was performed in a well-planned manner. The aim was to achieve R0 resection with the least morbidity, and the best possi-

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_3

ble reconstruction was done to provide good form and possibly function (as necessary).
– Adjuvant therapy was planned after postoperative staging and case-by-case scenarios.

2 Surgical Tips

The face is divided into complex aesthetic units, including the central face units of the lips, nose, eyebrows and eyelids and the lateral complex unit of the external ear. Reconstructing these units involves specific techniques to maintain function and appearance. Lip reconstruction aims to preserve oral competence, labial sensation and aperture size by properly aligning the orbicularis oris muscle and suturing the layers. Eyelid reconstruction involves rebuilding the trilaminar structure of the eyelid, with larger defects able to be closed directly in elderly patients due to laxity in the lower lid skin and tarsus. Nasal reconstruction involves restoring the skin, skeletal support and nose lining, with flaps taken from the forehead, nasolabial region or glabella often used for the skin only, and full thickness or total defects requiring the reconstruction of all three layers.

In reconstructive surgery, the main principles are to place scars in natural lines of facial expression, reconstruct aesthetic subunits, replace like with like, consider colour differences in the head and neck skin and mimic the natural contours of the head and neck, avoiding excess bulk or thinness. The facial nerve branches exit the skull at the stylomastoid foramen, with landmarks such as the posterior belly of the digastric muscle, tragal pointer and tympanomastoid suture line aiding in identification. Proper wound closure without tension is important for optimal and early healing.

3 Concepts of Cutaneous Malignancies

Skin cancer rates have increased among higher socio-economic groups, possibly due to sunbathing and indoor tanning facilities. Tanning beds are a modifiable risk factor for all types of skin cancer and should be a focus for public education and awareness. Studies have shown that indoor tanning can increase the risk of squamous cell carcinoma by 15–67% and basal cell carcinoma by 15–29%. Using indoor tanning more than four times per year can also increase the risk of melanoma by 11%. Other risk factors for skin cancer include prolonged sun exposure, sunburns, having a Fitzpatrick skin type I or II, genetics, exposure to ionising radiation and immune system suppression. Increased ultraviolet radiation makes skin cancers more common at high altitudes and in locations closer to the equator. UVB radiation can cause DNA damage and is linked to non-melanoma skin cancers. Intermittent and intense sun exposure in early life increases the risk of basal cell carcinoma, while cumulative UV radiation exposure is a risk factor for SCC. Multiple sunburns may also increase the risk of melanoma.

3.1 Basal Cell Carcinoma

The basal layer of the epidermis and its appendages give birth to BCC, a slow-growing epithelial cancer. Even though BCC seldom metastasises (1:35,000), its invasive and locally destructive behaviour can nonetheless be exceedingly morbid. BCC is the most prevalent form of human cancer worldwide, accounting for about 80% of newly diagnosed cases in the west. Nearly 25% of these occur on the nose, and around 80% occur in the head and neck [1].

A BCC can be recognised clinically by its nodular, pearly opalescence and noticeable telangiectasia. It frequently has a rolling, elevated border with ulceration in the centre and may itch. Some subtypes manifest as a scar-like lesion in a region that has never been damaged or as a patch of the skin that regularly ulcerates and heals in a normal-appearing area. The benign melanocytes in pigmented BCC may give it a dark colour [2].

BCC appears as nests of symmetrical, incredibly dark basaloid cells with scant cytoplasm and oval nuclei under a microscope. The cells can be seen penetrating the papillary dermis and frequently going deeper into the epidermis. These cells consist of separate islands and cords, many of which have palisading borders. Cells frequently form a ‘picket fence’ pattern comparable to the skin’s basal layer [3]. Some tumours secrete keratin, which congeals into pearl-like

forms surrounding the tumour. This is different from SCC pearls, which originate inside the tumour.

BCC nearly always develops in the skin with hair, and its growth relies on the area's dermal stroma. This reliance on cutaneous fibroblast-produced loose connective tissue that may account for the BCC has a limited metastatic potential. Similar to healthy, cycling adult hair follicles, BCC cells communicate with the mesenchymal cells of the stroma. Proteolytic enzymes that the tumour produces break down pre-existing dermal tissue and promote the development of tumour cells.

BCC is a type of skin cancer that can take various forms, including nodular, superficial multifocal, sclerosing, infiltrative and morpheaform. The nodular form is the most common, appearing as a pearly, dome-shaped nodule with central ulceration. Superficial multifocal BCC appears as a scaly red-brown patch with a subtle pearly border, often found on the trunk of patients. This subtype is often seen in people exposed to arsenic or radiation but can also occur without such exposure. Sclerosing BCC appears as an atrophic plaque with telangiectasias and ulceration and is typically found around the ears and nose. It can be difficult to diagnose and identify the margins of this type due to its desmoplastic reaction.

Infiltrative BCC, which occurs in 20% of cases, is characterised by only a small portion of the tumour being visible, while the rest extends widely beneath the skin. These tumours have a high recurrence rate and a widespread invasion pattern. Morpheaform BCC appears as a yellow, scar-like plaque with telangiectasias and indistinct margins. It can spread widely within the skin and often requires radical surgery for complete removal. Ulcus terebrans is an aggressive form of BCC that can invade large vessels, bones and even the meninges and can be fatal due to bleeding or infection.

Unlike other types of cancer, BCC does not tend to become more aggressive over time despite continued exposure to UV radiation. It is not fully understood why BCC cells do not acquire additional mutations that lead to more aggressive behaviour. However, certain characteristics are associated with more aggressive BCC, including a size greater than 6 mm on the face or greater than 10 mm on the forehead, scalp and neck, perineural invasion, a subtype other than nodular or superficial, previous radiation exposure and poorly defined clinical borders.

In 2012, the US Food and Drug Administration approved the use of vismodegib to treat metastatic and locally advanced BCC. This is the first systemic medication to be approved for this use. Vismodegib is taken as a 150 mg capsule daily until the disease progresses and is an option for patients who are not candidates for surgery or radiation.

4 Complications

Wound breakdown, also known as dehiscence, is the splitting or separation of a wound along a surgical incision. It can be caused by excessive wound tension or an infection. If a wound breakdown occurs within 24 h after surgery, it may be possible to try closing the wound again. However, attempting to close the wound after this time increases the risk of infection. Dehisced wounds are usually allowed to heal on their own, with revision surgery considered after the wound has fully healed.

Flap necrosis occurs when the blood flow to the distal end of a flap is impaired, resulting in ischemia. This can happen if the pedicle, or the blood vessels that supply the flap, is too small to support the flap, if it is too thin and disrupts the dermal plexus, or if excessive undermining of the surrounding tissue disrupts the perforating vessels feeding the pedicle. Flap necrosis may present as sloughing of the epidermis, and it is important not to debride the affected area until the wound has fully healed. In some cases, only the epidermal portion of the flap may have necrosed and debriding the flap could disturb the viable tissue. Even when the entire flap has necrosed, the eschar (dead tissue) over the wound can act as a natural dressing and allow the wound to heal on its own.

Scarring can occur when a wound heals by secondary intention, resulting in a disfigured appearance. Facial weakness may be temporary if the injury is a neuropraxia or permanent if the main trunk or a terminal branch of the facial nerve is transected or injured by cautery. The exact cause of neuropraxia is not well understood. Still, it is thought to be caused by a combination of factors such as trauma to the nerve during surgery, trac-

tion injury, heat injury from cautery and prolonged surgery time. The lower division branches of the facial nerve, particularly the marginal mandibular branch, are more thin and fragile and may be more prone to neuropraxia.

5 Excision of Tumour of the Nasoethmoidal Region

5.1 Case 1

A 5-year-old girl presented with a 2-month history of growth in the right nasoethmoidal region, gradually progressive. Contrast-enhanced computerized tomography (CECT) was suggestive of epithelial lesion without bony involvement. A preoperative biopsy was suggestive of BCC, and the final HPE report was BCC.

5.2 Operative Technique

The surgical steps are depicted in Figs. 1, 2, 3, 4, 5, 6 and 7. The postoperative follow-up of the patient in the ninth month is shown in Fig. 8.

Fig. 1 Incision marking with 0.5 cm margin

Fig. 2 Incision deepened up to the bone, preserving the medial canthus. The angular vessels may need to be ligated

Fig. 3 The specimen is gently elevated off the bone, preserving the medial canthal ligament and lacrimal apparatus (whenever oncologically safe)

Fig. 4 The arrow depicts the preserved lacrimal apparatus and medial canthal ligament; this ensures the stability of the upper and lower eyelids

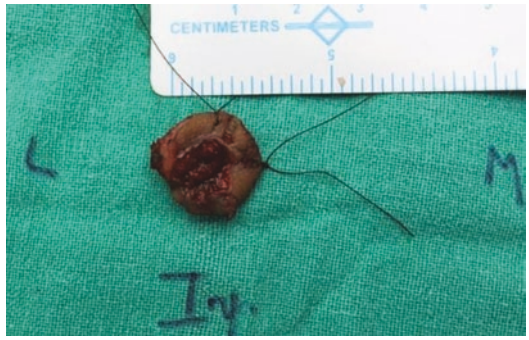

Fig. 5 Resected specimen must be oriented and marked for accurate histopathological diagnosis

Fig. 6 A paramedian forehead flap based on supratrochlear and supraorbital artery was performed to close the defect

Fig. 7 The paramedian flap second-stage division and reinsetting procedure done in the third postoperative week

Fig. 8 Postoperative 9-month follow-up showing excellent healing with good locoregional control

6 Excision of Tumour of the Nasal Tip with the Forehead Flap

6.1 Case 2

A 56-year-old male presented with a 3-month history of gradually progressive growth on the nasal tip. CECT suggested an epithelial lesion without underlying cartilage involvement. A preoperative biopsy was suggestive of poorly differentiated carcinoma, but the final HPE report was suggestive of well-differentiated squamous cell carcinoma (WDSCC).

6.2 Operative Technique

The surgical steps are depicted in Figs. 9, 10, 11, 12, 13 and 14. The postoperative follow-up of the patient in the third week after flap division and sixth month is shown in Figs. 15 and 16.

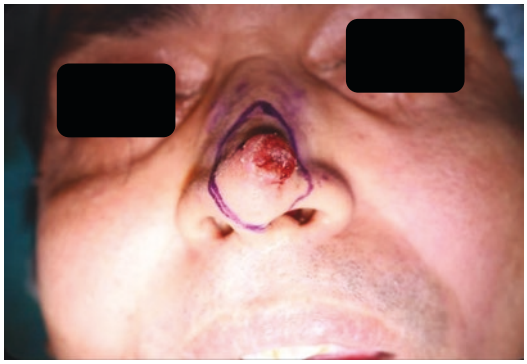

Fig. 9 A 0.5 × 0.8 cm ulceroproliferative growth on he nasal tip, more on the left side

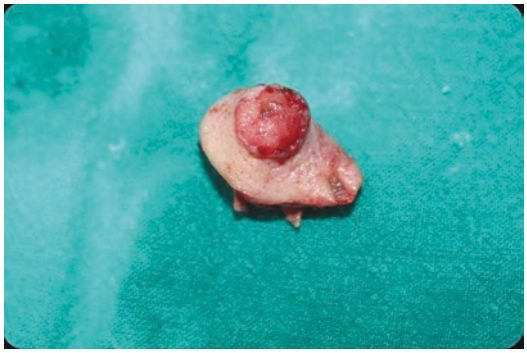

Fig. 10 A complete three-dimensional R0 resection was done

Fig. 11 Excision of the nasal septum

Fig. 12 A Paramedian forehead flap was planned

Fig. 13 The flap was bipaddled to create the nasal alar portion

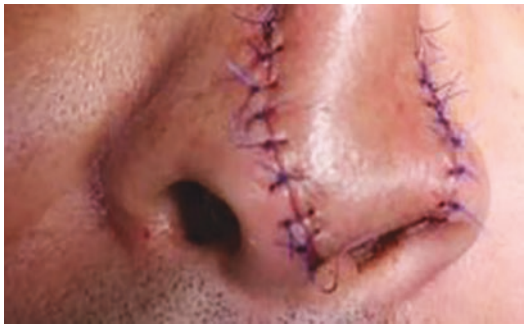

Fig. 14 Well-maintained contour of the ala of the nose

Fig. 15 Flap division was done at the third week

Fig. 16 A 6-month postoperative follow-up photograph showing excellent healing

7 Excision of Nasal Bridge Tumour with the Glabellar Flap

7.1 Case 3

A 48-year-old male presented with a 9-month history of growth on the nasal dorsum, gradually progressive. CECT was suggestive of the epithelial lesion without bony involvement. A preoperative biopsy was suggestive of BCC, and the final HPE report was also suggestive of BCC.

7.2 Operative Technique

The surgical steps are depicted in Figs. 17, 18, 19, 20 and 21.

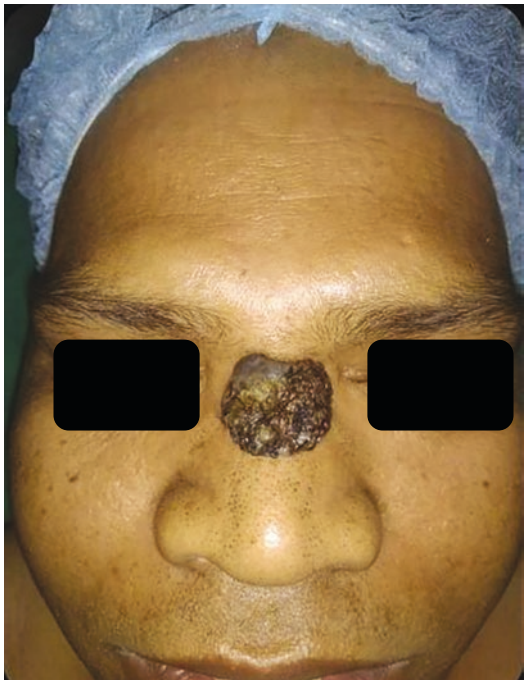

Fig. 17 A 2 × 3 cm pigmented ulceroproliferative growth with rolled edges on the nasal dorsum

Fig. 18 WLE was done with a 0.5 cm margin. The base of excision was up to the periosteum of the nasal bone

Fig. 19 A glabellar rotation flap was planned to close the defect, and one should note that the length of the flap reduces by 40% when it is rotated by 180 degrees

Fig. 20 Glabellar flap is raised and rotated to inset into the defect

Fig. 21 The forehead defect is closed primarily, and the inset is done without tension

8 Excision of Left Cheek Tumour with the Mustarde Flap

8.1 Case 4

A 65-year-old male presented with a 6-month history of gradually progressive growth on the left cheek. CECT was suggestive of the epithelial lesion without bony involvement. A preoperative biopsy was suggestive of BCC, and the final HPE was also suggestive of BCC.

8.2 Operative Technique

The surgical steps are depicted in Figs. 22, 23, 24, 25, 26 and 27. The postoperative follow-up of the patient in the sixth month is shown in Fig. 28.

Fig. 22 A 2 × 3 cm dark pigmented ulceroproliferative growth with rolled-up edges

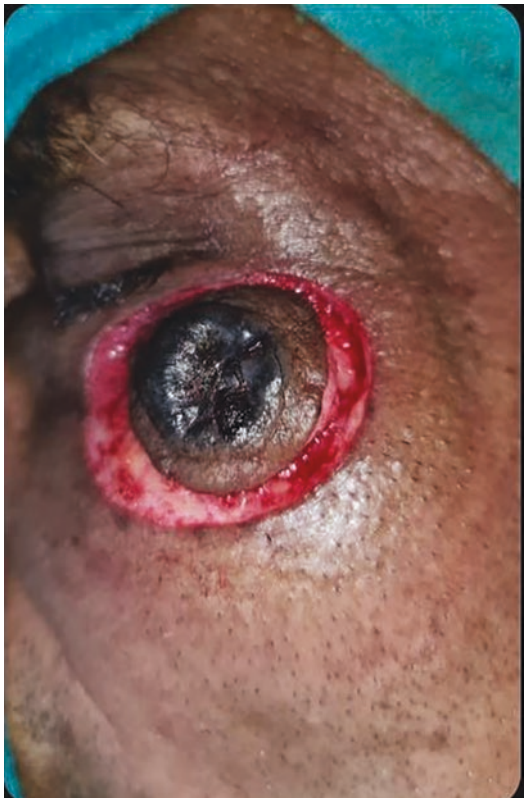

Fig. 23 The tumour was excised circumferentially, and the muscle was taken as the base. Indiscriminate cautery must be avoided as it may lead to inadvertent injury to the facial nerve branches

Fig. 24 The specimen shrinkage can be clearly seen. More shrinkage (up to 30%) is expected following formalin fixation

Fig. 25 The Mustarde flap was raised by staying in the supra-SMAS plane. The incision was planned along Pitanguy's line which is 0.5 cm inferior to the tragus to a point 1.5 cm superior and lateral to the eyebrow to preserve the temporal and frontal branch of facial nerve [4]

Fig. 26 The Mustarde flap is rotated onto the defect, and the incision can be extended inferiorly, if the flap does not reach the defect without tension

Fig. 27 Closure done

Fig. 28 A 6-month postoperative follow-up with no residual nerve weakness

9 Excision of Nasal Basal Cell Carcinoma with Forehead Flap Reconstruction

9.1 Case 5

A 63-year-old male patient presented with a 7-month history of gradually progressive growth on the nasal dorsum. CECT was suggestive of epithelial lesion with involvement of the lateral nasal cartilage. Preoperative biopsy was suggestive of BCC, and the final HPE report was also suggestive of BCC.

9.2 Operative Technique

The surgical steps are depicted in Figs. 29, 30, 31, 32, 33, 34 and 35.

Fig. 29 Extensive ulceroproliferative growth on the nasal dorsum

Fig. 30 Adequate margin all around the tumour

Fig. 31 Meticulous dissection was performed, and the muscle was taken as the base

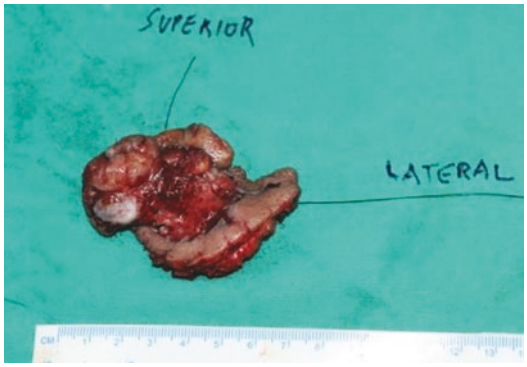

Fig. 32 The specimen must be oriented for accurate histopathological examination

Fig. 33 Post-resection defect with exposure of the left nasal cavity

Fig. 34 Full-thickness forehead flap was raised to close the primary surgical defect

Fig. 35 The donor defect was closed with full-thickness skin grafting. The nasopharyngeal airway was placed to prevent collapse and adhesion between the septum and reconstructed left nasal ala. Flap division was performed at 3 weeks

10 Excision of Temporal Region Tumour with Radial Free Forearm Flap

10.1 Case 6

A 69-year-old male presented with a 4-month history of gradually progressive growth on the skin over the right temporal region (Fig. 36). CEMRI was suggestive of epithelial lesion without bony involvement; no lymph nodes were found in the bilateral parotid glands and bilateral neck. A preoperative biopsy was suggestive of WDSCC, and the patient underwent circumferential three-dimensional resection preserving the zygomatic nerve to preserve eye closure. A free radial artery forearm flap was done to close the defect (Fig. 37). Following surgery, the patient received adjuvant RT as per the standard protocol.

10.2 Operative Technique

A postoperative 9-month follow-up shows a good flap uptake (Fig. 38).

Fig. 36 A 3 × 4 cm ulcerative lesion in the right temporal region. The lesion was just lateral to the lateral canthus

Fig. 37 A circumferential three-dimensional resection was done by preserving the zygomatic nerve to preserve eye closure. A free radial artery forearm flap was done to close the defect. The radial artery and its vena comitantes were anastomosed with the facial vessels

Fig. 38 A 9-month postoperative follow-up photograph showing good flap uptake and preserved eye closure

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Salivary Glands

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1 Parotid Gland Lesions

The most morphologically and clinically varied solid epithelial tumours are salivary gland neoplasms. The current World Health Organization (WHO) classification lists 25 various forms of salivary gland tumours (Table 1). Salivary gland neoplasms exhibit a diversity that is arguably unmatched by any other organ in comparison, notwithstanding their rarity. Less than 4% of all head and neck neoplasms are salivary gland neoplasms, making them rare. The parotid gland is where most salivary gland neoplasms are found, and in adults, 80% of these are benign. Recent years have seen a clearer definition of the cytologic characteristics of salivary gland neoplasms on fine-needle aspiration, making documenting and diagnosis easier. More and more often, salivary gland tumours are found to have genomic abnormalities, such as translocations and gene fusions.

Based on histopathologic traits and the extent of the tumour at presentation, salivary gland tumours are widely divided into benign and malignant categories. Grossly, benign tumours have well-defined borders, are thinly encapsulated and range in consistency from soft to somewhat firm. According to histology, these neoplasms typically have a consistent composition of myoepithelial and/or epithelial cells in a variety of forms. Malignant tumours, on the other hand, typically exhibit hard, less mobile, poorly defined and infiltrative characteristics. Tumours exhibit a variety of neoplastic cellular and structural characteristics histologically.

Table 1 Overview of the World Health Organization classification of benign salivary gland tumours (2017)

Benign tumours	Pleomorphic adenoma
	Myoepithelioma
	Basal cell adenoma
	Warthin tumour
	Oncocytoma
	Lymphadenoma
	Cystadenoma
	Sialadenoma papilliferum
	Ductal papillomas
	Sebaceous adenoma
	Canalicular adenoma and other ductal adenomas
Other epithelial lesions	Sclerosing polycystic adenosis
	Nodular oncocytic hyperplasia
	Lymphoepithelial lesions
	Intercalated duct hyperplasia
Soft tissue lesions	Hemangioma
	Lipoma/sialolipoma
	Nodular fasciitis
Borderline tumour	Sialoblastoma

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_4

1.1 Preoperative Evaluation

1.1.1 Ultrasonography

- Ultrasonography has the advantage of being inexpensive, non-invasive and simple to perform.
- It can be used to differentiate solid from cystic masses in the salivary glands.
- Ultrasound (US) guidance may also enhance the accuracy of FNAC in nonpalpable tumours and in masses with a heterogeneous architecture [1].
- A colour Doppler sonography has been used to evaluate the vascular anatomy of the salivary glands.
- It can distinguish between physiologic changes that occur during salivary stimulation in normal subjects and flow alterations that occur in diseased glands.
- Currently, the role of ultrasonography is limited and is generally used to determine the presence of hypo- or anechoic collection from a solid mass.
- It can also determine the presence of enlarged intraparotid nodes.
- The sensitivity of US is reported to range from 62% to 84%, the specificity from 88% to 96% and the accuracy from 57% to 96% [2].

1.1.2 Fine-Needle Aspirations

- Initial assessment of salivary tumours commonly entails a fine-needle aspiration cytology evaluation.
- The primary purpose of this procedure is to exclude metastasis, lymphoreticular disorders, infectious processes and reactive lesions and to ascertain the primary salivary nature of mass.
- FNAC, however, is limited in delineating benign and malignant nature of basaloid and oncocytic and myoepithelial and of carcinoma ex pleomorphic adenoma.
- The procedure is also valuable as a follow-up tool for harvesting cells for ancillary testing.
- The overall sensitivity ranges from 85.5% to 99%, and the overall specificity ranges from 96.3% to 100% [3].
- Seeding along the needle track after FNAC or core needle biopsy of a lump in the head and neck is rarely reported, and an accurate estimate of its incidence is difficult to ascertain. Crude estimates suggest 0.00012% and 0.0011% after FNAC and core needle biopsy, respectively [4].
- The *Milan System for Reporting Salivary Gland Cytopathology* is shown in Table 2.

1.1.3 Core Biopsy

- Occasionally, core biopsy is performed for salivary tumour diagnosis; however, this procedure should be limited to non-resectable, recurrent and metastatic tumours.
- Open biopsy is rarely indicated but can be considered in (1) skin-invading tumours where an incisional biopsy is in the area where the skin will have to be resected anyway in an eventual subsequent extended parotidectomy and in (2) advanced tumours that, at presentation, are already beyond surgical cure, so the

Table 2 The Milan system for reporting salivary gland cytopathology [5]

Category	Diagnosis	Risk of malignancy	Management
1	Nondiagnostic	25%	Clinical and radiologic correlation or repeat fine-needle aspiration (FNA)
2	Non-neoplastic	10%	Clinical follow-up and radiologic correlation
3	Atypia of undetermined significance (AUS)	10%–35%	Repeat FNA or surgery
4	Neoplasm		Surgery or clinical follow-up
4a	Benign	<5%	
4b	Salivary gland neoplasm of uncertain malignant potential (SUMP)	35%	
5	Suspicious for malignancy	60%	Surgery
6	Malignant	90%	Surgery

biopsy may be the only tissue sample obtained. (3) Increasingly, molecular biological studies can be carried out, also on the incisional biopsy material, especially when lymphoma is suspected.

1.1.4 Magnetic Resonance Imaging

- Magnetic resonance imaging (MRI) is the modality of choice for staging salivary gland tumours due to its optimal soft tissue contrast.
- High-resolution turbo-spin-echo T1-weighted (T1W) and T2-weighted (T2W) sequences, in addition to post-contrast images with fat saturation (FS), are the essentials for the assessment of salivary gland neoplasms [6].
- T1W sequences provide excellent information regarding tumour margins, tumour deep extension and pattern of infiltration. In addition, post-contrast T1W images with fast spin (FS) technique are useful to best address bone invasion, meningeal infiltration or perineural spread [7].
- T1W fat-saturated sequences of the bone marrow, the cortex and the skull base will have suppressed signal compared to the hyperintense, enhancing tumoural lowing for the detection of bony and meningeal invasion as well as tumour spread along the facial and trigeminal nerves (up the stylomastoid foramen, the foramen ovale and the foramen rotundum).
- T2W images have been reported to be helpful in predicting whether a salivary gland neoplasm is benign or malignant.
- A mass with low-to-intermediate signal intensity on T2W images is associated with malignancy, while hyperintense masses on T2W images might be considered benign. An exception is represented by malignant tumour that shows cystic/necrotic changes, which may be a confounding factor with benign ones.
- Flow void in the retromandibular vein is taken as a surrogate radiological marker to identify the plane of the facial nerve.
- MRI sensitivity and the specificity in predicting malignancy were reported to be 70% and 73%, respectively, and MRI sensitivity for perineural spread has been reported to be up to 95% [6].
- A tumour characterized by high cellular density shows high signal on DWI sequence. The signal on DWI sequence can be quantitatively expressed by means of apparent diffusion coefficient (ADC).
- An overlap in terms of ADC values has been reported between a large part of low-/high-grade malignant neoplasm and benign lesion [8].

1.1.5 Computed Tomography

- Computed tomography (CT) is the preferred modality in patients with MR contraindication (claustrophobia, cardiac pacemakers, metallic devices) or when further information about bone structures is required.
- A satisfactory CT examination should always provide images with thin slices (up to 1 mm) and multiplanar reconstruction with bone and soft tissue algorithms.
- It is well known that soft tissue contrast of CT images is lower compared to MRI.
- Perineural spread could be suspected only in the case of skull base foramina asymmetry, due to cortical erosion consequent to neural thickening.
- The use of iodinated contrast medium is mandatory to increase soft tissue resolution and depict pathology with better accuracy.
- CT scans are more widely available and easier to perform, compared to MRI, due to shorter time of acquisition.

1.1.6 Positron Emission Tomography

- An increase of fluorodeoxyglucose (FDG) uptake, expressed by standardized uptake value (SUV), is associated with cell vitality and proliferative activity.
- Positron emission tomography (PET)-CT is not a useful imaging method for distinguishing between benign and malignant tumours.
- FDG PET-CT might be useful in the detection of cervical lymph nodes and distant metastases in patients with high-grade tumours.

- The use of hybrid PET/MRI images might offer a higher sensitivity and specificity to assess the presence of malignancy on initial staging [9].

1.2 Principles of Treatment

- Management of tumours of the salivary glands requires a detailed understanding of the anatomy and pathologic processes that affect these glands.
- Benign salivary gland tumours should be excised completely with an adequate margin to avoid local recurrences.
- Generally, tumours in the parotid gland are removed with an adequate cuff of the surrounding normal tissue, and the facial nerve is dissected and carefully preserved.
- The malignant nature is frequently unclear before surgery but is suggested in many other instances by clinical signs, rapid growth, enlarged neck lymph nodes (29%), deep fixation or skin invasion (9%), pain (44%) and CN VII dysfunction (19%), a finding independent of the tumour diameter [10].
- The extent of surgery of the primary tumour is determined by the size of the lesion, the relationship to the facial nerve and eventual extraparotid tissue invasion.
- Initially, Tweedie and Jacob had proposed a classification system for parotidectomies that included total parotidectomy with or without facial nerve dissection, complete superficial parotidectomy, partial superficial parotidectomy (involving the upper, middle or lower segment), selective deep lobe parotidectomy and extracapsular dissection [11].
- The different types of parotidectomies that are classically described all have identification of the facial nerve—and preservation if possible—as a common principle:
  - Partial superficial parotidectomy implies resection of the tumour with a cuff of the normal tissue where possible.
  - Superficial or lateral parotidectomy implies removal of all tissue lateral to the facial nerve.
  - Total parotidectomy implies removal of all tissue lateral and medial to the facial nerve.
  - Radical parotidectomy implies all tissue, including the nerve.
  - Extended parotidectomy implies a radical parotidectomy with adjacent invaded structures such as the skin, bone of the mastoid or mandible, temporomandibular joint, masticatory muscles and infratemporal fossa.
- The ESGS classification for partial parotidectomies includes the following: superficial superior (I), superficial inferior (II), deep inferior (III), deep superior (IV) and accessory (V) [12].
- The parotidectomy performed is described by an enumeration of the levels resected in combination with additional non-parotid structures that are sacrificed. These are represented by capital letters placed in between brackets after the resected levels (CN VII, facial nerve trunk and/or all the main branches; CN VII t-z-b-m-c, when only facial nerve branches have been resected; ECA, external carotid artery; GAN, greater auricular nerve; LTB, lateral temporal resection; MB, mastoid bone; MM, masseter muscle; S, skin).
- Katz and Catalano described the common variations in facial nerve anatomy within the parotid gland. Approximately 24% of patients had a straight branching pattern (type I), 14% of patients had a loop involving the zygomatic division (type II), 44% of patients had a loop involving the buccal division (type III), 14% of patients had a complex pattern with multiple interconnections (type IV) and 3% of patients had two main trunks, one major and one minor (type V) [13].

1.3 Superficial Parotidectomy

1.3.1 Case 1

A 34-year-old female presented with a 3-year history of left parotid gland swelling. On examination - 2x3 cm firm and partially mobile swelling in the left parotid gland. FNAC—Milan IVa—Pleomorphic adenoma.

1.3.2 Operative Technique

- Under GA. Without long-acting paralytic agents, short acting—20 min.
- The head and thorax elevated at least 20 degrees—venous return and reduce congestion. The head is rotated to the opposite side and slightly extended. Table tilt by 10 to 15 degrees. 1:1 lakh saline Adr to help in hydrodissection and vasoconstriction. Shaving—minimal to no shaving; ladies hair knot. Eye ointment—vertical taping; facial muscles exposed.
- Marking of skin incision (Fig. 1).
- Flap elevation must be superficial to SMAS and superficial cervical fascia (Fig. 2). Supra-SMAS adipose tissue is easier and can have less bleeding. Sub-SMAS—better flap vascularity and lower Frey's but higher chances of VIIn Injury.
- While dissecting the flap, avoid monopolar surgery to restrict the thermal spread. The dissection must be parallel to the nerve. The skin flaps must not be raised too anteriorly or at the level of masseter. Inferiorly, it must be elevated along the planes of platysma.
- Stay suture and lobule stitch will improve the exposure. Posterior border of the gland is dissected off from sternocleidomastoid (SCM) muscle, and the posterior belly of digastric muscle is exposed. The GAN especially the posterior division must be preserved, as it supplies the skin over the pinna and mastoid area. Early ligation of EJV and RMV may increase venous pressure and cause bleeding.
- Dissection is carried along the cartilaginous external auditory meatus, the cartilaginous tragal pointer is identified and the parotid gland is retracted anteriorly. From here it is helpful to continue dissection in a plane parallel to the floor; a more perpendicular course will lead to the styloid, deep to the facial nerve.
- The parotidomasseteric fascia is incised. This exposes the parotid tissue and allows access to the plane in which the nerve courses.
- Palpation of the tympanomastoid suture line, which is located between the tragal pointer and mastoid tip, helps identify the spot at which the nerve will enter the gland parenchyma (large tumours may displace the main trunk in a more vertical direction, paralleling the course of the sternocleidomastoid (SCM)). Staying over the suture line and carefully dividing small pieces of the fascia and parotid are very reliable methods of exposing the facial nerve.
- Identification of the posterior belly of the digastric muscle is also helpful, as the stylomastoid foramen lies just medial to its attachment to the mastoid and facial nerve course is posterosuperior to its attachment.
- Retrograde dissection and tracing of the marginal mandibular nerve to the main trunk are seldom used but are reliable techniques used

Fig. 1 The modified Blair/Bailey incision is most commonly used. The classic modified Blair incision is marked in the preauricular crease at the level of the meatus, behind the lobule and down into a neck crease

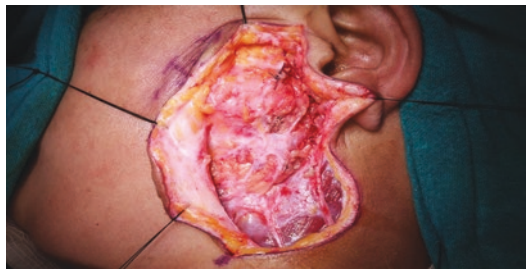

Fig. 2 Flaps—raised in the subcutaneous adipose tissue, avoiding injury to the hair follicles—Frey's syndrome. Avoid buttonhole. Identification of the greater auricular nerve. Scars must be included in the incision especially in the reoperations. Skin flap at the mastoid portion must be over 2 cm; else it leads to necrosis. Rhytidectomy incision and facelift have a cosmetic approach; they utilize the SMAS as the plane of dissection. Yellow arrow, greater auricular nerve; blue arrow, parotid tumour

to identify the main trunk of the nerve when the standard approach is not possible.
- Once the nerve has been identified, dissection continues along the main trunk and branches (Figs. 3, 4, 5, 6, 7 and 8).
- To avoid injury to the nerve, the soft tissue should be lifted, spread and then cut.
- The parotid lateral to the visible portion of the nerve is divided.

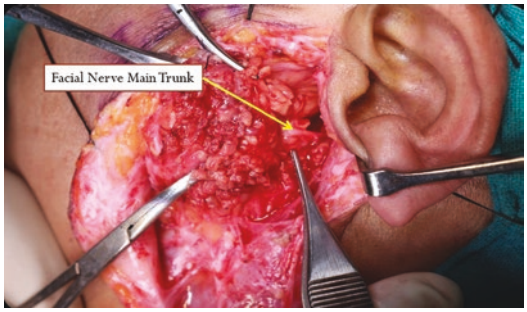

Fig. 3 Identification of facial nerve main trunk

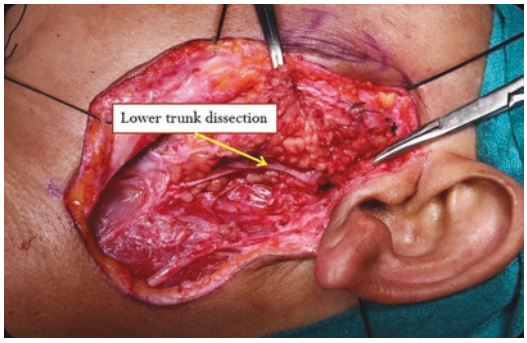

Fig. 4 Proceeding to the lower trunk dissection

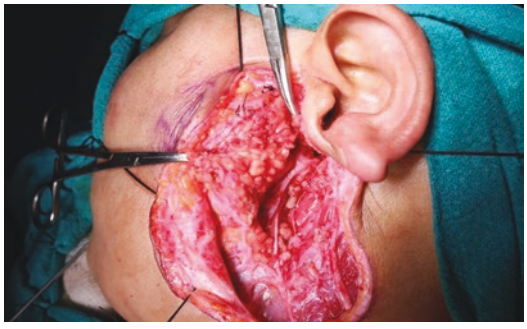

Fig. 5 After dissecting the lower trunk, the tumour with the gland is mobilized superiorly to expose and dissect the upper trunk

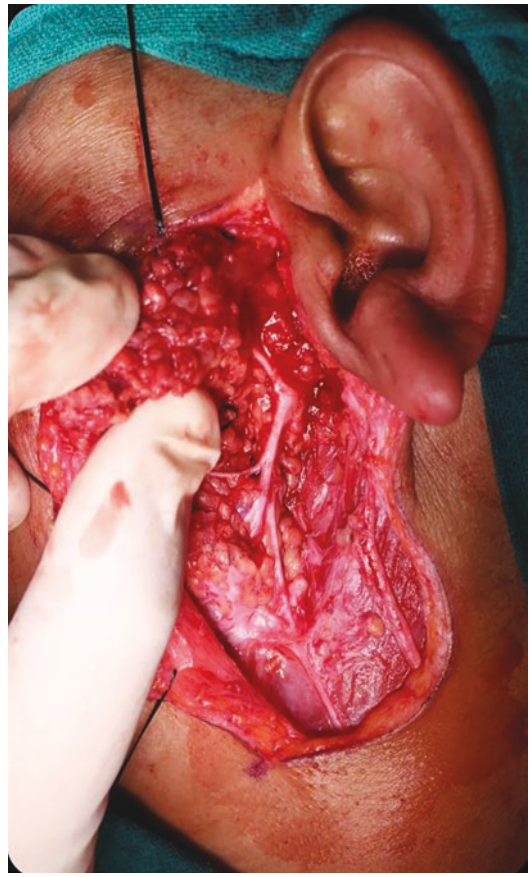

Fig. 6 Upper trunk dissection

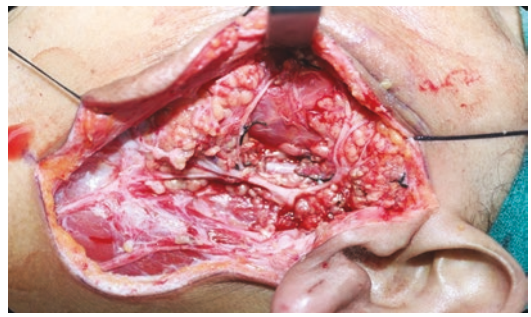

Fig. 7 The retromandibular vein can be clearly seen medial to the facial nerve trunk

- Overstimulation of the nerve is to be avoided. Overuse of the facial nerve stimulator can cause praxis of the nerve, as can improper dissecting technique. The tissues over the nerve should be spread apart in a plane parallel to the nerve fibres; any angulation away from

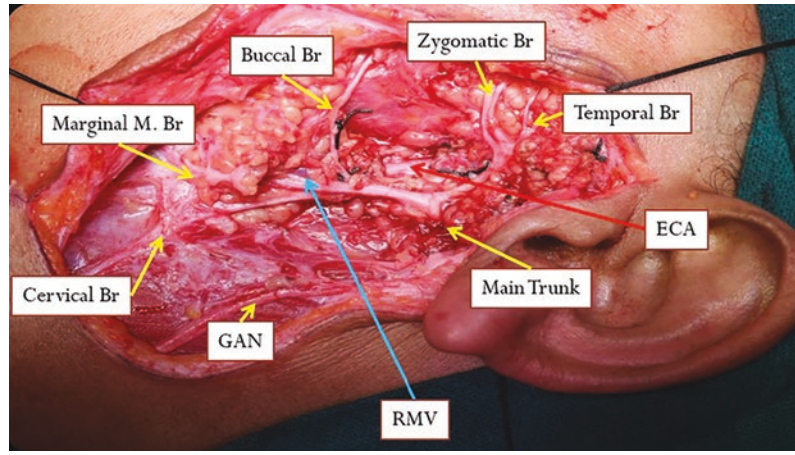

Fig. 8 Facial nerve trunk following completed superficial parotidectomy

this plane may cause undue trauma to the nerve.

- Extracapsular dissection descended from the technique of intracapsular enucleation, which was performed up to the 1960s. It was intended to decrease injuries to the facial nerve but resulted in recurrence rates of over 40% due to incomplete resection of the capsule.
- Benign parotid neoplasms also require excision of a small cuff of the normal gland.
- However, the deep portion of the dissection of the facial nerve often yields no margin of the tissue other than the tumour capsule itself.
- Careful dissection of the tumour away from the nerve is necessary to avoid injury to the facial nerve and complete excision of the capsule.
- Once the tumour has been dissected off the facial nerve beyond the anterior limit of the lesion, the parotid is divided, and the specimen is removed.
- In cases of malignancy, particularly low-grade tumours, the extent of parotidectomy may be similar to that involved in the resection of benign tumours.
- Suture ligation of small vessels or bipolar cautery is used to avoid injury to the nerve.
- Dexamethasone instilled locally over the facial nerve had been found to reduce the facial nerve paresis postoperatively.
- Insertion of a suction drain is useful after the wound is irrigated (Fig. 9).

Fig. 9 The skin closed in layers with closed suction drain

- Defects resulting in obvious contour hollowing can be repaired by:
  - Approximating the SMAS to the SCM or creating a small rotation flap by dividing some of the superior fibres of the SCM and suturing them anteriorly. Care must be exercised to avoid injury to the spinal accessory nerve.
  - The use of allogenic dermal grafts. These have been reported to be safe, but this avascular foreign body has also been found to promote infection and salivary fistula.
  - Insertion of adipose tissue grafts, which are limited by reabsorption and a separate donor site.
- Subcutaneous layer approximated with an interrupted suture (Fig. 10).
- Steri-Strips are applied, followed by a sterile parotid dressing.

Fig. 10 A postoperative 4-week, well-healed scar

Fig. 12 Arrow pointing towards the swelling

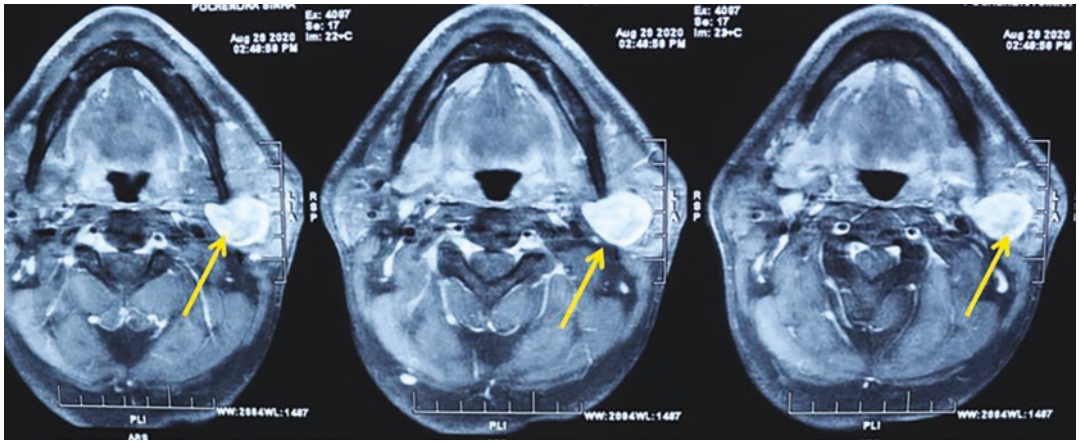

Fig. 11 A 2.2 × 3.1 cm benign deep lobe of the parotid neoplasm

The fourth-week postoperative follow-up shows a barely visible surgical scar (Fig. 10).

1.4 Trans-parotid Deep Lobe Tumour Excision

1.4.1 Case 2

A 55-year-old male presented with a 4-year history of left cheek swelling. The MRI showed a 2.2 × 3.1-cm-deep lobe of parotid neoplasm (Fig. 11). FNAC—Milan IVa—Pleomorphic adenoma.

1.4.2 Operative Technique

All operative steps are depicted in Figs. 12, 13, 14, 15, 16, 17, 18, 19 and 20.

1.5 Radical Parotidectomy with SND (II–V)

1.5.1 Case 3

A 64-year-old female presented with a 12-year history of left cheek swelling, increase in its size since 1 year and facial asymmetry since 1 month. On examination a 6 × 8 cm hard mass in the left

Fig. 13 Modified Blair incision

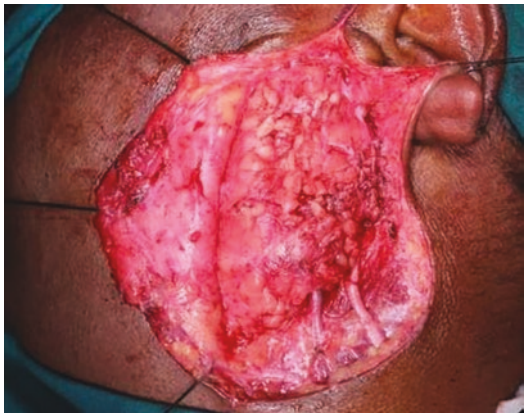

Fig. 14 Skin flap elevation and identification of GAN (yellow arrow) and EJV (green arrow)

parotid region, fixed to the underlying masseter muscle. Left VII N—HB grade III. FNAC—Milan V—Suspicious of mucoepidermoid carcinoma.

1.5.2 Operative Technique

All operative steps are depicted in Figs. 21, 22, 23, 24, 25, 26, 27, 28, 29 and 30.

1.6 Extended Radical Parotidectomy

1.6.1 Case 5

A 57-year-old female presented with a 4-year history of progressive right parotid swelling and

Fig. 15 Excision of the superficial parotid tissue to expose the deep lobe of the parotid gland

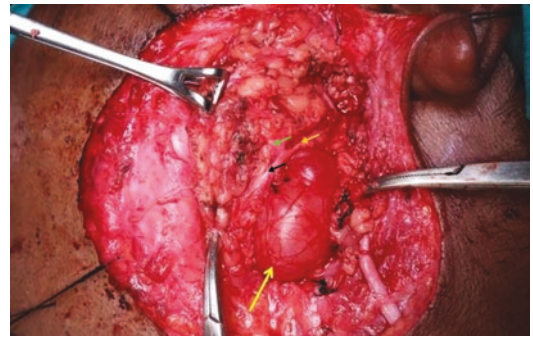

Fig. 16 Mobilization of the deep lobe tumour by blunt dissection. Small yellow arrow, common facial trunk; green arrow, upper division; black arrow, upper division

rapid increase in size (2 months). On examination a 6 × 8 cm right parotid mass, skin involved, masseter involved (Fig. 31). FNAC—(1) Milan IVB—SUMP. (2) Milan IVA—Pleomorphic Adenoma.

Intraop Frozen suggestive of High-grade malignancy with skin invasion.

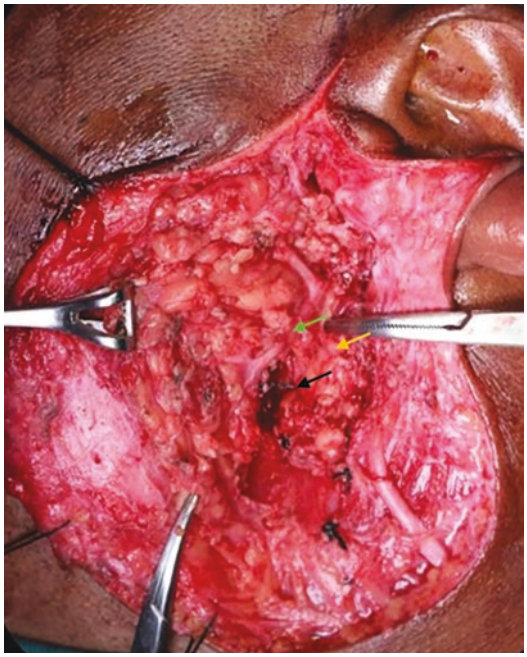

Fig. 17 Preserved facial nerve trunk and its branches following deep lobe tumour excision. Yellow arrow, common facial trunk; green arrow, upper division; black arrow, upper division

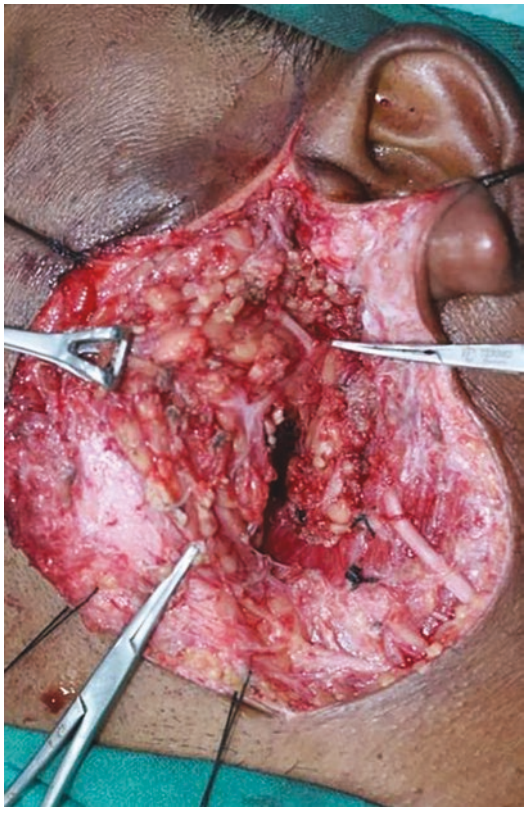

Fig. 18 Post resection

Fig. 19 Excised the superficial parotid lobe

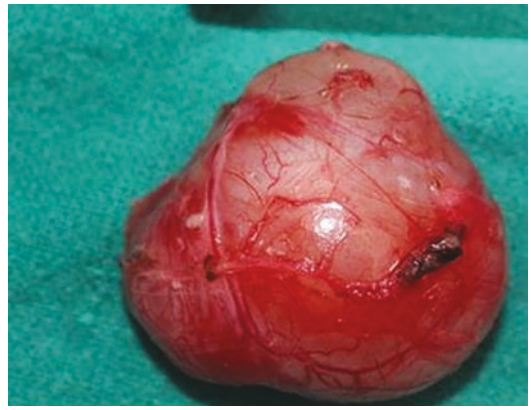

Fig. 20 Excised well-capsulated deep lobe tumour

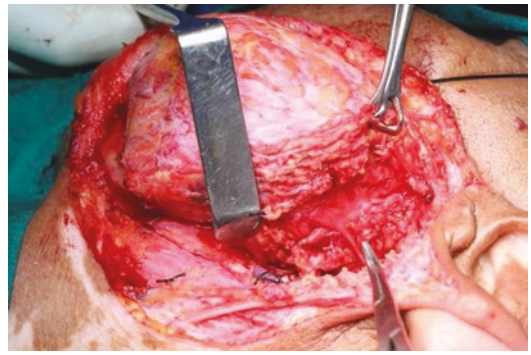

Fig. 21 Identification of the main trunk of the facial nerve following skin flap elevation separating the parotid gland from SCM identification of the posterior belly of the digastric muscle

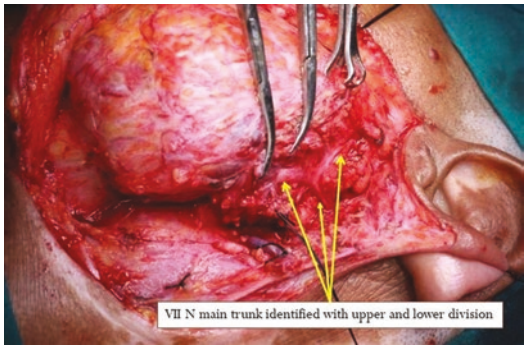

Fig. 22 Further dissection of the facial nerve trunk to expose the upper and lower divisions of the facial nerve

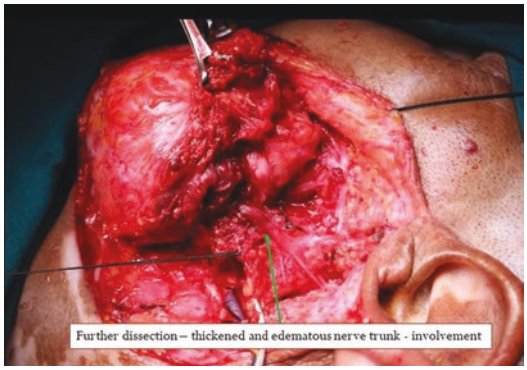

Fig. 23 The nerve trunk is thickened and oedematous suggesting nerve infiltration

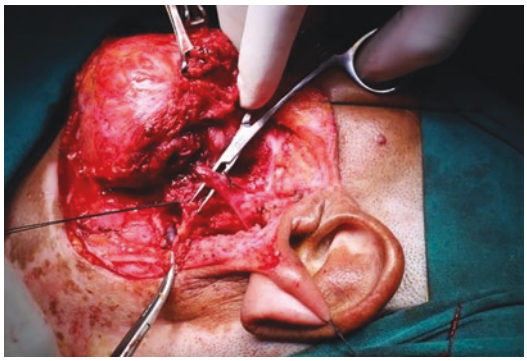

Fig. 24 Facial nerve sacrifice due to involvement (for oncological reasons)

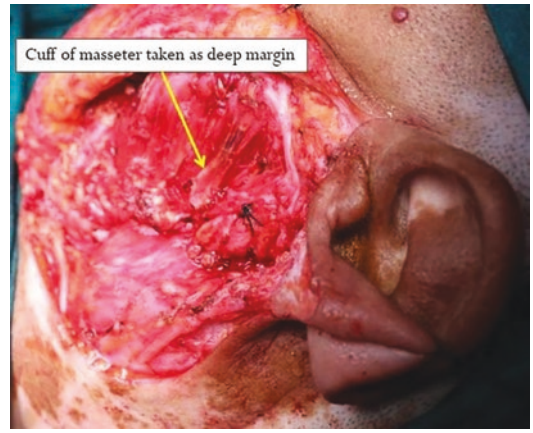

Fig. 25 A cuff of the masseter muscle was taken as the deep margin

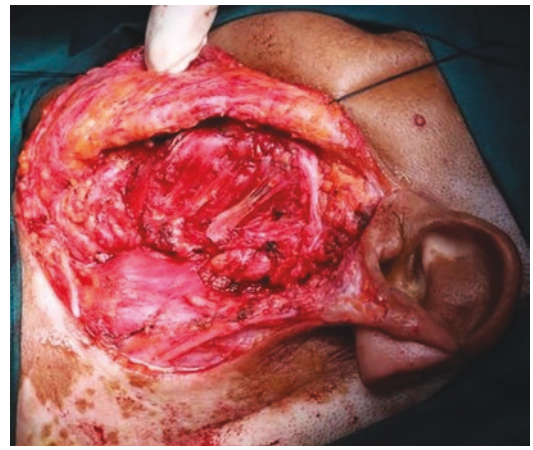

Fig. 26 Post-resection bed

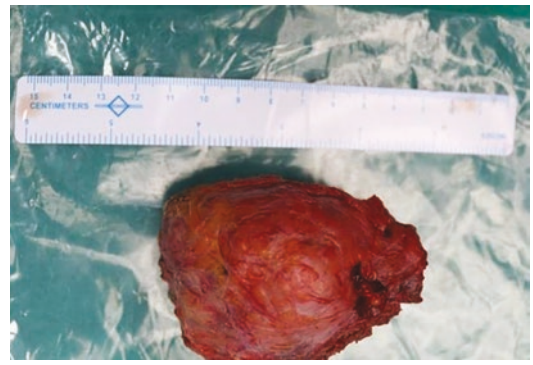

Fig. 27 Specimen of radical parotidectomy

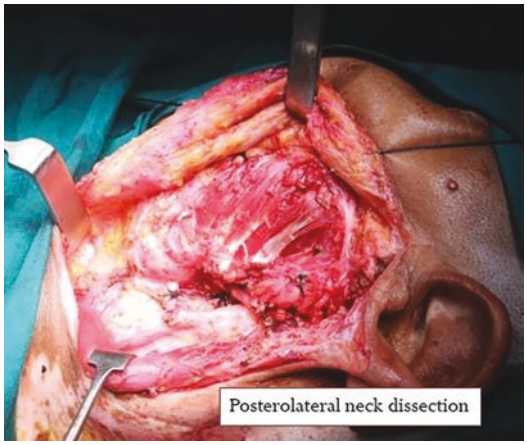

Fig. 28 A posterolateral neck dissection (II–V) was done

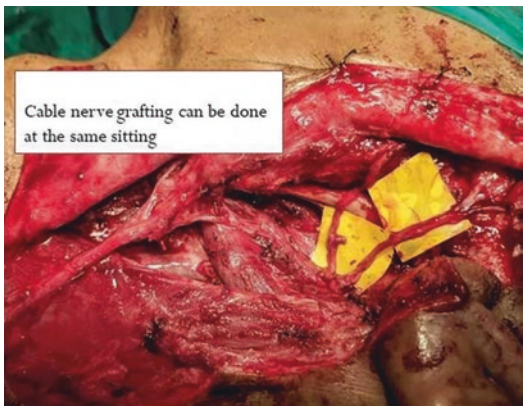

Fig. 29 A cable nerve grafting was performed at the same setting with GAN

Fig. 30 Wound closure and drain in situ

Fig. 31 Incision planning done as per the extent of the tumour. The involved skin was marked for excision along with the lesion

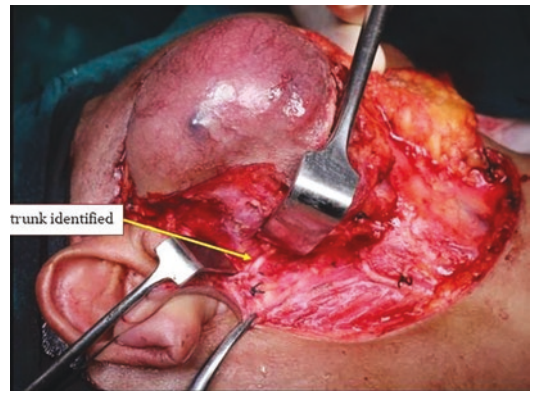

Fig. 32 The nerve trunk was identified

1.6.2 Operative Technique

All operative steps are depicted in Figs. 32, 33, 34, 35, 36, 37, 38, 39 and 40.

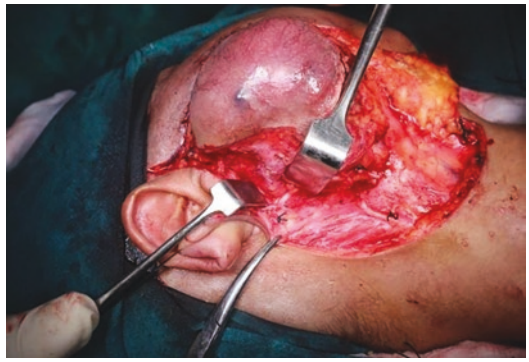

Fig. 33 Complete encasement of the nerve trunk can be seen

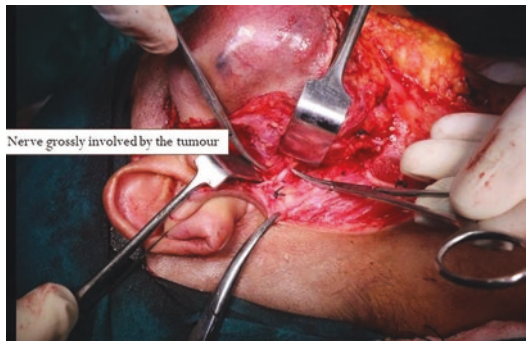

Fig. 34 The nerve is grossly encased by the tumour, and a decision to sacrifice the nerve was taken

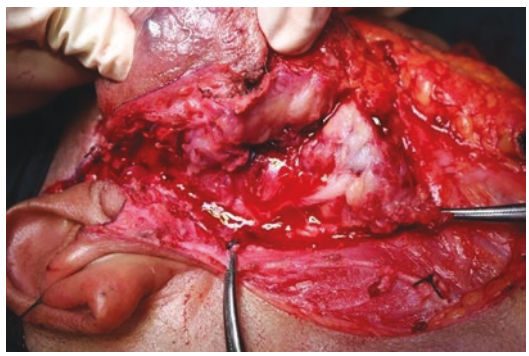

Fig. 35 The tumour was dissected circumferentially

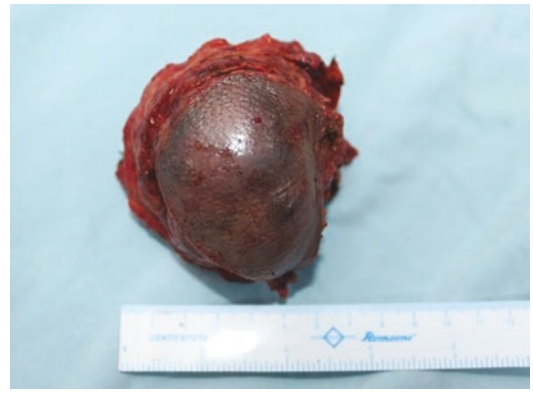

Fig. 36 Specimen with the involved skin

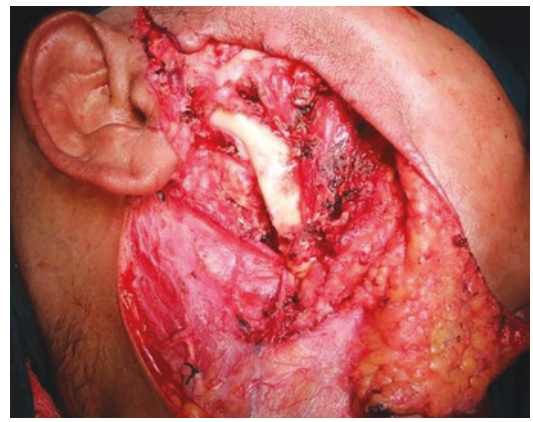

Fig. 37 Extended radical parotidectomy defect. The mandible and parapharyngeal space are exposed

Fig. 38 Masseter muscle was released and used to close the retromandibular hollow and parapharyngeal space

Fig. 39 A cervicofacial rotation flap was done to close the skin defect

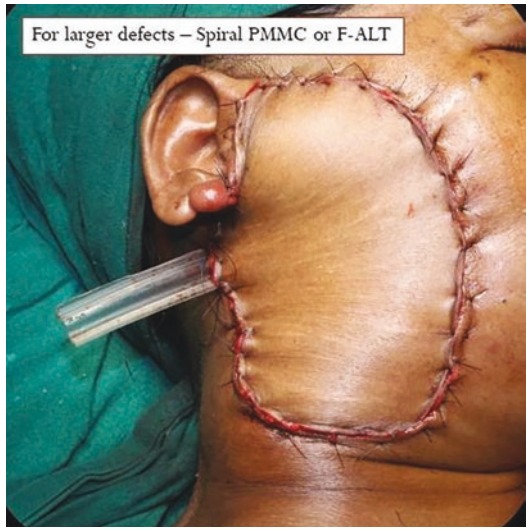

Fig. 40 A larger defect can be closed with free flaps/pectoralis major myocutaneous flap

1.7 Anticipated Complications: Mitigation and Management

1.7.1 Injury to the Facial Nerve

- Mild neuropraxia resolves in 4–6 weeks.
- More severe traction injuries resulting in axonal death will take much longer to recover (6 months); imperfect recovery leads to permanent weakness and synkinesis.
- Transected nerve fibres should be repaired if possible.
- Nerve ends should be examined and freshened by excising a small amount of the nerve to create a blunt edge.
- Epineurium is reapproximated using an 8- or 9-0 monofilament suture; a neural tube can be placed to support the repaired nerve.
- If a nerve graft is required, the greater auricular is readily available.
- When the nerve is repaired in a second stage, the sural nerve or medial branchial cutaneous nerve is a good graft choice.
- If the nerve was normal preoperatively and a microscopic repair is performed immediately, the best result possible is a House-Brackmann grade III. Recovery takes about 6–12 months.

1.7.2 Facial Nerve Function

- If facial paresis is found but the nerve was found to be intact intraoperatively, a full recovery of function is expected.
- High-dose steroids are generally not indicated as they do not improve the outcome.
- Reoperation is indicated only if the nerve was not identified during the dissection. In this case attempts to find and repair the nerve should be made.

1.7.3 Drains and Dressing

- Drains are left to bulb suction to avoid inadvertent stress to the nerve.
- The drain is removed if total output is less than 20 mL in 24 h. After that point, the drain is removed if there is less than 5 mL of drainage over the next 8 h.

1.7.4 Hematoma

- Sudden facial swelling or rapid filling of the drain should be treated by exploration of the wound in the operating room.
- Evacuation of the hematoma is important in order to avoid wound infection and necrosis of the flap.

1.7.5 Frey Syndrome

- Occurs in up to 60% of parotidectomy patients, but only about 10% has been considered to have a negative impact on quality of life.

- Antiperspirant application or botulinum toxin injections are temporary management options.
- Long-term control involves placement of the vascularized tissue between the parotid bed and the skin.
- Intraoperatively, a flap can be created by lifting the SMAS off the gland, leaving it pedicled anteriorly, and then placing in the defect before closure.
- Revision surgery for Frey syndrome should be carefully considered because of the increased risk of facial nerve injury due to scarring.

1.7.6 Infection

- Occurs in less than 5% of cases.

1.7.7 Sialocele Formation and Seroma

- It is a very uncommon (approximately 5%) but persistent complication of superficial parotidectomy.
- The use of allogenic dermal grafts or Surgicel and anterior location of the tumour have been implicated as risk factors.
- Ballotable swelling noted at the postoperative check should be aspirated in the office. The skin is prepped to avoid the introduction of bacteria, and a large-bore (16–20 French) needle is used to aspirate the collection. Pressure dressings at this point confer little advantage. Repeat aspiration may be necessary, especially in cases of seroma formation.
- Expectant management is also reasonable, and most of these conditions resolve without treatment in 4–6 weeks.

1.7.8 Flap Necrosis and Hypertrophic Scars

- Flap necrosis is avoidable if the skin is elevated with the subcutaneous adipose tissue.
- Keloid formation may be seen early in the postoperative period and is best treated when first noted. Injection of triamcinolone directly into the scar is effective.
- Local anaesthesia with lidocaine prior to injection or mixed with triamcinolone is important, as hypertrophic scar injection is often painful.

1.7.9 Recurrent Tumour or Infection

- Occurs in 2% of cases.
- Incomplete capsule removal and, less commonly, tumour spillage are causative.
- If capsule rupture occurs, the wound is irrigated with copious amounts of saline and carefully inspected to remove all tumour fragments.
- Imaging and FNAC are required.
- The goal of revision surgery is resection of all tumour deposits, which may not be possible without sacrificing a portion of the facial nerve.
- Radiation is used in cases of widespread recurrence or multiple surgeries.

2 Submandibular Gland Lesions

The submandibular gland is one of three paired major salivary glands that drains into the oral cavity. It is midway in size and location between the largest, the parotid gland, and the smallest, the sublingual gland. Histologically, it consists of both serous and mucinous acini, which collectively drain into an excretory duct that carries the secreted saliva into the oral cavity. A mass in the submandibular gland or a minor salivary gland is more likely to be malignant. In a review of more than 2000 salivary gland tumour cases, 73% of the tumours were found in the parotid with only 15% found to be malignant, while 11% were found in the submandibular gland with 37% found to be malignant. Tumours of the sublingual gland are extremely rare and comprise 0.5–1% of all salivary tumours, and 80–90% are malignant [2, 14, 15].

It is imperative for the physician to distinguish a chronic benign process, such as sialadenitis, from a submandibular gland neoplasm, and then further determine if a neoplasm is benign or malignant. This is done through careful history and physical exam, as well as utilization of preoperative imaging and fine-needle aspiration.

Management of submandibular gland malignancy can be challenging due to the relative rarity of the disease and the diversity of its behaviour

due to a variety of histologic subtypes and grades. Adenoid cystic carcinoma is the most common subtype in the submandibular gland, followed by mucoepidermoid carcinoma and then adenocarcinoma [14, 16].

2.1 Preoperative Evaluation

2.1.1 Imaging

- Imaging for submandibular and sublingual gland lesions augments the physical exam and aids in the following: determining if a lesion is intrinsic or extrinsic to the gland, evaluating the extent of the lesion with respect to local invasion, establishing perineural involvement and determining if there is a metastatic disease.
- Although imaging lacks the specificity to determine benign from malignant tumours, a computed tomography (CT) scan with contrast can provide valuable information regarding mandibular bone invasion, the local extent of the tumour and the presence or absence of pathologic lymphadenopathy. Magnetic resonance imaging (MRI) can provide superior soft tissue detail to CT scans and can help assess perineural spread.
- PET-CT in salivary gland disease can help rule out distant metastasis if the primary cancer has enhanced fluorodeoxyglucose (FDG) uptake. In FDG-avid cancers, PET-CT may be useful in initial staging, in histologic grading and in monitoring for recurrence [17].

2.1.2 FNAC

- The role of preoperative fine-needle aspiration cytology (FNAC) as a diagnostic test is a vital part of the clinical management algorithm for submandibular gland disease. For inflammatory causes of salivary gland enlargement, nonsurgical management can often be used, or a simple submandibular gland excision may be planned.
- Differentiating between benign and malignant salivary gland tumours can be difficult with FNAC. With experienced cytopathologists, FNAC is accurate in over 90–95% of patients.

2.2 Principles of Treatment

- A complete excision of the gland must be done to avoid any residual disease.
- The marginal mandibular branch of the facial nerve lies within the operative field and must be either identified or avoided, depending on the situation.
- When excision of the gland is being performed for benign disease, identification of the nerve may not be necessary. In such cases, the nerve may be protected by ligation and elevation of the facial vein (Hayes-Martin manoeuvre) (Fig. 41).
- When the gland is removed for malignancy or in conjunction with a neck dissection, the marginal mandibular nerve must be identified to allow the removal of the perifacial nodes.
- Identification of the nerve will help avoid injury of the nerve posterior to the gland, where the nerve may often be located a significant distance inferior to the level of the mandible.
- In cases in which there is extensive inflammation and scar tissue, a handheld nerve stimulator may be useful to help locate and preserve the marginal mandibular nerve.
- The proximal stump of the facial artery should be double-ligated, and the wound examined for bleeding vessels before closure.
- Care must be taken in patients who have had significant infection and scarring to ensure

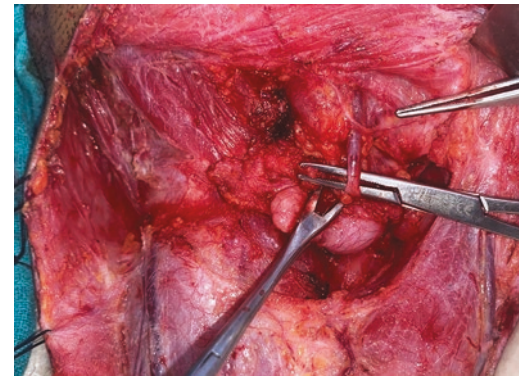

Fig. 41 The facial vein is medial to the marginal mandibular nerve

identification of the lingual nerve before sectioning Wharton's duct.
- Care should be taken after the gland is freed posteriorly to visualize the hypoglossal nerve as it courses deep to the vessels, parallel to the submandibular duct. If inflammation hinders identification of the nerve, it is advisable to find the nerve inferior to the posterior belly of the digastric muscle and follow it in a posterior-to-anterior direction through the submandibular triangle to avoid injury.

2.3 Excision of the Submandibular Gland

2.3.1 Case

A 42-year-old male presented with a 2-year history of swelling in the left submandibular fossa. FNAC was suggestive of Milan IVa—Pleomorphic adenoma, and the final histopathological examination (HPE) was suggestive of pleomorphic adenoma.

2.3.2 Operative Technique

- A curvilinear incision is placed, preferably in a natural skin crease, 3–4 cm below the lower border of the mandible to overlie the submandibular gland (Fig. 42).
- The incision is carried down through the subcutaneous fat and the platysma muscle. Great care should be taken to avoid injury of the marginal mandibular nerve (Fig. 43).
- The nerve lies immediately beneath the deep cervical fascia, and it can be identified crossing the anterior facial vein. The vein is doubly ligated and transected well below the nerve, and upward retraction of the superior ligature displaces the nerve superiorly and protects it from injury during further dissection.
- Superior dissection proceeds by double ligation and transection of the facial artery, which frees the superior attachment of the gland.
- Anteriorly, the vessels to the mylohyoid muscle are divided, and the gland is mobilized posteriorly to expose the free edge of the mylohyoid muscle (Fig. 44).
- The free (posterior) edge of the mylohyoid muscle is retracted anteriorly, while gentle posterior traction on the gland is maintained.
- This exposes the deep portion of the gland (Fig. 45) and its duct, the submandibular ganglion, and the lingual and hypoglossal nerves

Fig. 42 Left submandibular gland tumour along with the marking of the skin incision

Fig. 43 Elevation of the upper and lower skin flap

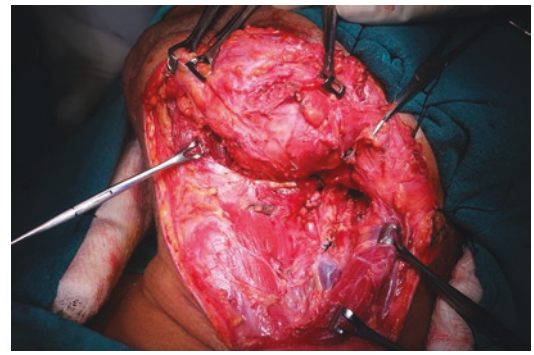

Fig. 44 Dissection all around the tumour to expose the anterior belly of the digastric muscle and the common tendon of the digastric muscle

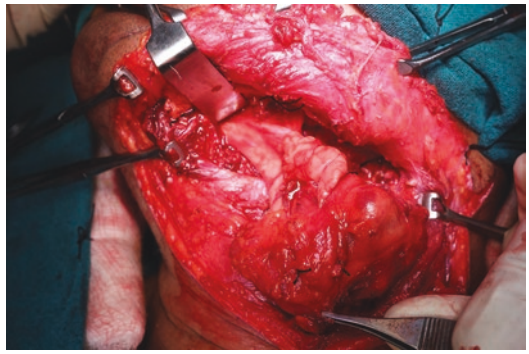

Fig. 45 Exposure of the deep lobe of the submandibular gland

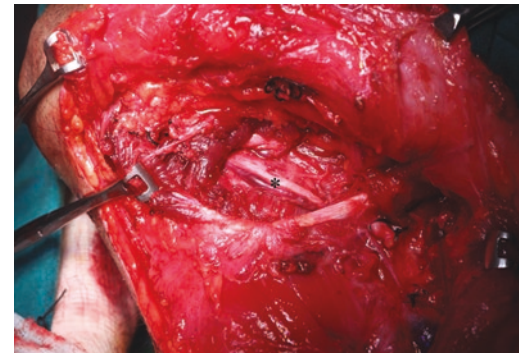

Fig. 47 Complete exposure of the tumour

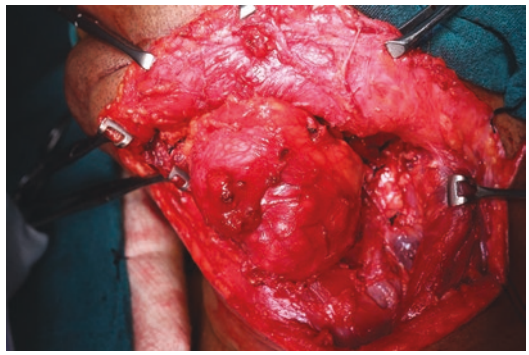

Fig. 46 Surgical bed following complete excision of the tumour. * shows the hypoglossal nerve

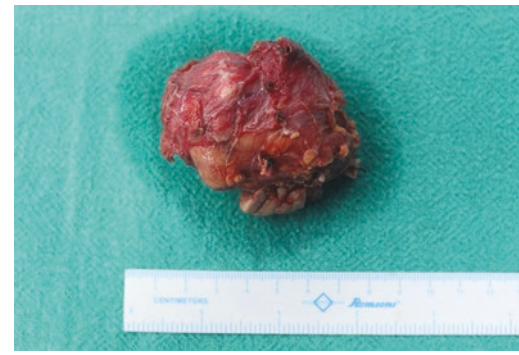

Fig. 48 Excised specimen of submandibular gland pleomorphic adenoma

(Fig. 46). These structures lie superficial to the hyoglossus muscle.

- The contribution of the lingual nerve to the submandibular ganglion is transected, and the Warthin duct is doubly ligated and divided.
- This delivers the deep portion of the gland. Care should be taken to avoid injury to the lingual and hypoglossal nerves. Finally, the facial artery is divided a second time, and the gland is removed (Fig. 47).
- In cases with tumours infiltrating the mandible, a reverse marginal mandibulectomy or a segmental mandibulectomy can be performed [18].

The excised specimen of the submandibular gland pleomorphic adenoma is shown in Fig. 48.

2.3.3 Anticipated Complications: Mitigation and Management

- The marginal mandibular nerve can be injured, which can result in paralysis of the lower lip, especially in patients with chronic inflammation of the gland where the anatomic planes of dissection are obliterated by inflammation and scarring.
- Failure to dissect Wharton's duct all the way to the mucosa of the floor of the mouth during excision of the submandibular gland can result in retained calculi with infection later on.
- Postoperative haemorrhage can lead to significant airway obstruction as a result of oedema of the tongue musculature.
- The lingual nerve can be injured by injudicious sectioning of the duct without first iden-

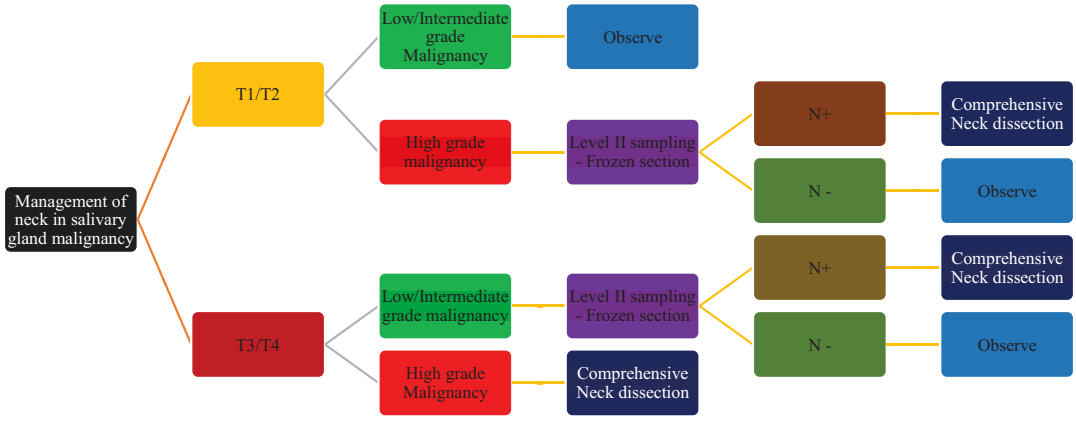

Fig. 49 Management algorithm for management of the neck in salivary gland malignancy

tifying and freeing the nerve where it lies directly superior to the duct.

- The hypoglossal nerve may be adherent to the medial aspect of the gland and inadvertently resected or transected.

2.4 Management of the Neck in Salivary Gland Malignancy

Based on the existing literature, a general outline for management of the neck in salivary gland tumours has been formulated and depicted in Fig. 49 [19–22].

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Thyroid Gland

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Thyroid diseases are common. They occur in the form of abnormalities in the size and shape of the thyroid gland (goiter) and as abnormalities of thyroid secretion. Nonthyroidal illness can be accompanied by any alteration in thyroid physiology that can complicate the evaluation of thyroid status. Thyroid nodules are common and may be found in up to two-thirds of the population [1]. Thyroid nodules are discrete lesions contained within, yet radiologically distinct from, the parenchyma of the thyroid gland [2]. A substantial increase in both thyroid nodule detection and thyroid cancer detection has occurred over the past three decades; this is largely attributable to increases in healthcare access/utilization and the use of medical imaging [3]. Despite increased detection of thyroid nodules and malignancies, thyroid cancer mortality has remained low and virtually unchanged [4]. Approximately 25% of the new thyroid cancers diagnosed in 1988–1989 were ≤1 cm compared with 39% of the new thyroid cancers diagnosed in 2008–2009. This tumour shift may be due to the increasing use of neck ultrasonography or other imaging and early diagnosis and treatment, trends that are changing the initial treatment and follow-up for many patients with thyroid cancer [5, 6]. The WHO classification scheme of thyroid neoplasms, fifth edition (2022) is given in Table 1. Differentiated thyroid cancer (DTC), which includes papillary and follicular cancer, comprises the vast majority (>90%) of all thyroid cancers [7]. These tumours have a propensity for multifocality and regional lymph node metastasis but are usually associated with an excellent prognosis. Around 10–25% of these tumours display aggressive behaviour, characterized by local invasion, distant metastasis, treatment resistance and increased mortality [8–11]. The incidence of thyroid cancer is not accurate as tertiary referral centres that see a larger number of malignancies have a higher incidence as compared to other centres. The incidence of early thyroid cancers is much higher in comparison with the thyroid cancer, probably attributable to the early detection and improved diagnostic modalities [4].

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_5

Table 1 WHO classification scheme of thyroid neoplasms, fifth edition (2022)

Developmental abnormalities
1. Thyroglossal duct cyst
2. Other congenital thyroid abnormalities
Follicular cell-derived neoplasms
1. Benign tumours
(a) Thyroid follicular nodular disease
(b) Follicular adenoma
(c) Follicular adenoma with papillary architecture
(d) Oncocytic adenoma of the thyroid
2. Low-risk neoplasms
(a) Non-invasive follicular thyroid neoplasm with papillary-like nuclear features
(b) Thyroid tumours of uncertain malignant potential
(c) Hyalinizing trabecular tumour
3. Malignant neoplasms
(a) Follicular thyroid carcinoma
(b) Invasive encapsulated follicular variant papillary carcinoma
(c) Papillary thyroid carcinoma
(d) Oncocytic carcinoma of the thyroid
(e) Follicular-derived carcinomas, high-grade
• Differentiated high-grade thyroid carcinoma
• Poorly differentiated thyroid carcinoma
(f) Anaplastic follicular cell-derived thyroid carcinoma
Thyroid C cell-derived carcinoma
1. Medullary thyroid carcinoma
Mixed medullary and follicular cell-derived carcinomas
Salivary gland-type carcinomas of the thyroid
1. Mucoepidermoid carcinoma of the thyroid
2. Secretory carcinoma of salivary gland type
Thyroid tumours of uncertain histogenesis
1. Sclerosing mucoepidermoid carcinoma with eosinophilia
2. Cribriform morular thyroid carcinoma
Thymic tumours within the thyroid
1. Thymoma family
2. Spindle epithelial tumour with thymus-like elements
3. Thymic carcinoma family
Embryonal thyroid neoplasms
1. Thyroblastoma

1 Preoperative Evaluation

1.1 Clinical Pointers

A high index of suspicion is necessary to identify patients with thyroid cancer preoperatively. If diagnosed preoperatively, this would allow for better planning of treatment strategies. Patients with invasion of the aerodigestive tract may present with specific symptoms that point towards the site of invasion (Table 2). Initial findings at presentation will include a palpable neck mass (98–100%), hoarseness (18–22%), dysphagia (25%), haemoptysis (11–25%) or dyspnoea (5–33%) [14, 15]. Many patients presenting with a paralyzed vocal cord may present without acute voice changes due to gradual compensatory function by the contralateral vocal cord [12].

The data on the incidence of each subsite involvement was given by Thomas McCaffrey of Mayo Clinic in 1994 [16]. In order of prevalence, the muscles (strap muscles and sternocleidomastoid muscles) (53%), recurrent laryngeal nerve (47%), trachea (37%), oesophagus (21%), larynx (12%) and other sites (jugular vein, carotid artery or prevertebral fascia) (30%) were found to be involved.

On clinical examination, most patients with invasive thyroid cancer will present with a palpable neck mass, but this has a low sensitivity in indicating an invasive cancer. A fixed cervical mass or frank adherence or invasion of the cervical skin can suggest locally advanced disease. This lack of accuracy in predicting thyroid cancer on examination of the neck highlights the need for a routine fiberoptic scopy or indirect laryngoscopy to assess vocal cord mobility. Findings such as true vocal cord paralysis or paresis, pooling of secretions, submucosal changes or distinct intramural invasion into the larynx, trachea or

Table 2 Clinical pointers for possible sites of involvement [12, 13]

Symptom	Possible site of involvement
Change in voice, voice weakness, hoarseness, vocal fatigue	Recurrent laryngeal nerve
Change in voice, dyspnoea, stridor, haemoptysis	Laryngeal framework
Dyspnoea, haemoptysis	Trachea
Odynophagia, dysphagia	Pharynx and oesophagus
Neck pain, neck stiffness	Prevertebral facia, neck muscles

hypopharynx must be duly investigated for locally advanced thyroid cancer (LATC).

In summary, on clinical examination, patients who should be investigated for LATC are:

1. Patient with thyroid cancer:
   (a) Extremes of age
   (b) Symptoms of change in voice, dysphagia, dyspnoea or haemoptysis
   (c) Fixed or restricted masses in the neck
   (d) Multiple nodal masses
   (e) Dilated neck and upper mediastinal veins
   (f) Restricted/fixed vocal cords
   (g) Identification of a subglottic or tracheal mass

2 Diagnostic Investigation

2.1 Routine Investigations for Evaluation of Thyroid Nodules

The preliminary evaluation of thyroid cancer is similar to evaluation of any thyroid nodule. Diagnostic modalities can be broadly categorized under two headings:

1. Routine investigations for evaluation of thyroid nodule
2. Investigations to assess the disease extent in a suspected case of thyroid cancer

Routine investigations for evaluation of thyroid nodule.

2.1.1 Ultrasonography

Ultrasonography (USG) is the most basic imaging modality and widely accepted as the first-choice imaging technique for detecting and diagnosing thyroid cancer. USG can also detect the presence of extrathyroidal extension (ETE) as an indicator of local invasion [17]. The sensitivity of ultrasonography for identifying ETE is variable, ranging from 42.9% to 91% [18–21]. USG cannot always adequately image deep anatomic structures and those acoustically shadowed by bone, cartilage or air.

Hence, when extrathyroidal extension of thyroid cancer is suspected based on the patient's history or physical examination, and on USG, a cross-sectional imaging should be performed.

2.1.2 Fine-Needle Aspiration Cytology (FNAC)

FNAC is a reliable diagnostic modality to differentiate a benign from a malignant nodule. The Bethesda System for Reporting Thyroid Cytopathology is given in Table 3. It however may not always be able to determine the poor histologic variants which may point towards patients having a LATC. Upfront diagnosis of poorly differentiated thyroid malignancy, Hurthle cell neoplasm and anaplastic carcinoma thyroid should raise concerns of advanced disease and warrant further investigations.

FNAC must be done under USG surveillance in cases with posteriorly located nodules and solid-cystic component in the nodule. USG-guided FNAC is known to reduce the sampling error [22, 23].

2.1.3 Core Needle Biopsy (CNB)

CNB is not the initial method of choice for all thyroid nodules. The tissue obtained by CNB provides more abundant material than FNA cytology, especially in cases with marked sclerosis and calcification [24, 25]. Certain studies have

Table 3 The Bethesda System for Reporting Thyroid Cytopathology

Bethesda grade	Diagnostic category	Estimated/ predicted risk of malignancy
I	Nondiagnostic or unsatisfactory	1–4%
II	Benign	0–3%
III	Atypia of undetermined significance or follicular lesion of undetermined significance	5–15%
IV	Follicular neoplasm or suspicious for a follicular neoplasm	15–30%
V	Suspicious for malignancy	60–75%
VI	Malignant	97–99%

demonstrated the advantages of histologic diagnoses made using CNB specimens over the cytological diagnosis using FNA for several specific diseases [26, 27]. Malignant lymphoma, medullary thyroid carcinoma, anaplastic thyroid carcinoma and parathyroid lesions can be confirmatively diagnosed with CNB based on the histologic morphology in conjunction with immunohistochemistry.

Investigations to assess the disease extent.

2.2 Investigations to Assess the Disease Extent

2.2.1 Contrast-Enhanced Computerized Tomography (CECT)

CECT is the most performed imaging in patients with suspected thyroid cancer. CT is usually performed to assess the presence of extrathyroidal invasion and nodal metastasis and is currently the primary imaging technique in the detection of extrathyroidal extension and cervical nodal metastasis from thyroid cancer [28]. Ishigaki and colleagues have demonstrated that CT is more accurate at detecting extrathyroidal extension when compared with ultrasonography [21].

The CT scan characteristics of structures involved by thyroid cancer are as follows:

Recurrent Laryngeal Nerve (RLN) Invasion

A normal RLN is posterior to the thyroid lobe, and in majority of its course, it usually lies in the fatty tissue in the tracheoesophageal groove. CT does not depict the fatty tissue in the tracheoesophageal groove well because of beam-hardening artefact, which is caused by the clavicle or by the presence of dense contrast material in the subclavian vein. There are no specific studies that have determined the efficacy of CT scan in determining the RLN involvement. In the study by Seo and colleagues [17], they determined the following criteria for RLN involvement: >25% tumour abutting posterior capsules of thyroid, finding of ipsilateral vocal cord palsy and loss of fatty tissue in the tracheoesophageal groove. When two out of three criteria were met, the sensitivity and specificity of predicting RLN involvement were found to be 78.2% and 89.8% respectively.

Tracheal Invasion

Invasion into the trachea can be determined by two criteria—the degree of encirclement [17] and depth of invasion [29]—deformity of the lumen, focal mucosal irregularity or thickening and intraluminal mass [30]. In the study by Seo and colleagues [17], if the tumour encircled >90° of tracheal circumference, then the CT scan is sensitive in 68.1% and specific in 76.6% for prediction of tracheal involvement. If the degree of encirclement was >180°, the sensitivity dropped to 55% and specificity to 67%.

A seminal paper by Shin and colleagues [29] gave an overview into the degree of tracheal invasion by locally advanced thyroid cancer. In his paper he described four pathological stages of tracheal invasion based on the resected specimens. Stage I disease invades through the capsule of the thyroid gland and abuts but does not invade the external perichondrium of the trachea. Stage II disease invades into the cartilage or causes cartilage destruction. Stage III disease extends into the lamina propria of the tracheal mucosa with no elevation or penetration of the mucosa. Stage IV disease is full-thickness invasion with expansion of the tracheal mucosa that is visible as a bulge or an ulcerated mass.

CECT validation of Shin's staging has not been performed, but radiologists should be encouraged to report tracheal involvement by thyroid cancers as per Shin's classification. This will help the surgeon plan the appropriate surgery preoperatively.

Esophageal Invasion

The oesophageal wall is more difficult to evaluate than the trachea because it is usually not distended with air. CT scan is unable to detect invasion into the layers of the oesophageal wall, especially the outer layer. The mean sensitivity, specificity and accuracy of CT for oesophageal invasion were found to be 78.6%, 96.2%, respec-

Table 4 Comparison between CT and MRI in determining the local invasion in LATC [17, 30–32]

	CT scan			MRI scan		
Structure involved	Sensitivity	Specificity	Accuracy	Sensitivity	Specificity	Accuracy
Trachea	59%	91%	83%	100%	84%	90%
RLN	29%	96%	91%	82%	94%	91%
Oesophagus	78%	90%	86%	94%	82%	88%

tively, when the angle of tumour contact was >180° with the oesophagus [17].

2.2.2 Magnetic Resonance Imaging (MRI)

MRI and CT have similar accuracy for predicting local invasion of the oesophagus, trachea and RLN (Table 4) [17, 30–32].

The presence or absence of tracheal invasion by LATC was diagnosed accurately (sensitivity of 100%, specificity of 84% and accuracy of 90%), but prediction with MR imaging of the precise depth of tumour invasion to the trachea was unreliable [30].

Takashima et al. reported 88% of diagnostic accuracy (94% sensitivity, 82% specificity) for the use of the MRI finding of effaced fatty tissue in the tracheoesophageal groove as the criterion for RLN invasion.

In a paper by Roychowdhury [33], the most suspicious finding for oesophageal invasion is a focal T2 signal in the outer layer of the oesophageal wall, and other signs include oesophageal wall thickening and effaced fat plane with the oesophagus.

2.2.3 Positron Emission Tomography (PET)

PET-CT typically has limited sensitivity for use in differentiated thyroid cancers. However it may have a role to play in assessing the extent of disease in dedifferentiated or poorly differentiated cancers [34]. The intensity of FDG uptake is correlated with progressive dedifferentiation and a more aggressive tumour [35]. Aside from regional metastases, PET-CT can also detect unrecognized distant metastases in the lungs and bones. It however is not the initial investigation of choice for patients with suspected LATC.

2.3 Endoscopic Studies

Additional investigations may be performed prior to surgery, including bronchoscopy/tracheoscopy and oesophagoscopy. These procedures are helpful in surgical planning and can be used in conjunction with preoperative CT information by allowing sensitive assessment for submucosal masses or bulges, increased mucosal vascularity or frank intraluminal invasion of the subglottis, trachea and oesophagus. Furthermore, endoscopic measurement of sites of intraluminal tracheal or oesophageal invasion obtained on initial endoscopy may facilitate the localization of surgical entry.

Endoscopic USG (EUS) is a procedure used in determining the size, depth of invasion and presence of paratracheal and paraoesophageal nodes in the oesophageal cancers [36]. EUS was reported to be more sensitive in detecting oesophageal invasion by thyroid cancer, especially in tumours involving lower and middle lobes, than oesophagography, oesophagoscopy and MRI [37, 38]. EUS examination of the thyroid gland has its limitations because it cannot visualize the upper lobes of the thyroid gland [38].

There are several case reports where endobronchial USG (EBUS) has been utilized to determine the extent of tracheobronchial involvement and obtain the tissue sample by transluminal needle aspiration [39, 40]. Although there are no studies which determine the use of EBUS in locally advanced thyroid cancers, it can have a potential use in determining the submucosal disease, extent of involvement, free tracheal rings and pretracheal and paratracheal nodes, and this potential benefit can be extrapolated by the use of EBUS in tracheobronchial invasion in oesophageal cancers [41].

2.4 Balloon Test Occlusion

Angiography with balloon occlusion studies may be required in patients with extensive extrathyroidal disease involving the great vessels in order to further clarify the surgical planning.

2.5 Serum Thyrotropin

A serum TSH level should be obtained if the nodule is >1 cm. If the serum TSH is subnormal, a radionuclide thyroid scan should be obtained to document whether the nodule is hyperfunctioning, isofunctioning or nonfunctioning [42]. Since hyperfunctioning nodules rarely harbour malignancy, if one is found that corresponds to the nodule in question, no cytologic evaluation is necessary. If overt or subclinical hyperthyroidism is present, additional evaluation is required. A higher serum TSH level, even within the upper part of the reference range, is associated with the increased risk of malignancy in a thyroid nodule, as well as more advanced stage thyroid cancer [43, 44].

3 General Principles of Thyroid Gland Surgery

- Successful thyroid surgery implies meticulous technical skill and attention to detail.
- As per widely accepted the Lahey's concept, routine visual identification of the RLN during thyroid surgery is now considered the gold standard for prevention of RLN injury.
- During positioning, 20° of reverse Trendelenburg position is maintained to avoid venous engorgement in the neck.
- There is little evidence that the timing of postoperative RAI is so crucial that iodine-containing preps cannot be used.
- Careful placement of the incision and protection of skin edges promote optimal cosmetic results.
- The division of the laryngeal head of the sternothyroid muscle is occasionally helpful to improve exposure of the superior pole of the thyroid gland. Dividing near the laryngeal attachment preserves its neurovascular supply.
- While dissecting below the isthmus, the surgeon must watch for the right and left inferior thyroid veins, which can blend in to form an inferior venous plexus, termed the plexus thyroideus impar, below the isthmus. The surgeon must also watch for a high-riding innominate artery or a thyroid ima artery. This artery arises as an unpaired inferior vessel from the innominate, carotid or aortic arch and occurs in 1.5–12% of cases.
- A constant midline tracheal reference point must be maintained as it helps as a landmark while dissection is continued and can be helpful if the neck base anatomy is distorted by malignant or benign goitrous.
- Identification and continued observation of the recurrent laryngeal nerve are the best ways to avoid injury to the nerve.
- Skeletonize the superior pedicle and ligate close to the thyroid gland to avoid injury to the superior laryngeal nerve and parathyroids.
- Identify and lateralize the parathyroid glands with their blood supply.
- Trachea may be deviated from it from its central position very often.
- Inferior thyroid artery should be ligated as proximal to the thyroid gland to preserve the blood supply to the parathyroid gland.
- In cases of large goiters, placing a nasogastric tube preoperatively can help in intraoperative localization of the oesophagus.

4 Hemithyroidectomy

4.1 Case

A 45-year-old female presented with a 6-month history of anterior neck swelling. Ultrasonography was suggestive of 4.8 cm hyperechoic nodule, solid nodule. FNAC was Bethesda II.

4.2 Operative Technique: Caveats

- Mark a 3–5 cm horizontal incision line between the cricoid cartilage and suprasternal notch (Fig. 1).
- Use an existing neck crease or line of relaxed skin tension.
- Mark the incision higher in younger patients, because the scar will migrate lower over time and a scar below the clavicle is often unsightly.
- Incise through the platysma to the subcutaneous tissue with electrocautery to the investing fascia. Platysma is deficient in the midline.
- Avoid injuring the communicating anterior jugular veins.
- Subplatysmal flaps are elevated with blunt dissection and electrocautery in the plane just above the investing fascia and anterior jugular veins up to the thyroid notch and down to the sternal notch (Fig. 2).
- The skin edges are protected with running, locking silk sutures after raising subplatysmal flaps.
- Identify the median raphe of the strap muscles.
- Divide the strap muscles in the midline up to the thyroid notch and down to the sternal notch with electrocautery.
- Elevate bluntly with a freer elevator, peanut sponge or electrocautery.
- The strap muscles should be sacrificed with a healthy margin if there is any tumour infiltration of the muscles.

Fig. 1 Horizontal collar incision is provided

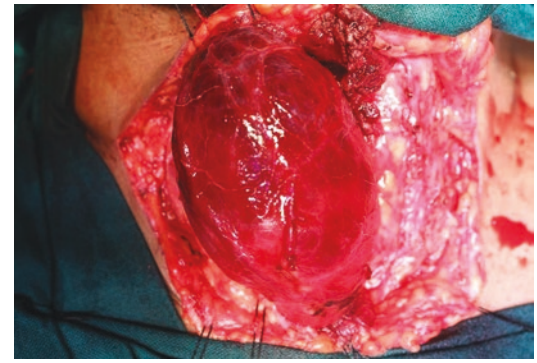

Fig. 2 Following skin flap elevation, the straps are cut and reflected. The enlarged thyroid lobe is delivered into the wound meticulously. Care must be taken not to injure any vessels

- Retract the strap muscles superolaterally to expose the superior pole.
- The sternothyroid and/or thyrohyoid muscles may have to be divided to provide better exposure of the superior pole in large goiters.
- Once the superior vascular bundle has been identified, skeletonize the vessels circumferentially using a right-angle clamp.
- Ligating the superior pole close to the thyroid helps decrease the risk of SLN injury.
- Dissect the thyroid carefully inferiorly and medially using a peanut sponge.
- The middle thyroid veins are encountered and ligated with the ultrasound scalpel/suture ligation.
- Once the RLN is identified, dissecting parallel and to the nerve sheath prevents damaging the nerve and its branches; very often we can encounter the branching of RLN at its point of entry to the larynx.
- Carefully dissect and lateralize the parathyroid glands with their blood supply when dissecting the thyroid capsule.
- The parathyroids usually receive their blood supply from the inferior thyroid arteries.
- Once the RLN and the inferior parathyroid gland have been safely identified, ligate the inferior vascular pedicle while the thyroid is mobilized medially.
- Make sure to identify early branching of the RLN near the inferior pedicle before ligating the pedicle (Fig. 3).

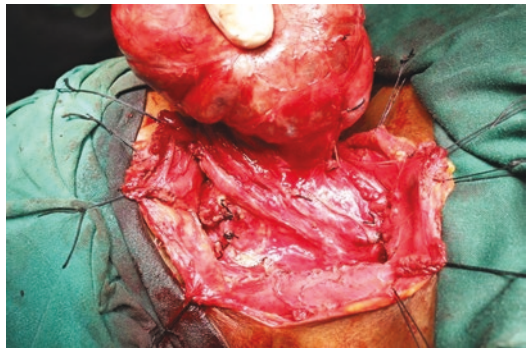

Fig. 3 Dissection is performed in the thyroid bed to identify and preserve the recurrent laryngeal nerve

Fig. 4 Intact specimen must be oriented prior to sending for histopathological examination

- Ligate the inferior pedicle close to the thyroid to avoid devascularizing the inferior parathyroid.
- Transect the thyroid at the junction of the isthmus and the contralateral lobe.
- Remove the pyramidal lobe if present, up to the thyroid notch.
- Irrigate the wound thoroughly, and evaluate bleeding during Valsalva by anaesthesia with bipolar cautery haemostasis.
- The strap muscles are approximated with absorbable sutures. Leave a space inferiorly to allow drainage of potential hematomas.
- Approximate the platysma layer carefully. Close the skin with absorbable subcuticular stitches (4-0 Monocryl).
- The specimen in toto is shown in Fig. 4.

5 Total Thyroidectomy for Thyroiditis

5.1 Case

A 39-year-old female presented with a 2-year history of anterior neck swelling. Diagnosed case of Hashimoto thyroiditis was not responding to medical therapy. Ultrasonography was suggestive of 2.5 cm hyperechoic nodule, solid nodule. FNAC was Bethesda II.

5.2 Operative Technique: Caveats

All operative steps are depicted in Figs. 5, 6, 7, 8, 9, 10, 11 and 12.

- At high risk for associated thyroid cancers [45].
- The parathyroid glands in patients with Hashimoto's thyroiditis are possibly more susceptible to injury, either due to the inflammation or due to the additional retraction required to mobilize the firmer than normal thyroid glands [42].
- The blood vessels are usually dilated and fragile, and they tend to rupture with aggressive manipulation. The bleeding may be difficult to control.

Fig. 5 A symmetrical cervical collar incision is marked. A taut suture thread can be used to mark the skin

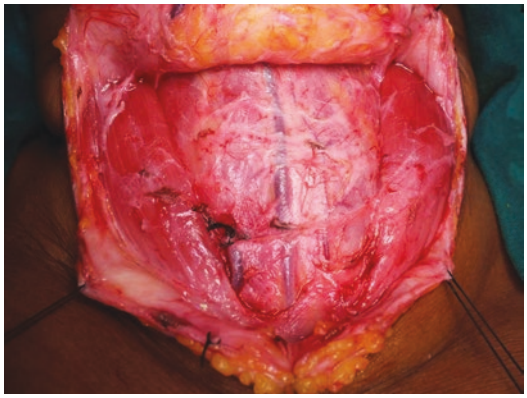

Fig. 6 Subplatysmal flaps are elevated, superiorly up to the hyoid and inferiorly up to the sternal notch. Care must be taken not to injure the anterior jugular vein in the midline and laterally the external jugular vein

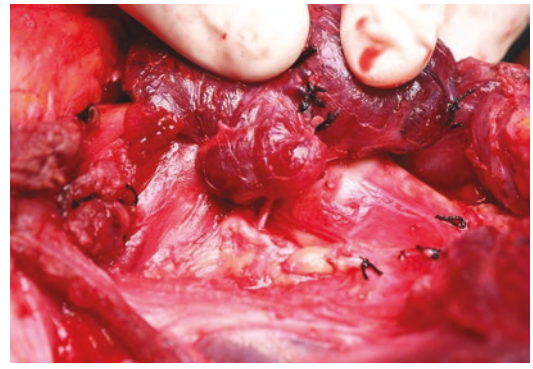

Fig. 9 Right RLN can be seen entering the larynx. We must also note the tubercle of Zuckerkandl just overlying the RLN

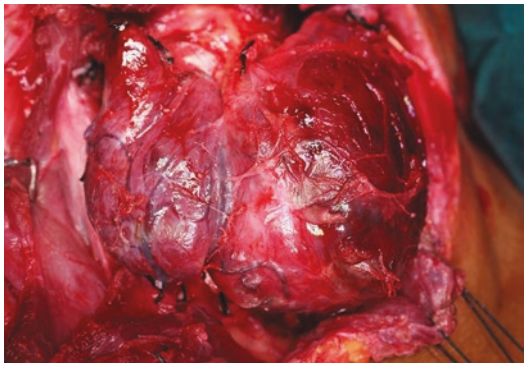

Fig. 7 Notice the dilated veins over the enlarged thyroid gland

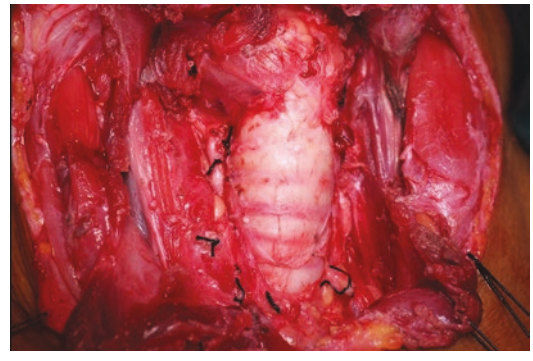

Fig. 10 Thyroid bed after completing the thyroidectomy

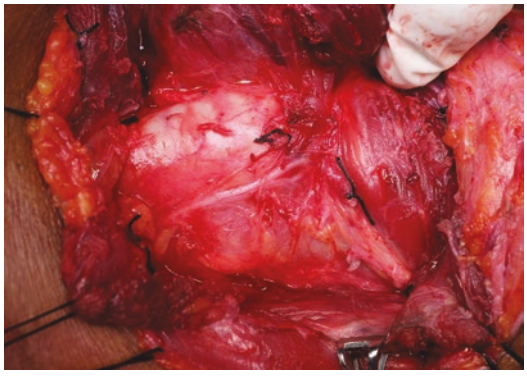

Fig. 8 The left recurrent laryngeal nerve is seen at the tracheoesophageal groove. One can appreciate the RLN being parallel to the trachea and entering the larynx at the cricothyroid joint

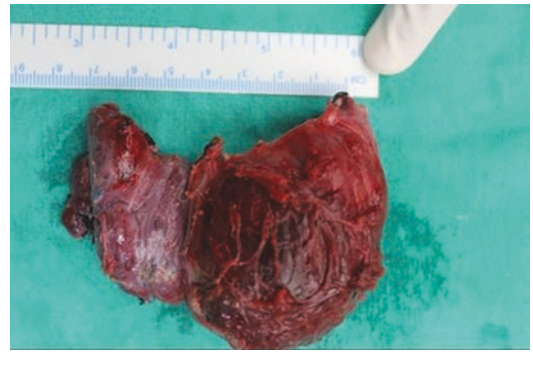

Fig. 11 Excised thyroidectomy specimen

Fig. 12 Postoperative follow-up after 6 months with almost no visible scar

- There may be a presence of multiple nodes in the paratracheal, prelaryngeal and pretracheal lymph nodes due to thyroiditis.
- The rate of temporary and permanent RLN palsy is described to be higher compared to thyroidectomy for benign disease [43].

6 Total Thyroidectomy for Large Multinodular Goiter

6.1 Case

A 56-year-old female presented with an 8-year history of anterior neck swelling. Ultrasonography was suggestive of multiple nodules with the largest nodule measuring 8 cm TIRADS 3 nodule. FNAC was Bethesda II.

6.2 Operative Technique: Caveats

All operative steps are depicted in Figs. 13, 14, 15, 16 and 17.

Fig. 13 Symmetrical Kocher's incision is marked

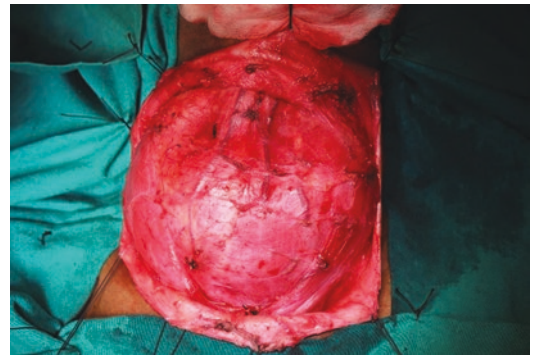

Fig. 14 Skin flaps are raised superiorly and inferiorly; one must note the engorged anterior jugular venous system

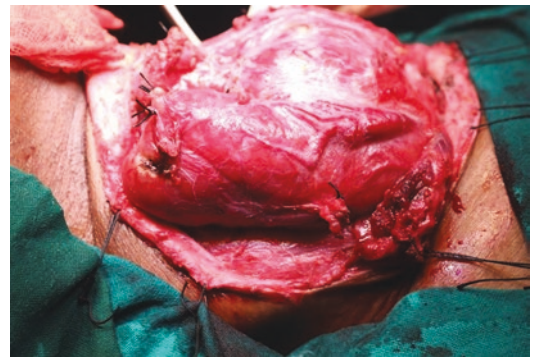

Fig. 15 A large thyroid gland's veins usually dilate. In cases with large thyroid glands, strap muscles can be divided to provide adequate exposure

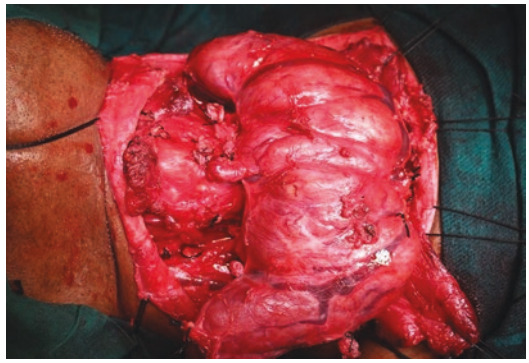

Fig. 16 Complete exposure of the thyroid gland

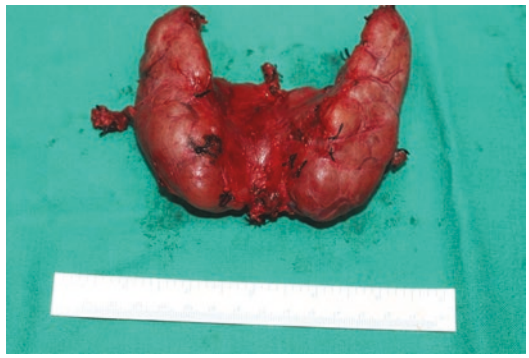

Fig. 17 Total thyroidectomy specimen

- Difficulty of intubation may be caused by an enlarged thyroid gland producing tracheal deviation or compression.
- The transient RLN palsy rates in total thyroidectomy have a higher rate than other operative methods for MNG. It is mainly due to traction on the RLN due to retraction and gland mobilization.
- The patients with MNG who had papillary carcinoma had an increased risk of multifocal cancers compared with the patients with solitary nodule who developed papillary carcinoma [44].
- Patients with long-standing goiter, even when benign, are more prone to develop tracheomalacia. A discussion with the anaesthetist is of utmost importance.
- Asymmetric nodules in the thyroid gland may displace the trachea to one side leading to gross tracheal deviation.
- Due to long-standing nature of the disease, there may be a considerable number of dilated vessels. Vessels must be carefully dissected and ligated when necessary.
- The lateral dissection must be done meticulously as there may be considerable shape distortion of the gland shape with nodules protruding out of the gland.
- Identification of parathyroid gland may be difficult due to enlarged thyroid gland.
- It is utmost important to ensure that all the nodules are dissected out, as any remaining nodules may lead to persistent or recurrent disease.

6.3 Case

A 48-year-old female presented with a 12-year history of anterior neck swelling. Ultrasonography was suggestive of multiple nodules with the largest nodule in the right lobe measuring 12 cm TIRADS 3 nodule. FNAC was Bethesda II.

All operative steps are depicted in Figs. 18, 19, 20 and 21.

Fig. 18 Skin incision and identifying the subplatysmal plane. The platysma is absent in the midline, and one must be careful during the flap elevation

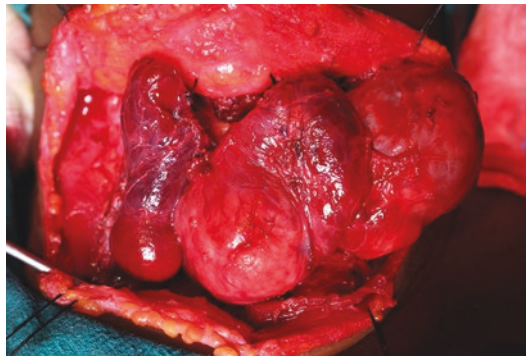

Fig. 19 In certain large multinodular goiter, the glands can insinuate under the sternocleidomastoid muscle. We must dissect along the plane and capsule of the thyroid gland to ensure the removal of entire thyroid gland

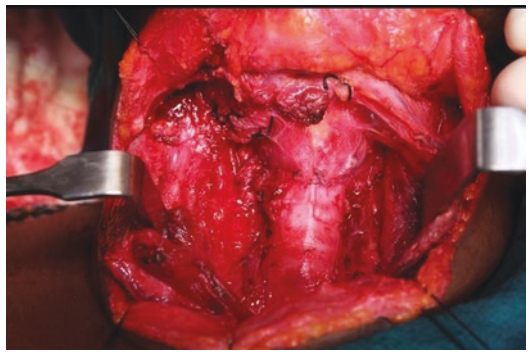

Fig. 20 Thyroid bed following total thyroidectomy and scabbard trachea appearance due to the compression of the trachea by the enlarged thyroid gland

Fig. 21 We can appreciate the multiple lobulations of the thyroid gland

7 Total Thyroidectomy with Sternotomy for Large Multinodular Goiter

7.1 Case

A 48-year-old female presented with a 7-year history of anterior neck swelling, and the lower pole of the swelling was not palpable despite adequate neck extension. Ultrasonography was suggestive of multiple nodules with the largest nodule measuring 6 cm TIRADS 3 nodule. FNAC was Bethesda II. CECT was suggestive of mediastinal extension up to the subclavian artery.

7.2 Operative Technique: Caveats

All operative steps are depicted in Figs. 22, 23, 24, 25, 26, 27, 28, 29 and 30.

- Crile initially defined mediastinal goiters as those that extend to or are inferior to the aortic arch [46].
- The need for sternotomy must be discussed with the patient and relatives preoperatively. A cardiothoracic team must be on standby for all the substernal goiter cases.

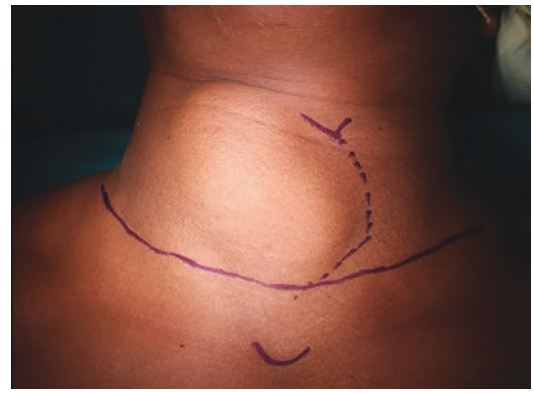

Fig. 22 In cases of large thyroid glands, the trachea may be grossly deviated

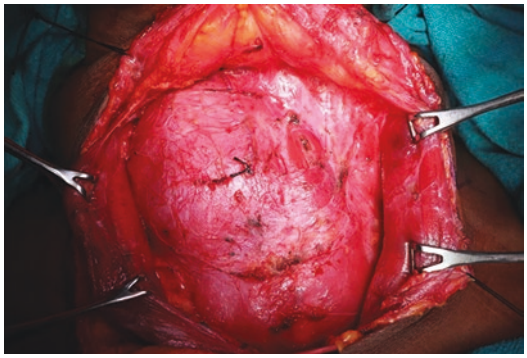

Fig. 23 Subplatysmal flaps are raised, and sternocleidomastoid muscles are retracted laterally to expose the thyroid gland completely

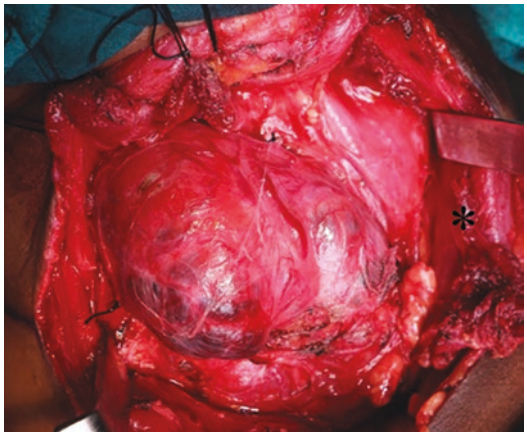

Fig. 24 Lower pole of the thyroid gland is seen passing below the sternum. * shows the pericardia

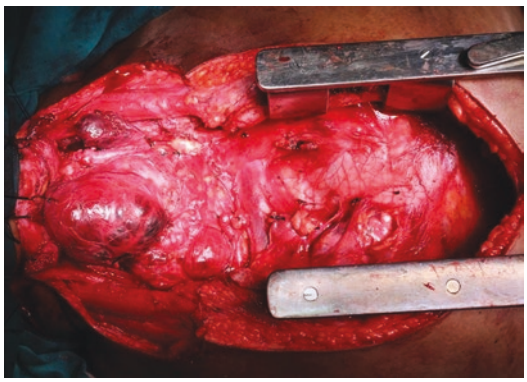

Fig. 25 A complete sternotomy is done

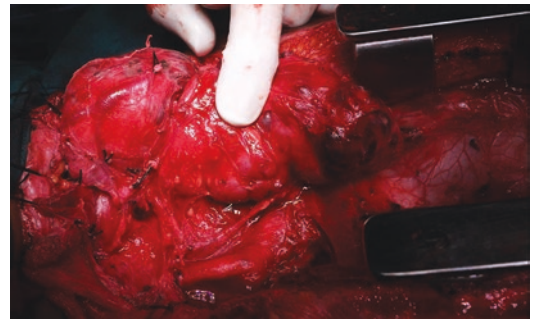

Fig. 26 The thyroid gland is freed from all sides, and the dissection is continued laterally to identify and preserve the RLN

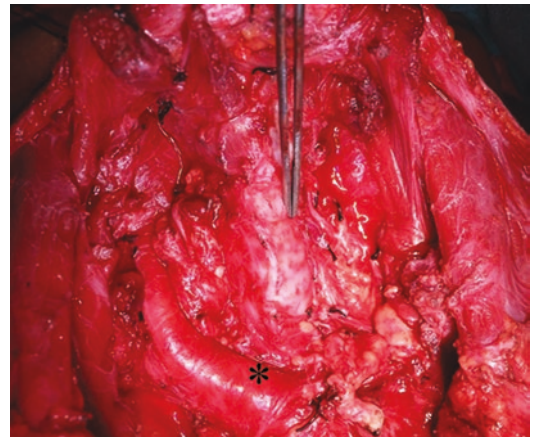

Fig. 27 The lower pole of the gland was reaching up to the right innominate artery. * shows the innominate artery

Fig. 28 Total thyroidectomy specimen with cervical and mediastinal parts. * shows the cervical part and ** shows the mediastinal part

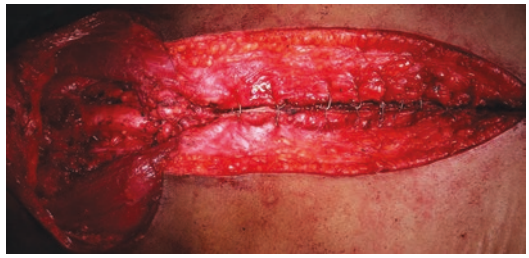

Fig. 29 The sternotomy wound is closed with steel wires to provide adequate strength

Fig. 30 Following layered skin closure

- The sternotomy is required in 5–29% of cases of substernal goiters based on the pooled evidence [46].
- A history of goiter with retrosternal extension beyond 160 months is a risk factor for sternotomy. Thyroid tissue density, posterior mediastinal location and subcarinal extension, as measured using CT imaging, are independent preoperatively obtained risk factors for sternotomy [47].
- An initial transcervical collar incision is taken to dissect the thyroid by the standard approach.
- The lower pole dissection is done cautiously as long-standing goiters and some ectopic nodules can derive additional blood supply from the mediastinal vessels.
- Mercante in 2011 [48] proposed a classification system for the substernal goiters based on the CECT imaging:
  - Grade I—the lower border of the thyroid is above the aortic arch.
  - Grade II—the lower border of the thyroid is between the convex and concave parts of the aortic arch.
  - Grade III—the lower border of the thyroid is below the concave part of the aortic arch.
- Early retrosternal extension cases may be completely approached by transcervical pull-up technique.
- The evidence of an ectopic nodule, a dumbbell-shaped goiter, a conical-shaped goiter constricted by an isthmic thoracic inlet or a thoracic goiter component wider than the thoracic inlet, can also predict the need to undergo sternotomy [47].
- Mediastinal goiters can remain asymptomatic until compression of the structures located in the thoracic inlet occurs. Life-threatening mechanical compression can occur because of the limited space below the thoracic inlet.
- The pooled incidence of malignancy in retrosternal goiters has been reported between 3 and 21% [49, 50]. Rugiu and colleagues consider malignancy as a high risk of sternotomy procedure due to the higher chances of extrathyroidal extension and/or the need to perform level VII lymph node clearance [51].
- In a recent Italian case series of sternotomy for thyroidectomy showed that postoperative bleeding (0.5%), permanent unilateral recurrent laryngeal nerve palsy (1.3%), bilateral recurrent laryngeal nerve palsy (0.6%), transient hypoparathyroidism (14%) and permanent hypoparathyroidism (4.1%) [52]. The mortality rate of substernal goiter surgery has been reported as high as 2.3%, the surgery for retrosternal goiters involves a higher risk for complications than cervical goiters do and the risk does not differ between patients with and without symptoms [53].

8 Total Thyroidectomy with Ministernotomy and Neck Dissection

8.1 Case

A 63-year-old female presented with a 7-year history of anterior neck swelling. Ultrasonography was suggestive of multiple nodules with the largest nodule measuring 4 cm TIRADS 5 nodule.

FNAC was Bethesda VI—papillary thyroid cancer. CECT was suggestive of retrosternal extension and a level VII lymph node.

8.2 Operative Technique: Caveats

- Ministernotomy involves a midline osteotomy from the sternal notch down the manubrium to the second intercostal space, where a second horizontal osteotomy is performed (Fig. 31).
- A thyroidectomy is attempted first through a cervical approach, before a powered saw is used to make the osteotomies and a sternal retractor used to access the mediastinum, and a total thyroidectomy is then completed.
- Anterior mediastinal nodal clearance can be performed after opening the sternum (Fig. 32).
- The sternum is closed with wires (Fig. 33) as a drain is placed and the wound is closed in layers. The advantages, compared with a conventional sternotomy that continues from sternal notch to xiphoid process, include a smaller incision, decreased pain, faster recovery, shorter hospital stay and fewer complications.

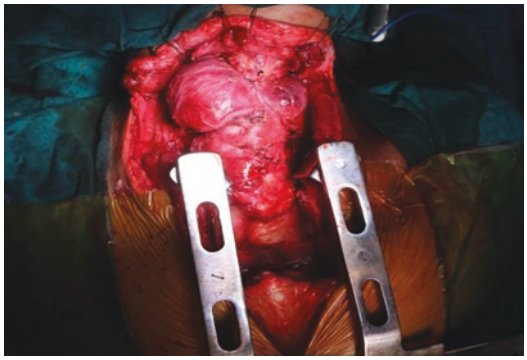

Fig. 31 Ministernotomy can be used as an alternative to complete sternotomy, where osteotomy is done only for the manubrium sterni

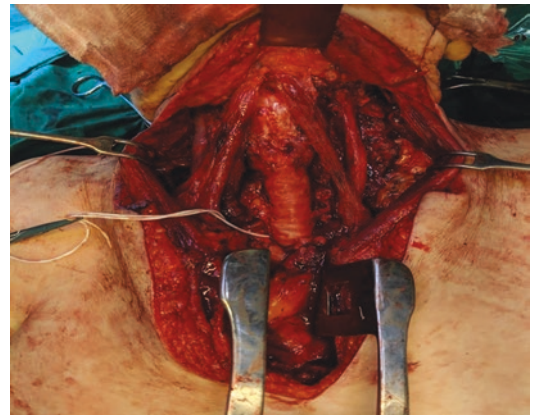

Fig. 32 The thyroid bed following thyroidectomy with bilateral central compartment and bilateral neck dissection. We can appreciate the yellow-amber-coloured thymic tissue in the mediastinum

Fig. 33 Sternotomy wound closed with steel wires

9 Thyroidectomy for Carcinoma Thyroid

9.1 General Range of Surgeries for Carcinoma of Thyroid and Their Indications

1. Hemithyroidectomy:
   (a) If the nodule size is <4 cm, unifocal.
   (b) There are no cervical lymph nodes.
   (c) No distant metastases.
   (d) No extrathyroidal extension.
   (e) No prior radiation exposure.
   (f) No high-risk mutation on genetic testing.
2. Completion thyroidectomy:
   (a) Adverse histology of the primary tumour and initial disease features (tall cell variant, insular, poorly differentiated).
   (b) The rapid Tg-level doubling time (less than 1 year or 3 years). This is a dynamic representation of an aggressive tumour and its growth.
   (c) Non-iodine avidity or inability to secrete thyroglobulin.
   (d) The presence of significant uptake on FDG PET.
   (e) Evidence of invasion of vital structures.
   (f) Response to initial treatment.
   (g) Presence of BRAF, TERT or PAX8/PPRG mutations.
   (h) Rate of growth of the lymph nodes.
   (i) Presence of extranodal extension to trachea, oesophagus or carotid vessel.
3. Total thyroidectomy:
   (a) If the nodule size is >4 cm, multifocal disease.
   (b) Presence of cervical lymph nodes.
   (c) Presence of metastases.
   (d) Extrathyroidal extension.
   (e) Prior radiation exposure.
   (f) High-risk mutation on genetic testing.
4. Total thyroidectomy with central compartment clearance:
   (a) T3/T4 disease.
   (b) Presence of extrathyroidal extension.
   (c) Central compartment lymph nodes.
   (d) Metastatic lateral compartment lymph nodes.
5. Total thyroidectomy with central compartment clearance and bilateral neck dissection:
   (a) Presence or lateral compartment lymph nodes.
   (b) Grossly raised calcitonin in medullary thyroid cancer.
6. Total thyroidectomy with RLN sacrifice:
   (a) Aggressive histology and genetic variants
   (b) Iodine refractory, previous EBRT and recurrence following adjuvant therapy
   (c) Nerve invasion at point of entry to the larynx
   (d) Normal contralateral vocal cord function
   (e) No distant metastasis
7. Total thyroidectomy with laryngotracheal shave-off procedure:
   (a) Locally advanced thyroid cancer with SHIN I–II laryngotracheal invasion
8. Total thyroidectomy with total laryngectomy:
   (a) Locally advanced thyroid cancer with gross laryngotracheal invasion SHIN IV
9. Emergency airway management for huge aggressive thyroid malignancies:
   (a) As a part of palliative care in advanced anaplastic thyroid cancer

10 Hemithyroidectomy for Thyroid Cancer

- Whether hemithyroidectomy or total thyroidectomy is elected as the index operation, meticulous dissection to preserve the parathyroid glands and protect the recurrent laryngeal nerve is essential.
- The American Thyroid Association (ATA) and National Comprehensive Cancer Network (NCCN) thyroid cancer guidelines recommend consideration of hemithyroidectomy for nodules smaller than 4 cm without other worrisome features.
- Nodules that appear to have radiologic evidence of extrathyroidal extension (ETE) or

invasion into adjacent structures suggest a higher risk of aggressive disease and local recurrence and should be planned for total thyroidectomy.

- If the contralateral lobe is normal, then initial hemithyroidectomy is appropriate, provided that the lobe with the malignant nodule does not show the features of ETE.
- A hemithyroidectomy for solitary thyroid nodules with Bethesda III or IV indeterminate cytology can be considered as per the ATA guidelines [2].
- TERT promoter mutations, alone or in combination with BRAFV600E mutations, predict a high risk of recurrence or more aggressive disease course; thus, total thyroidectomy is recommended if these mutations are present [54].
- When considering the need for postoperative thyroid hormone, it is important to look at higher predictors of needing postoperative thyroid hormone, such as preoperative TSH, echogenicity of the thyroid evaluating for heterogeneity and presence of thyroid antibodies. If someone is already on thyroid hormone prior to surgery, they may be more inclined to have a total thyroidectomy. Additionally, the ideal TSH postoperatively will depend on the final pathology, as patients with malignancy will have a lower TSH goal than those with benign disease. The patient's preoperative TSH level should be considered in concert with the likelihood of malignancy when counselling on the possibility of requiring thyroid hormone postoperatively.
- One must also understand that operative outcomes for patients undergoing thyroid surgeries—hemithyroidectomy, total thyroidectomy and completion thyroidectomy—were comparable including hematoma requiring reoperation, seroma formation, temporary vocal cord paralysis and temporary hypoparathyroidism [55].
- Exploring patients' preferences preoperatively and assessing their understanding of what each option entails in terms of clinical, biochemical and radiological follow-up and the possible need for further surgical intervention are vital.

11 Completion Thyroidectomy for Thyroid Cancer

The follow-up of the DTC patients was RAI based until recently where the follow-up has now shifted to Tg based. So the need for completion thyroidectomy is declining as in properly selected low- to intermediate-risk patients (patients with unifocal tumours of <4 cm, and no evidence of extrathyroidal extension or lymph node metastases by examination or imaging), and the extent of initial thyroid surgery probably has little impact on disease-specific survival [56]. Recurrence rates are quite low in these patients. In properly selected low-risk patients, lobectomy alone is sufficient. Completion thyroidectomy should be offered to patients for whom a bilateral thyroidectomy would have been recommended had the diagnosis been available before the initial surgery or in cases where the primary histology after lobectomy has adverse histology. The anatomical changes in revision surgery are given in Table 5.

There are certain factors which will guide the treating physician in making decision regarding the revision thyroid surgery in a recurrent setting [57]. These factors include:

1. Primary tumour factors:
   (a) Adverse histology of the primary tumour and initial disease features (tall cell variant, insular, poorly differentiated).
   (b) The rapid Tg-level doubling time (less than 1 year or 3 years). This is a dynamic representation of an aggressive tumour and its growth.
   (c) Non-iodine avidity or inability to secrete thyroglobulin.
   (d) The presence of marked uptake on FDG PET.
   (e) Evidence of invasion of vital structures.
   (f) Response to initial treatment.
   (g) Presence of BRAF, TERT or PAX8/PPRG mutations.
2. Lymph node factors:
   (a) Rate of growth of the lymph nodes
   (b) Presence of extranodal extension to the trachea, oesophagus or carotid vessel

Table 5 Anatomical changes in revision surgery

Anatomical changes in revision surgery	
Structure	Importance in revision surgery
Carotid sheath	• Constant lateral boundary of dissection • Initially identifying the RLN
Strap muscles	If excised in previous surgery: • Great vessels may be superficial and medial • Scarring in the central neck
Internal jugular vein	Medialized and adherent to undersurface of the sternomastoid muscle
Brachiocephalic artery	Drawn up in the neck due to contracture of central compartment
Ipsilateral RLN	• Superficial and buried under scar tissue • May be adherent to strap muscles • Generally, it is scarred immediately to the upper cervical trachea over the first few tracheal rings • Lateral cricoid and is most injured in this segment
Parathyroid glands	• Along with RLN • Requires high index of vigilance

The decision regarding intervention versus active surveillance after detection of recurrent or persistent tumour is controversial and complex. After discussion with multidisciplinary team about the disadvantages and advantages of all therapeutic interventions, effective therapy must be customised to the individual. Factors to be considered will include whether the primary tumour had an adverse histology, the rate of change of Tg levels, the rate of growth of imaged lymph nodes and the presence of extranodal extension to the central aerodigestive tract or carotid system. Additional factors to be considered include age, inability of non-iodine avidity, no increase in Tg levels, presence of molecular pointers for aggressive behaviour, distant metastases and comorbidities. A surgical exploration should under no circumstances be performed, if a lesion cannot be located on sufficient anatomical imaging. One should not shy away from referring a patient to a more experienced surgeon, if his or her complication risks are higher or if the volume of revision surgeries performed are lower.

Optimal timing of completion thyroidectomy is primarily determined by the timeframe probability within which laryngeal recovery is expected, along with oncologic treatment timeframe issues in the setting of malignancy.

Several studies put forth a range of timing that are considered safe for completion surgery, with some studies showing an increased risk of complications when completion surgery is performed in an early timeframe. Based on these evidences, the optimal timing of completion surgery can be narrowed to less than 3 days or greater than 3 months [58–62]. It is postulated that this timeframe will help in minimizing complications, as surgery is either performed before the fibrosis starts or can be done after fibrosis is completed. It should be kept in mind that laryngeal recovery is a predominant and overwhelming consideration for safe timing of completion surgery.

When completion thyroidectomy is performed within 6 months of the first surgery, there is generally no alteration of oncologic risk for most thyroid cancers in patients without evidence of residual tumour or distant metastasis [58, 62, 63].

When the surgery is planned on the only function RLN side or at the site of previously handled nerve. Several studies show that most neuropraxic RLN injuries generally recover within 2–6 months [64–66].

12 Total Thyroidectomy for Thyroid Cancer

An attempt to perform a complete resection (R0) is vital and offers the patient the best possible chance of cure [67]. As per the Memorial Sloan Kettering LATC case series, elderly patients, tumours more than 4 cm in size, gross residual disease and distant metastases were predictors of poor survival [67]. The spectrum of surgeries performed in patients with LATC range from a total thyroidectomy with bilateral central and lateral neck dissections to multiorgan resections, sometimes requiring sternoto-

mies and vascular resections. The aim of performing such extensive surgeries is to achieve R0/R1 resections, thus optimizing outcomes. A balance between the morbidity of such surgeries, the poor prognostic factors and the possible outcomes should be maintained prior to decision-making. Radical surgeries must not be performed in poor surgical candidates and in patients with poor prognosis.

12.1 Recurrent Laryngeal Nerve

Management of recurrent laryngeal nerve during surgery for LATCs is critical. Falk et al. and Nishida et al. studied the effect of RLN preservation versus resection in functionally intact nerves in LATC cases (Table 6). They found that there was no difference in survival when the RLN was preserved, if patients received adjuvant RAI therapy with TSH suppression and there was no gross residual following resection [11, 68]. The basic principle of management of the RLN during surgery for LATCs is to make all attempts to dissect the nerve out of the tumour/nodal mass and preserve the continuity, without leaving back gross residual disease (Figs. 40, 41 and 42).

A nonfunctioning RLN involved by tumour may be resected en bloc with the thyroid resection. However, if there is no evidence of tumour infiltrating the RLN, then even in a nonfunctional nerve, it must be meticulously dissected and preserved to allow for recovery [11, 66]. The use of IONM here may help in determining the amplitude of EMG, suggesting the possible recovery pattern in the postoperative setting.

If the RLN is resected for oncological purposes, every attempt should be made to reconstruct the nerve using the ansa cervicalis or other nerve grafts (Fig. 43). This would only be possible if the proximal and distal nerve ends are available for anastomosis. Typically, this is not feasible when the disease is infiltrating the nerve at the entry point, near the Berry's ligament. This kind of resection and anastomosis is generally possible when a nodal mass, rather than a primary thyroid tumour, involves the nerve.

Table 6 Summary of the European thyroid association consensus statement on RAI Therapy

Summary of the European thyroid association consensus statement on RAI therapy	
Recommendations	Factors to be considered
RAI therapy should be based on initial prognostic indicators for thyroid cancer-related death and recurrence	• ATA risk groups: (1) low, (2) intermediate and (3) high; post-surgical evaluation: (1) neck ultrasound and (2) thyroglobulin
The use of I-131 therapy as adjuvant treatment or treatment of known disease is indicated in the high-risk group	• Overall survival and disease-free survival are improved with RAI; activities >100 mCi should be considered
In the intermediate-risk category, RAI therapy may be indicated according to individual risk factors	• The greatest benefit in patients with the following: (1) advanced age, (2) aggressive histologies, (3) increasing volume of nodal disease, (4) extranodal extension of the tumour, (5) multiple N1 and/or (6) lymph node metastases outside the central neck; the final results of prospective trials are expected
In low-risk patients, RAI therapy should be based on individual risk modifiers	• RAI treatment not indicated in PTC <1 cm (uni- or multifocal); abnormal neck ultrasound or high Tg may indicate need for RAI therapy
Recombinant human TSH is preferred for TSH stimulation	• Indicated for all RAI activities; approved in all risk groups but metastatic disease
Activities of 30 mCi are equally effective as higher activities for remnant ablation	• If low-risk patients are referred for thyroid remnant ablation, activity of 30 mCi should be considered as effective and safer than higher activities
Before RAI therapy diagnostic scan is not routinely required	• RAI low activities before RAI treatment can induce stunning and reduce treatment effectiveness
Before RAI therapy any iodine-containing drug should be avoided	• Low-iodine diet may be advised

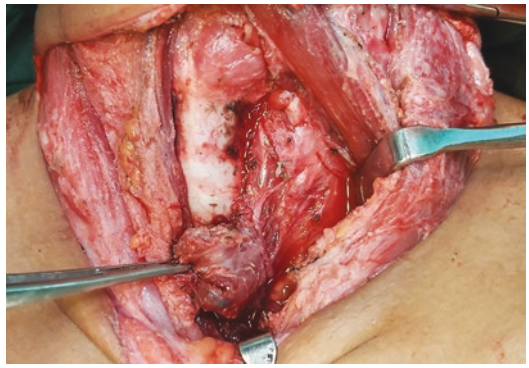

Fig. 34 The thyroid gland is adherent to the trachea and the RLN on the left side

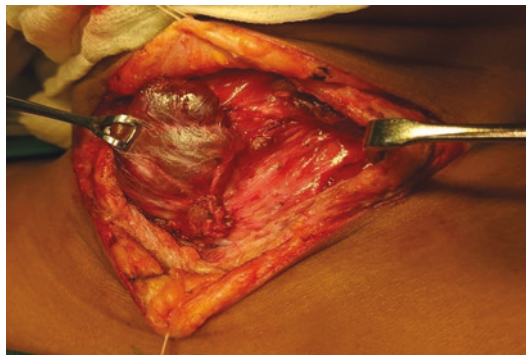

Fig. 35 The dissection in the thyroid bed shows an enlarged central compartment lymph node

12.2 Parathyroids

Concomitant performance of central compartment clearance along with thyroid surgery increases the rates of the more frequent complications of surgery such as RLN injury and hypoparathyroidism. The incidence of inadvertent parathyroidectomy during central compartment surgery has been reported to be between 6.4% and 21.6% [69–73].

The status of the functioning parathyroid in revision surgery can be assessed preoperatively by the corrected serum calcium levels and serum parathormone levels. It is exceedingly difficult to identify and differentiate parathyroid tissue from recurrent disease and central compartment nodes by any preoperative imaging.

The parathyroid glands may be found among resected paratracheal lymph nodes because their identification and distinction from the lymph nodes may be difficult. There may be higher possibility of glands being devascularized if they are inadvertently handled. A small frozen section biopsy, if available, may serve the dual purpose of confirming the histology and possibly even vascularity of the glandular tissue in question [74]. It should be noted that a reimplanted parathyroid tissue has a higher chance of surviving and functioning than a devascularized parathyroid tissue [75].

Factors that determine the sacrifice and safety of the recurrent laryngeal nerve (RLN) can be categorized into those favouring nerve sacrifice and those favouring nerve sparing. Factors favouring nerve sacrifice include aggressive histology and genetic variants, cases that are iodine refractory or have a history of previous external beam radiation therapy (EBRT) or recurrence following adjuvant therapy, nerve invasion at the point of entry to the larynx, normal contralateral vocal cord function and the absence of distant metastasis. On the other hand, factors favouring nerve sparing include young patients, particularly those with radioiodine avid papillary thyroid carcinoma, good efficacy to radioactive iodine (RAI) and EBRT, elderly patients with an increased risk of aspiration, reduced pulmonary capacity, individuals who rely on their voice professionally, contralateral RLN paralysis and the presence of distant metastasis [76].

12.3 Laryngotracheal and Oesophageal Invasion

Laryngeal invasion: The various images depicting gross extrathyroidal extension are depicted in Figs. 36, 37, 38 and 39. Less than 12% of patients are known to have laryngeal infiltration. Laryngeal involvement is generally restricted to involvement of the outer framework of the larynx in most cases, without mucosal involvement. Every attempt should be made to preserve the larynx by performing conservative procedures rather than a total laryngectomy. Total laryngectomy may benefit patients with a varying degree of airway obstruction, intraluminal bleed, intraluminal invasion or loss of laryngeal function and in patients who are unsuitable for

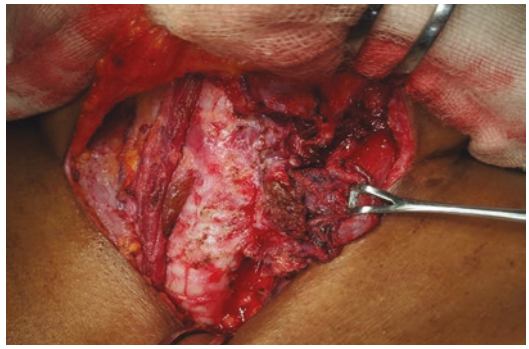

Fig. 36 Disease infiltrating the laryngotracheal complex: a shave-off procedure was done

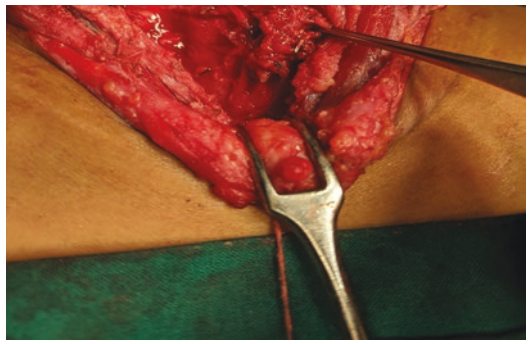

Fig. 37 A densely adherent disease in the tracheoesophageal groove: the RLN was sacrificed on the same side

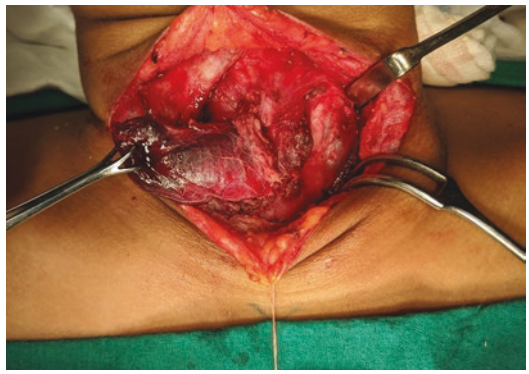

Fig. 38 * shows an extrathyroidal extension into the strap muscles

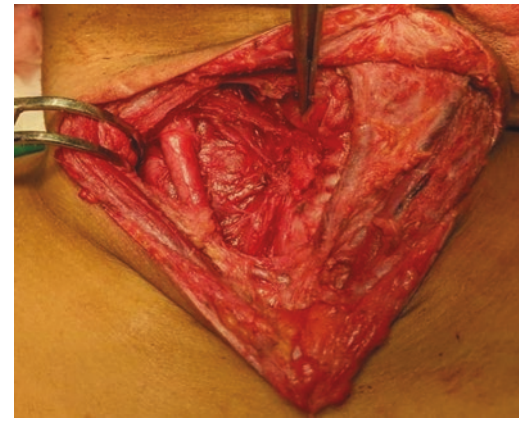

Fig. 39 Disease infiltrating into the oesophagus and completely encapsulating the RLN. * shows the common carotid artery

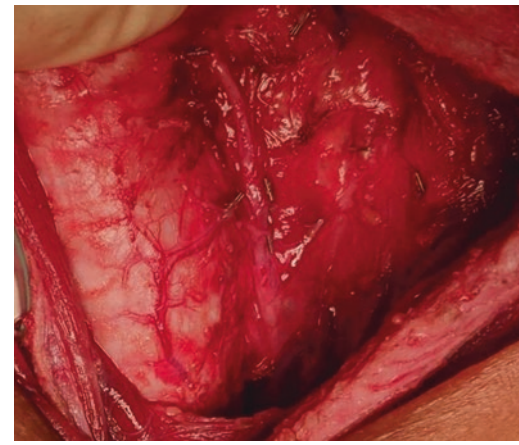

Fig. 40 The thyroid bed following a meticulous central compartment dissection. RLN can be seen in the left tracheoesophageal groove

conservative laryngeal surgery. The commonly performed operative procedures, in patients with laryngeal invasion, are peeling or shaving the tumour off the laryngeal framework, window resections of the thyroid cartilage, partial laryngectomy and total laryngectomy. A simple shave excision can be considered if the tumour is just abutting the outer perichondrium without gross involvement. Mucosal laryngeal invasion may be amenable to vertical laryngectomy if the disease is lateralized and the patient had a functioning larynx.

Tracheal invasion: Conflicting data on management of tracheal invasion is due to non-standardized definitions of invasion and performing complete versus conservative procedures. Tracheal invasion is most often classified by the Shin's staging:

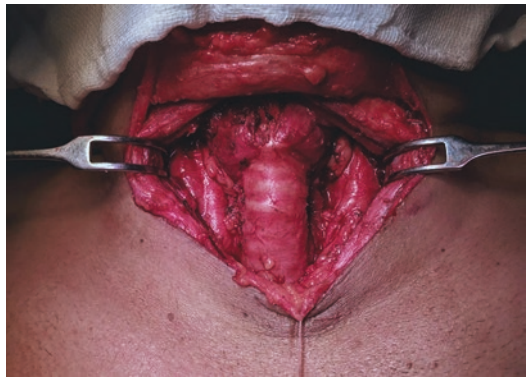

Fig. 41 The thyroid bed following total thyroidectomy with bilateral central compartment clearance with bilateral neck dissection

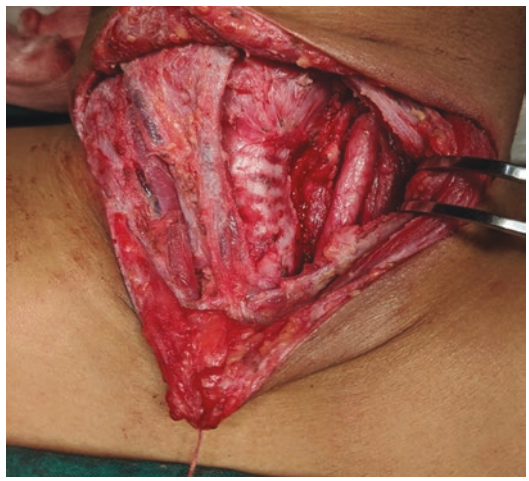

Fig. 42 Following a shave-off procedure with left RLN sacrifice

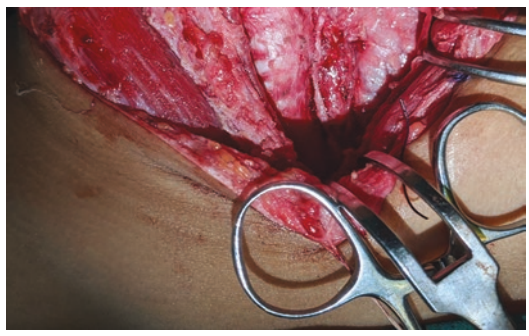

Fig. 43 Ipsilateral cable nerve grafting with the sural nerve

- Stage I: Invasion of the capsule of the thyroid with the disease abutting the outer perichondrium of the trachea without eroding the cartilage.
- Stage II: Outer tracheal cartilage invasion, either in between the tracheal rings or destroying the cartilage.
- Stage III: Tracheal cartilage invasion or extension between the rings into the underlying tracheal lamina propria without mucosal invasion.
- Stage IV: A complete tracheal cartilage invasion with intraluminal disease, seen as an ulceration [29].

Intraoperatively, tracheal shaving of the tumour can be done for Shin I tumours, and a window/sleeve resection can be advocated in Shin II tumours, with lateral or anterior invasion of the trachea. Circumferential resection is generally performed in patients with Shin III and IV tumours [16]. All attempts should be made to avoid leaving back gross residual disease on the trachea as this is associated with poorer outcomes [15, 77, 78].

Oesophageal involvement: This invasion typically occurs along with tracheal invasion but can also occur when paratracheal or paraoesophageal lymph nodes have extranodal extension. It is usually considered a poor prognostic factor and correlates with significant reduction in overall survival [11, 16]. All attempts should be made to perform a simple cuff excision of the muscularis layer of the oesophagus, and in most cases, this is adequate. Segmental resection should be performed if the tumour is extensively involving all three layers. Appropriate reconstruction will be required if full-thickness excisions are needed and a preoperative suspicion of oesophageal involvement will help plan this better.

12.4 Intraoperative Neuromonitoring

Intraoperative neuromonitoring (IONM) can help the surgeon identify the RLN in a distorted anat-

omy, it can estimate the residual nerve function following surgery, it can provide pointers to decision-making when both the RLNs are at risk due to bilateral disease and the postoperative residual nerve potential might provide clues to possible postoperative complications. The exact role of IONM has not been clearly defined and whether IONM can decrease the incidence of temporary or permanent RLN palsy, especially in these cancers, is a matter of contention.

IONM may not be mandatory for every thyroidectomy, but surgeons should use IONM, if they feel more comfortable using the device during thyroidectomy, especially for LATCs where there is a chance of distorted anatomy. In LATC, where bilateral RLNs are engulfed and/or involved, IONM may guide the surgeon to assess the functional integrity of the RLNs following a complete dissection, in addition to its anatomical integrity. Vagal stimulation before and after dissection helps confirm the critical point of injury and may guide us in identifying the correct segment in case of a segmental injury. The advances in continuous IONM (C-IONM) technology may minimize the limitations of intermittent IONM monitoring [79]. However, IONM should not replace anatomical identification of the nerve and following of correct techniques of surgery.

Skin closure and follow-up photos are depicted in Figs. 44 and 45.

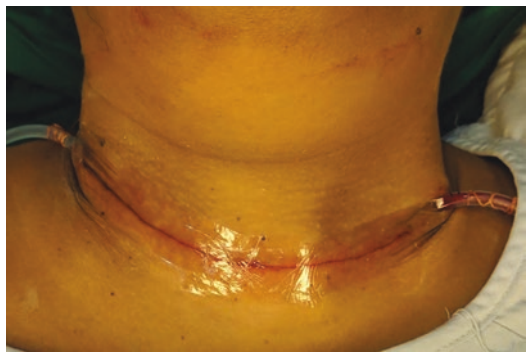

Fig. 44 The skin is closed in a subcuticular manner with a clear film bandage to see the wound through it. Bilateral neck drains needed in cases where the neck is addressed on both sides. An optional compression dressing may also be provided

Fig. 45 One-year follow-up scar photograph: the scar is well hidden in the lower neck crease

13 Radioactive Iodine therapy

The non-descript colloquial use of the word ‘ablation’ has thus far frustrated a constructive scientific dialogue. A precise nomenclature to describe these three important goals has not been widely accepted. A nomenclature that uses ‘RAI therapy’ as the broad term that encompasses the three primary goals associated with an administered activity of RAI: (a) remnant ablation, (b) adjuvant treatment or (c) treatment of known disease.

In this context, remnant ablation refers to the use of RAI to destroy postoperatively remaining, presumably benign residual thyroid tissue to facilitate follow-up studies (such as serum thyroglobulin and RAI imaging).

Within the context of thyroid cancer care, adjuvant therapies can be defined as I-131 therapy after surgical resection of all known primary tumour tissue and metastatic foci in an effort to destroy subclinical microscopic tumour deposits that may or may not be present. The goals of adjuvant therapy are to improve disease-specific survival and disease-free survival.

Treatment of known disease refers to the goal of destroying persistent or recurrent DTC foci with RAI in order to improve progression-free, disease-specific and overall survival. It can be given either with curative or palliative intent.

After total thyroidectomy, the size of normal, presumably benign remnants is usually small,

resulting in very low or frequently even undetectable serum thyroglobulin (Tg) levels on levothyroxine (LT4) treatment. In these cases, particularly in low-risk patients, the goal of remnant ablation is already achieved following the surgical procedure alone just by the surgical procedure, and thus, there is no rationale to perform RAI ablation. Currently, even without postoperative RAI therapy, most patients can be followed up with serum Tg on l-T4 treatment: an undetectable level is reassuring as well as a low but detectable level. In the latter case, the trend of serum Tg over time should be monitored: a declining or a stable Tg is reassuring, whereas an increase should lead to imaging in order to localize and treat the disease and possibly RAI therapy. Summary of the European thyroid association consensus statement on RAI Therapy shown in Table 6.

14 Adjuvant Radiotherapy

The evidence for the use of adjuvant EBRT in advanced thyroid cancers is derived from retrospective studies, and it has been found to offer a small benefit in improving the locoregional control and in R1 resections with gross ETS and multiple nodes with perinodal extension [80]. In a systematic review by Fussey et al., it was found that the use of EBRT in patients with thyroid cancer improved the locoregional control in the high-risk cases and those aged over 45 years [81]. A case series from the Memorial Sloan Kettering Institute showed that EBRT was effective in advanced and recurrent thyroid cancers with acceptable acute toxicities [82]. IMRT has shown to have limited toxicity in these patients and should be the radiation technique of choice. However, there have been two large retrospective studies which did not show improvement in outcomes with the use of EBRT [83, 84].

Therefore, there is no consensus on the routine use of EBRT in patients with LATC with R0/R1 resection, and RAI therapy may suffice in them. In the absence of any prospective data on this issue, decisions for adjuvant RT should be made after a multidisciplinary discussion along with the radiation oncologist, endocrinologist, pathologist and nuclear medicine physicians. It is important to balance the risks with benefits prior to the decision of the use of EBRT in patients with complete resections and well-differentiated thyroid cancers.

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Parathyroid Glands

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1 Introduction

In patients with hyperparathyroidism, one or more parathyroid glands are surgically removed (parathyroidectomy). The only permanent cure for primary hyperparathyroidism, a disorder in which the parathyroid glands overproduce parathyroid hormone (PTH), is this procedure. A single parathyroid adenoma is present in the majority of people with primary hyperparathyroidism, but in a small percentage of cases, all four glands may be hyperplastic or there may be two adenomas. The conventional parathyroidectomy procedure entails examination of all four glands and removal of the adenoma in accordance with gland size. However, some surgeons have used minimally invasive surgery and quick intraoperative PTH monitoring to avoid examining all glands since the emergence of preoperative localization procedures, including radio-guided surgery, ultrasound (US) imaging, and high-resolution endoscopic techniques. The four tiny parathyroid glands, which are situated behind the thyroid gland, control the body's calcium levels by secreting PTH. The inferior parathyroid glands are often found below the thyroid gland and in front of the recurrent laryngeal nerve, while the superior parathyroid glands are typically found behind the recurrent laryngeal nerve. These glands can occasionally be found in unexpected places, such as the mediastinum, thymus, carotid sheath, lower neck, thyroid gland, and retro-esophageal region. The inferior thyroid artery and, in certain situations, the superior thyroid artery supply the parathyroid glands. When the amount of serum calcium drops, the parathyroid glands' main cells release PTH, which boosts calcium absorption in the kidney, encourages the conversion of 25-hydroxyvitamin D into active 1,25-dihydroxyvitamin D3, activates osteoclast activity in the bone, and raises serum calcium levels. Activated vitamin D levels rise in the intestines.

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2 Hyperparathyroidism

A condition known as hyperparathyroidism is defined by an excessive production of parathyroid hormone (PTH), which is brought on by an overactive parathyroid gland or glands. The most prevalent type of the disorder, primary hyperparathyroidism, is brought on by the autonomous hypersecretion of

N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_6

PTH from an aberrant parathyroid gland, usually an adenoma or hypercellular parathyroid gland. Primary hyperparathyroidism may occasionally be brought on by parathyroid cancer or multiple endocrine neoplasia (MEN) type 1, type 2A. High PTH levels as a physiological reaction to hypocalcemia, which is most frequently brought on by chronic renal disease or vitamin D insufficiency, result in secondary hyperparathyroidism. Patients with secondary hyperparathyroidism who have undergone kidney transplantation and continue to secrete PTH have tertiary hyperparathyroidism. PTH overproduction can cause kidney stones, osteoporosis, and symptoms in the neuromuscular system.

3 Preoperative Evaluation

For preoperative localization, a number of imaging tests are available, including ultrasound, magnetic resonance imaging (MRI), 99mTechnetium-sestamibi (MIBI), parathyroid four-dimensional computed tomography (4D-CT), parathyroid venous sampling, and positron emission tomography (PET). Preoperative imaging is largely acknowledged as standard of care and is advised by society guidelines; however there is no agreement on the best diagnostic imaging method, and clinical practice varies. The choice of imaging tests ultimately depends on the skill of the local radiologist, the preference of the surgeon, the availability, and the cost.

3.1 Ultrasound

Due to its accessibility, safety, accuracy, and affordability, ultrasound (US) is one of the most widely used and well-established imaging tools for preoperative localization in PHPT. A parathyroid adenoma often has the characteristics of an ovoid, homogenous, well-defined, hypoechoic (black) lesion (compared to thyroid tissue) in the traditional site of a parathyroid gland. Adenomas often have higher blood vessel density than lymph nodes and thyroid nodules. The performance of ultrasonography was evaluated by meta-analysis, which revealed a combined sensitivity or detection rate of 76.1% and a positive predictive value (PPV) of 93.2% [1]. The absence of ionizing radiation, the non-invasive approach, and relative affordability as compared to other imaging modalities are the key benefits of US. The portability of the US test enables for bedside and in-office imaging, in contrast to the MIBI scan's lengthy picture capture. One of US's drawbacks is that it is less adept at picking up ectopic glands or multiglandular illness. Patients with less severe disease (lower calcium and PTH levels), smaller adenomas, greater body habitus, or concurrent nodular thyroid disease tend to have ultrasounds that are less reliable.

3.2 Sestamibi Scintigraphy

A nuclear medicine gamma camera is used for imaging after injecting a radiotracer named 99mTechnetium-sestamibi (Tc-MIBI) intravenously (Fig. 1). In clinical practice, there are several alternative imaging techniques. These protocols vary in time (dual-phase MIBI imaging), extra radiotracer (dual-isotope subtraction), and

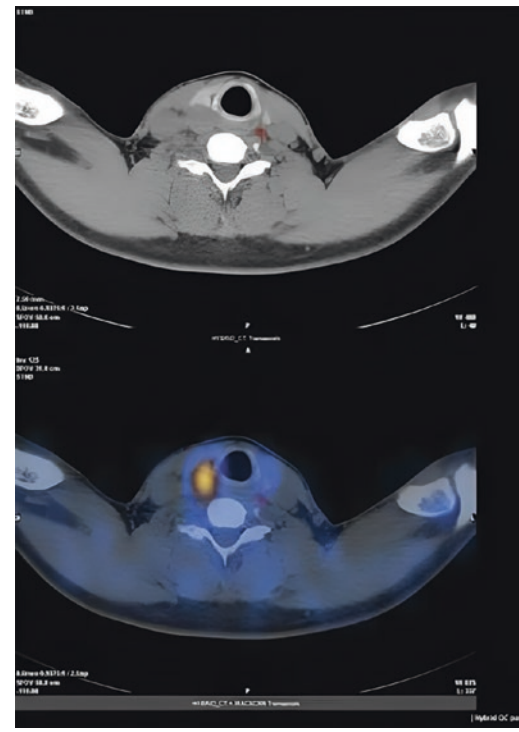

Fig. 1 Tc-99m-sestamibi parathyroid SPECT-CT scans showing right inferior lobe parathyroid adenoma

kind of acquisition (e.g., planar vs single photon emission computed tomography) (SPECT). According to a recent meta-analysis of MIBI SPECT/CT investigations using dual-phase or dual-isotope subtraction, the pooled sensitivity or detection rate was 88% and 88%, respectively [2]. MIBI offers a lower radiation dosage than 4D-CT and can be used in individuals who cannot receive iodinated CT contrast or undergo 4D-CT due to a contrast allergy or renal failure. Long imaging acquisition periods, long waits between early and delayed time points, higher cost compared to ultrasonography or 4D-CT, unusual washout patterns that make interpretation difficult, and false-positive accumulation in thyroid nodules are all disadvantages [3]. Parathyroid adenoma has better uptake when compared to parathyroid hyperplasia.

3.3 Parathyroid 4D-CT

A triple-phase CT of the neck and upper chest known as a 4D-CT is essentially a triple-phase CT with CT acquisitions made in noncontrast, arterial, and delayed phases, similar to triple-phase CTs of other body parts (i.e., CT of the liver). The axial, coronal, and sagittal anatomic body planes of CT scans are the first three dimensions of the term "4D," while contrast enhancement over time on serial phases is the fourth dimension [4]. 4D-CT showed sensitivity and specificity of 85% and 93%, respectively [5]. The primary benefit of 4D-CT is its high spatial resolution and outstanding anatomic detail, which enable the surgeon to precisely localize and delineate significant anatomic landmarks. Additionally, 4D-CT receives more radiation than MIBI.

3.4 MRI

MRI diagnostic imaging that offers both anatomical and functional details. MRI has been employed as a second-line imaging modality in the context of persistent or recurrent PHPT, a history of prior surgery, or in the case of negative or discordant results from first-line imaging modalities. Although less frequently employed than US and MIBI for preoperative localization, MRI has demonstrated comparable sensitivity in identifying aberrant parathyroid glands. On diffusion-weighted MRI, parathyroid lesions typically provide a greater (brighter) signal with increasing diffusion strength, which can help differentiate them from other soft tissue structures in the head and neck region. With the use of "4D" (time-resolved enhancement kinetics) approaches, MRI sensitivity rises to over 90% [6]. By better distinguishing the parathyroid glands from nearby lymph nodes and thyroid tissue, MRI can better distinguish minor parathyroid gland lesions and perhaps minimize the incidence of false positives in the neck. MRI frequently requires more time to gather images and is prone to motion and breathing aberrations.

3.5 PET/CT

Additional functional and metabolic data are provided by PET. To improve anatomic localization, PET/CT imaging fuses and combines PET and CT images. The most widely used PET tracer in the world is 18F-fluorodexyglucose (FDG); however its utility in detecting parathyroid carcinomas outweighs its limited ability to detect benign parathyroid adenomas. Choline PET has the highest diagnostic performance and clinical value among the PET tracers that have been evaluated for parathyroid imaging [7]. Increased uptake of choline is seen in abnormal but benign parathyroid cells due to upregulation of choline kinase related to the secretion of parathyroid hormone. Overall sensitivity of choline PET/CT was 95% and PPV 97% [8]. PET/CT may localize an abnormal parathyroid gland in 92% of patients with a negative US and/or MIBI imaging [9]. Currently, there is no accepted standardized protocol for imaging timing, dynamic acquisition phases, or the radiotracer dose [10]. FCH PET/CT has higher costs compared with conventional imaging modalities.

3.6 Parathyroid Venous Sampling

Selective parathyroid venous sampling (PVS), an invasive localization method, is now normally only used in individuals who have chronic or recurrent PHPT following parathyroidectomy and/or who

have had unfavorable or inconsistent imaging results. Typically done by a skilled interventional radiologist, venous samplings from veins draining the anterior mediastinum and around the thyroid bed are taken. The procedure's main goal is to localize the aberrant gland. When PTH levels are two times higher than in the inferior vena cava, there is an irregular gradient, and the territory of the draining vein can be used to determine the location of the hypersecreting gland [11]. Given the variability of sampling protocols, venous drainages patterns, as well as indications for selective parathyroid venous sampling, its diagnostic accuracy may be difficult to determine. The sensitivity of parathyroid venous sampling from the included studies ranges from 32% to 100% [12]. The advantages of selective parathyroid venous sampling are that it can guide surgery for patients who have failed a previous parathyroidectomy or localize a culprit parathyroid gland if non-invasive imaging modalities have been negative or inconclusive.

4 General Principles of Parathyroidectomy

- The main treatment for hyperparathyroidism is parathyroidectomy, which involves performing surgery to remove one or more parathyroid glands.
- In contrast to the conventional surgical method, which includes dissecting all four parathyroid glands, targeted parathyroidectomy can be utilized when a single, well-localized adenoma is present.
- Although targeted parathyroidectomy has similar clinical results to the conventional method, it has fewer general complications and requires less time to perform.
- Total or partial parathyroidectomy may be required in cases of secondary or tertiary hyperparathyroidism with parathyroid hyperplasia.
- Total parathyroidectomy carries a higher risk of long-term hypocalcemia and cardiovascular issues, while subtotal parathyroidectomy carries a higher risk of hyperparathyroidism recurrence.
- Parathyroid surgery can also make use of contemporary tools including radio-guided surgery, endoscopic-assisted parathyroidectomy, and ultrasound localization.

5 Parathyroidectomy for Primary Hyperparathyroidism

5.1 Case

A 41-year-old male presented with pain abdomen and chronic constipation. Serum corrected calcium levels were 12.8 g%. 4D-CT revealed solitary left superior parathyroid adenoma. Tc-99m-sestamibi parathyroid SPECT-CT scans are shown in Fig. 2.

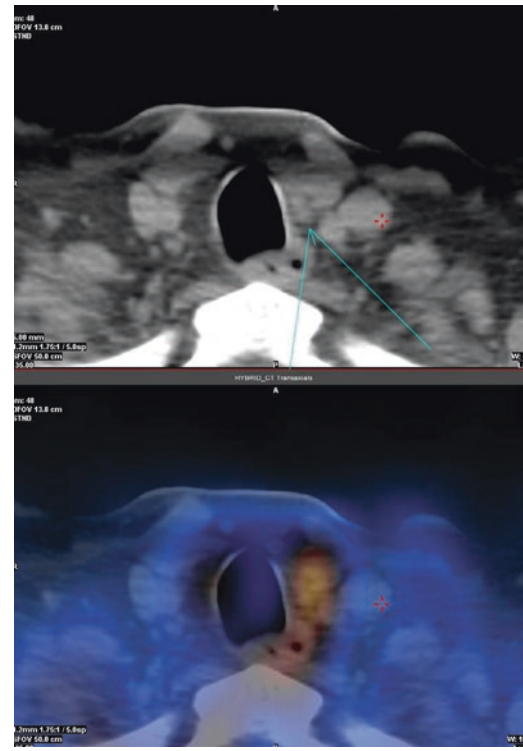

Fig. 2 Tc-99m-sestamibi parathyroid SPECT-CT scans showing left superior lobe parathyroid adenoma

5.2 Operative Technique

- In bilateral exploration, a transverse low collar incision is made about one finger wide above the clavicular head in an existing skin crease. The dissection is carried down through the subcutaneous tissue and the platysma muscle. Hemostasis is obtained by electrocautery or ligation using ties.
- Dissection continues to elevate the upper subplatysmal flap to the thyroid notch and the lower flap to the sternal notch.
- The deep fascia is opened by a midline incision along the median raphe of the strap muscles. Dissect the fascia along the anterior margins of the sternocleidomastoid muscle and either retract the strap muscles or divide the sternohyoid muscles and sternothyroid muscles vertically. Blunt-dissect the plane of cleavage between the sternocleidomastoid muscle and the outer boundaries of the sternothyroid muscle.
- A midline vertical incision is made between the sternohyoid muscles, extending from the thyroid notch to the level of the sternal notch. The sternohyoid muscles are then elevated to develop a plane between the thyroid gland and the sternohyoid muscles. Blunt dissection with the index finger or other blunt instruments such as a Kittner is performed in this plane to the lateral edge of the thyroid to mobilize the entire lateral surface of the thyroid and expose the paratracheal space between the trachea and carotid sheath.
- Ligate the middle thyroid vein, and then retract the thyroid lobe medially and anteriorly; the recurrent laryngeal nerve and the parathyroid glands can be identified at the upper and lower poles of the thyroid lobe. A similar exploration is made on the other side. The serum sample of PTH is collected for the baseline level.
- The solitary adenoma often appears to be enlarged in size and discolored. The enlarged gland is dissected and divided from the surrounding tissue without injuring the recurrent laryngeal nerve especially isolating the inferior parathyroid glands. It is important not to rupture the gland capsule in cases of malignancy but also in benign diseases to prevent parathyromatosis.
- The tissue specimen is sent for frozen section pathology to confirm if it is the parathyroid gland. The PTH serum level is repeated, and the removal of the overactive parathyroid gland is confirmed if the level drops to >50% and within the normal PTH range. Continue to explore additional adenoma if the PTH level doesn't fall to the expected level.
- Irrigate the wound, approximate the strap muscles with interrupted absorbable sutures, close the platysma with interrupted absorbable suture, and close the skin with a running subcuticular absorbable suture.
- With preoperative localization of abnormal parathyroid gland, focused parathyroidectomy can be performed only in the imaging identified area. The incision size, the extent of dissection, and the duration of surgery are limited.
- It is important to fully mobilize the thyroid to search for a missing parathyroid gland. If the upper gland is missing, explore the tracheoesophageal groove, the retropharyngeal space, and above the thyroid cartilage. The search for a missing lower gland should begin with a thorough exploration of the inferior pole of the thyroid and the surrounding soft tissue. If the gland is not found, performing a formal cervical thymectomy (or at least pulling up the thymus for examination is reasonable).
- In the patient with four gland hyperplasia, remove three-and-a-half glands. The remnant half gland can be left in situ or implanted into the sternocleidomastoid muscle. In some patients with secondary or tertiary hyperparathyroidism, total parathyroidectomy with subcutaneous forearm autotransplantation is recommended.
- For patients with multiple endocrine neoplasia (MEN) type 1-associated PHPT, the initial surgical procedure usually includes resection of three-and-one-half hyperplastic parathyroid glands (subtotal resection) with a strong

consideration for concomitant cervical thymectomy. For patients with MEN type 2A-associated PHPT, parathyroid hyperplasia is heterogeneous; bilateral exploration is usually performed as the initial procedure, and only the visibly enlarged glands are resected. For patients with MEN type 2A-associated recurrent hyperparathyroidism, a complete parathyroidectomy with forearm autotransplantation is typically performed.

Surgical steps are as depicted in Figs. 3, 4, 5, 6, 7, and 8.

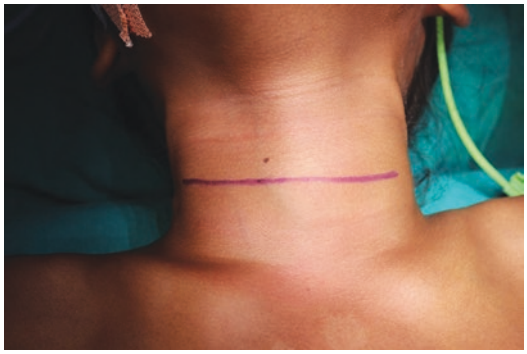

Fig. 3 A horizontal collar incision is preferred

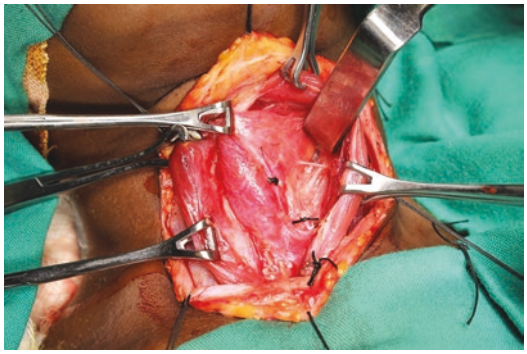

Fig. 4 Skin flaps are raised, and strap muscles are reflected. The sternocleidomastoid muscle is retracted laterally. The thyroid gland is medially rotated to visualize the parathyroid gland

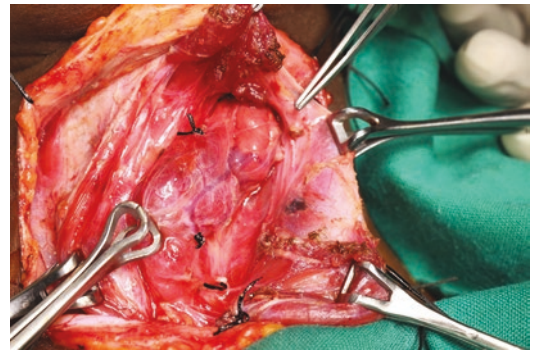

Fig. 5 Enlarged left superior parathyroid gland

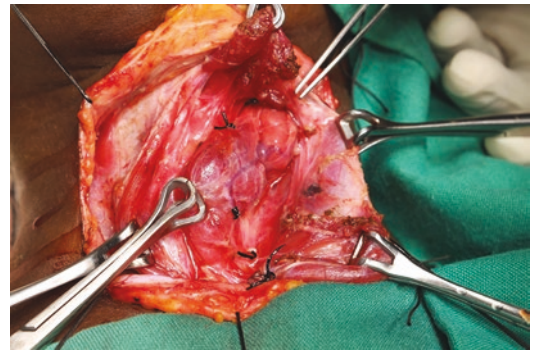

Fig. 6 The parathyroid gland is meticulously dissected all around

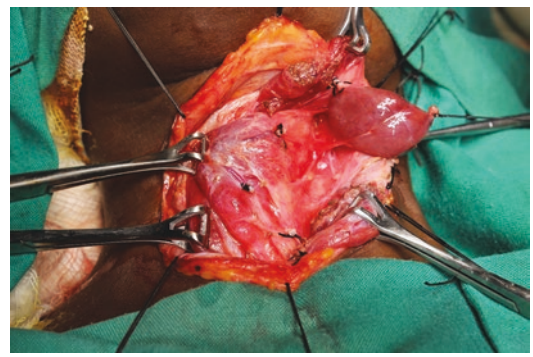

Fig. 7 Care must be taken to not injure the surrounding structures

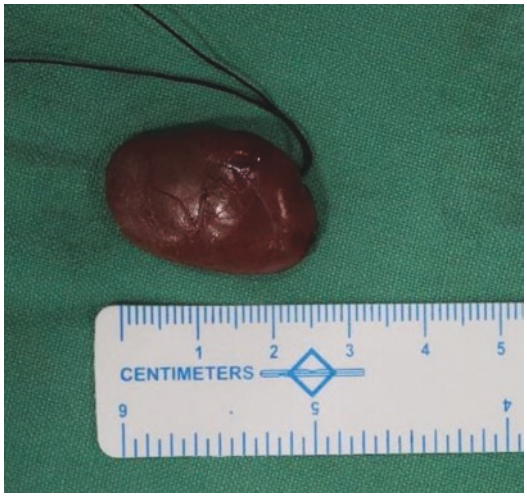

Fig. 8 Left parathyroid adenoma specimen

6 Parathyroidectomy for Secondary Hyperparathyroidism

6.1 Case

A 59-year-old lady presented with generalized pruritus and recurrent renal stones. Serum corrected calcium levels were 11.8 g%. 4D-CT localization study showed an enlarged left parathyroid gland. Tc-99m-sestamibi parathyroid SPECT-CT scans are shown in Fig. 9.

6.1.1 Operative Technique

Surgical steps are as depicted in Figs. 10, 11, 12, 13, and 14.

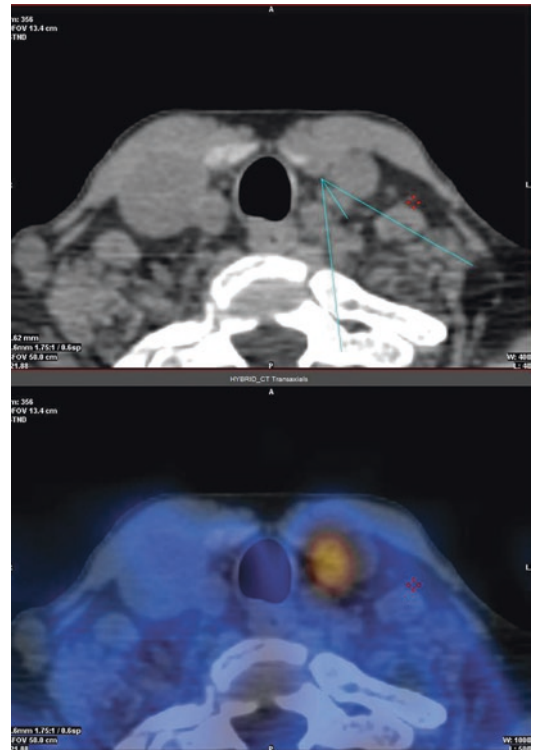

Fig. 9 Tc-99m-sestamibi parathyroid SPECT-CT scans showing left inferior lobe parathyroid adenoma

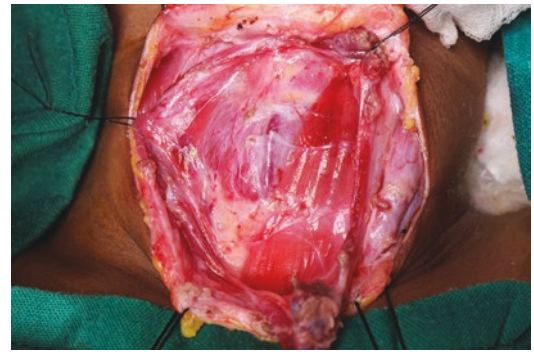

Fig. 10 Skin flaps are raised, and strap muscles are reflected to expose the thyroid gland

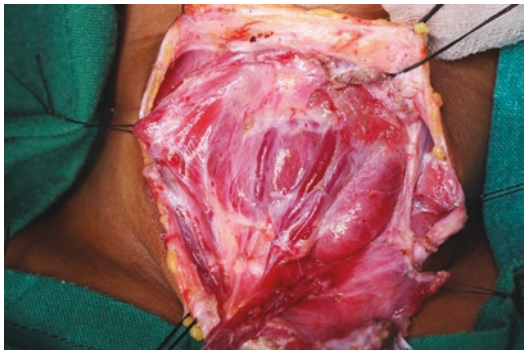

Fig. 11 The parathyroid gland is localized intraoperatively by retracting the thyroid gland and dissecting in the tracheoesophageal groove

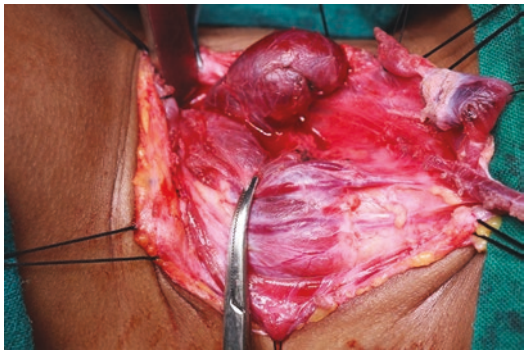

Fig. 12 The parathyroid gland is meticulously freed from the thyroid gland. Care must be taken to ensure there is no damage to the recurrent laryngeal nerve

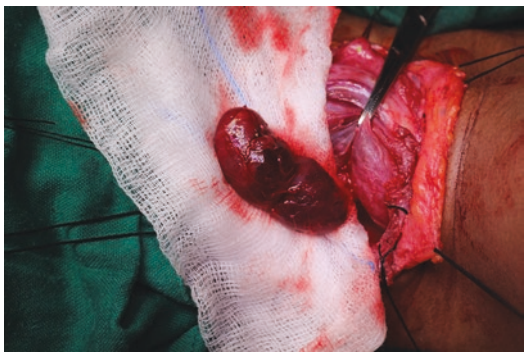

Fig. 13 The parathyroid gland is mobilized and dissected out

Fig. 14 Excised parathyroid adenoma

7 Parathyroidectomy for Tertiary Hyperparathyroidism

7.1 Case

A 64-year-old man, a known renal transplant patient on regular dialysis, presented with weakness, change in facial features, multiple fractures, and reduction in height. Serum corrected calcium levels were 13.8 g%. 4D-CT localization study showed enlarged three parathyroid glands. Tc-99m-sestamibi parathyroid SPECT-CT scans are shown in Fig. 15.

7.2 Operative Technique

Surgical steps are as depicted in Figs. 16, 17, 18, 19, 20, 21, and 22.

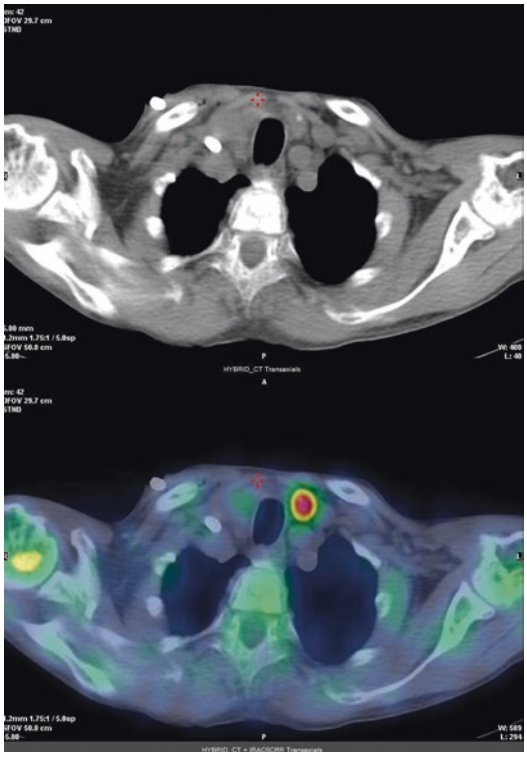

Fig. 15 Tc-99m-sestamibi parathyroid SPECT-CT scans showing left inferior lobe parathyroid gland having higher uptake

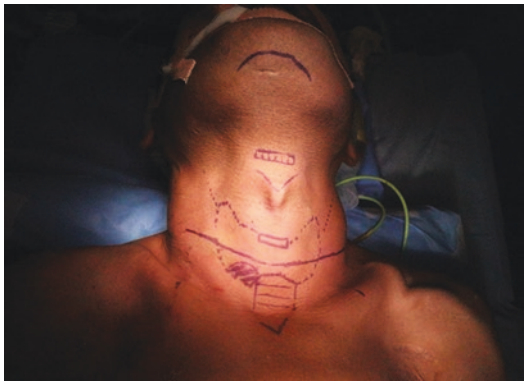

Fig. 16 A low cervical collar incision is given to facilitate the four-gland exploration

Fig. 17 The subplatysmal skin flaps are raised

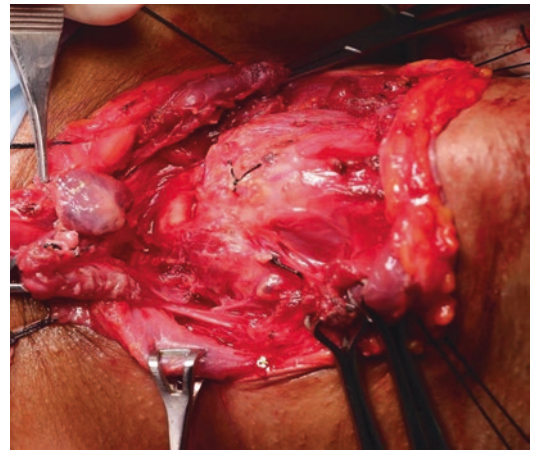

Fig. 18 Left inferior parathyroid adenoma excision

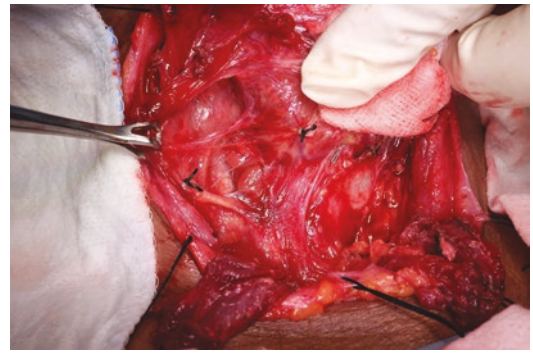

Fig. 19 Right inferior parathyroid adenoma excision

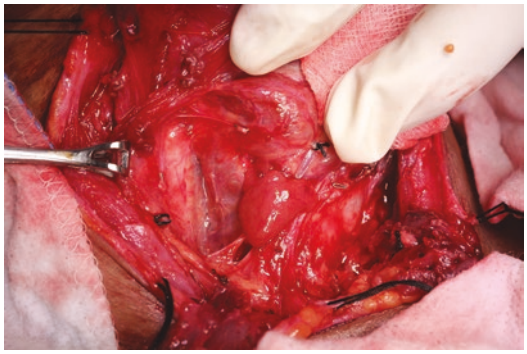

Fig. 20 Right superior parathyroid adenoma excision

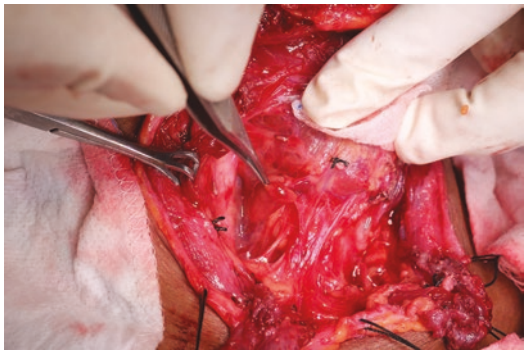

Fig. 21 Meticulous dissection was to ensure no injury to the surrounding structures

Fig. 22 Well-healed scar at the ninth month of follow-up

8 Complications Associated with Parathyroidectomy

Parathyroidectomy should be a relatively straightforward surgical procedure devoid of complications when carried out by a head and neck surgeon with appropriate training. Complications relating to the wound and damage caused to nearby structures should be of very low incidence. Complications relating to operative strategy, pathology (hyperplastic disease vs multiple adenomas), and failure to find an adenoma are much more complex and difficult to correct.

8.1 Wound Complications

Wound complications are hematoma, infection, and keloid formation.

8.2 Recurrent Nerve Injury

The recurrent nerve will be vulnerable to injury when dissection and removal of adenomas occurs close to the branches of the inferior thyroid artery.

Golden rules to help avoid nerve injury are:

- Stay close to the adenoma surface during dissection.
- Be exceedingly careful if diathermy is used—remember the circumference of thermal injury extends well beyond the points of the forceps, especially if monopolar diathermy is used.
- Be thoroughly conversant with the course of the nerve, and if the adenoma overlies or comes in close proximity, be sure that the nerve is seen and preserved throughout mobilization and dissection.
- Do not sling the nerve on tapes or threads and avoid handling or palpating the nerve.

8.3 Adenoma/Disease Not Found

The most important localization study is to identify a parathyroid surgeon. Such a surgeon

will successfully identify and remove the abnormal tissue in 97% of patients, resulting in eucalcemia [13].

If no abnormal glands are uncovered think:

- Incorrect diagnosis.
- The patient may have primary parathyroid hyperplasia—consider biopsy of one of the apparently normal glands.
- How many glands can be confidently identified—mark them with a silver clip and/or suture to aid subsequent localization by radiology or surgery.
- If only one gland is missing, this should help in focusing or directing appropriate exploration in that quadrant of the neck.
- The surgeon should be leaving the neck after a negative exploration having confidence that the tumor must be truly ectopic.

8.4 Persistent/Recurrent Disease

The criteria of Muller are as follows [14] (1975):

- Biopsy confirmation of all four glands.
- Removal of all abnormal tissue.
- Achievement of normal calcium for 1 year after surgery.
- Identification of abnormal pathology at the site of a previously normal gland.

8.5 Hypocalcemia

Contributions to the incidence and severity of hypocalcemia include the atrophy of residual glands in the face of a single hyperfunctioning adenoma (especially if there has been long-standing and significant hypercalcemia), so-called hungry bone syndrome and previous parathyroid or thyroid surgery. It may need treatment with vitamin D and/or calcium supplements. Very rarely a patient may need vitamin D and calcium supplements in the long term especially after reoperative parathyroid surgery or after resection of multiple gland disease.

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Oral Cavity and Neck Dissection

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and Ambesh Singh

Cancer of the oral cavity is one of the most common malignancies, especially in developing countries but also in the developed world. Squamous cell carcinoma (SCC) is the most common histology, and the main etiological factors are tobacco and alcohol use. Although early diagnosis is relatively easy, presentation with advanced disease is not uncommon. The standard of care is primary surgical resection with or without postoperative adjuvant therapy. Surgery remains the mainstay for nearly all tumours of the oral cavity. On the other hand, non-surgical approaches have shown equally effective tumour control and better functional outcomes in selected patients with tumours of the oropharynx. Multiple surgical approaches have been described for resection of primary oral cavity tumours, including peroral, mandibulotomy, lower cheek flap, visor flap and upper cheek flap approaches. The selection of a particular approach depends on several factors, including the tumour size and site, depth of infiltration and proximity to the mandible or maxilla. The principles of an adequate oncological resection should not be compromised by efforts to minimise the extent of the procedure. Improvements in surgical techniques combined with the routine use of postoperative radiation or chemoradiation therapy have resulted in improved survival statistics over the past decade. Successful treatment of patients with oral cancer is predicated on multidisciplinary treatment strategies to maximise oncologic control and minimise the impact of therapy on form and function.

Anatomy—The oral cavity includes the mucosa of the lips (not the external, dry lip), the buccal mucosa, the anterior tongue, the floor of the mouth, the hard palate and the upper and lower gingiva [1]. The anterior border of the oral cavity is defined by the portion of the lip that contacts the opposed lip (wet mucosa). The posterior border is defined by the circumvallate papillae of the tongue, the anterior tonsillar pillars (palatoglossus muscles) and the posterior margin of the hard palate. The hard palate defines the superior boundary of the oral cavity. Inferiorly, the oral cavity is defined by the mylohyoid muscles. The lateral boundary of the oral cavity is defined by the buccomasseteric region (buccal mucosa of the cheeks) and the retromolar trigone (which is located behind the mandibular third molar).

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_7

1 Preoperative Evaluation

The initial assessment of the primary tumour is based upon a thorough history and combination of inspection, palpation, indirect mirror examination or direct flexible laryngoscopy. Physical examination should include careful assessment of the nasal cavity and oral cavity with visual examination and/or palpation of mucous membranes, the floor of the mouth, the anterior two-thirds of the tongue, tonsillar fossae and tongue base (best seen on mirror examination or flexible laryngoscopy), palate, buccal and gingival mucosa and posterior pharyngeal wall.

For patients with non-laryngeal lesions but a strong alcohol or smoking history, flexible laryngoscopy is commonly undertaken to visualise potential other lesions and to document vocal cord mobility. A metastatic work-up with appropriate imaging is recommended for all newly diagnosed head and neck cancer patients, with particular attention to regional lymph node spread. For those with locoregionally advanced tumours, distant metastatic imaging, with attention to the lungs, is often performed. Patients with severe dysplasia or carcinoma in situ who have a strong smoking, alcohol or family history of cancer may also benefit from a more extensive work-up for metastases or a second primary including a screening chest computed tomography (CT). The use of clinical judgement is important. Visualisation of lesions outside the mouth is best accomplished by mirror examination and/or the use of a flexible fiberoptic endoscope with the goal of examining all of the mucosa in the nasopharynx, oropharynx, hypopharynx and larynx. Aside from mucosal irregularities, other abnormalities that should be specifically searched for are impairment of vocal cord mobility, pooling of secretions, anatomic asymmetries and bleeding. The appropriate nodal drainage areas are examined by careful palpation of the neck. Examination of the neck for pathologic adenopathy or other masses is best done according to neck levels. The parotid glands are also palpated for abnormalities. Due to improved radiologic and in-office biopsy techniques, an examination under anaesthesia (EUA) is most often performed only to obtain a tissue diagnosis, for surgical (e.g. robotic) planning and to search for carcinoma of unknown primary [2]. Symptom-directed panendoscopy (laryngoscopy, bronchoscopy and esophagoscopy) reveals a 2.4–4.5% incidence of second primary tumours of the upper aerodigestive tract but not of the lower airways [3].

1.1 Biopsy

Obtaining the tissue from the oral cavity is an essential first step towards the early identification of potentially malignant lesions and the development of targeted treatments and screening strategies. The common sequential analysis includes histopathological examination such as histopathological examination or immunohistochemistry (IHC) to identify various pathogenic features. Surgical biopsy is a gold standard for diagnosis [4].

1.2 Fine-Needle Aspiration Cytology

Fine-needle aspiration cytology (FNAC) is frequently used to make an initial tissue diagnosis of a head and neck cancer when a patient presents with a neck mass (metastatic cervical lymph node) without an obvious primary mucosal/upper aerodigestive tract site. This technique has high sensitivity and specificity and a diagnostic accuracy that ranges from 89 to 98% [5].

1.3 Computerised Tomography (CT)

CT can identify tumours of the head and neck based upon either anatomic distortion or specific tumour enhancement. Compared with MRI, CT provides greater spatial resolution, can be performed with faster acquisition times (thereby virtually eliminating motion artefact) and is better at evaluating bone destruction. CT technology that reduces metallic artefact is also being incorporated into routine clinical practice [6].

Modern multidetector CT allows scanning to be performed with slice thickness less than 1 mm.

Slice thickness of 3 mm is generally optimal, while slice thickness greater than 5 mm does not offer sufficient spatial resolution. Images should be reconstructed and viewed in both soft tissue and bone windows. Dental amalgam can create severe beam hardening image artefacts that obscure image details in the scan plane. This problem can be remedied by rescanning the obscured area with angulated gantry.

Primary site—For cancers of the oral cavity, contrast-enhanced CT can help determine the extent of tumour infiltration into deep tongue musculature and whether or not the mandible is involved. The "puffed cheek" technique improves evaluation of lesions of the oral cavity. This technique requires patients to self-insufflate their oral cavity with air by puffing out their cheek [7]. For other head and neck cancers, CT is particularly useful in upstaging cancers that have deeper local invasion or infiltration into adjacent structures that is difficult to detect on physical examination. Images are acquired in soft tissue windows and further reconstructed in bone algorithms with multiplanar reconstruction, which provides us insight into the spread of disease, relationship with important structures and the presence of bone erosion and perineural spread. Involvement of the gingivobuccal sulcus (GBS) and perineural spread going above the mandibular notch are best evaluated on coronal reformation. In contrast, the oblique sagittal view is best used for assessment of the retromolar trigone. Paramandibular soft tissue extent, mandibular foramen widening and extension into the high ITF are best evaluated on axial images. Coronal images are used for measuring the depth of invasion. Recently, the infratemporal fossa is subdivided into—Compartment 1, low ITF (medial pterygoid and masseter); Compartment 2, anterior high ITF (retroantral fat); Compartment 3, posterior high ITF; Compartment 3a, paramandibular compartment (paramandibular fat/temporalis); Compartment 3b, muscle compartment (lateral pterygoid); and Compartment 3c, perineural compartment (pterygopalatine fossa and pterygomaxillary fissure) [8].

Regional nodes—Imaging by CT or MRI is complementary to the clinical examination for the staging of the neck lymph nodes. CT evaluation of regional lymph nodes primarily relies upon size criteria as well as the appearance of lymph nodes to differentiate involved from uninvolved lymph nodes. The use of size criteria alone results in frequent false-positive and false-negative assessment of regional nodes. CT is also highly sensitive for detection of extracapsular spread of tumour. Pathologic lymphadenopathy is usually defined radiologically as a node greater than 10 to 11 mm in minimal axial diameter or one that contains central necrosis [9]. The choice of how a lymph node is measured is often controversial and reflects a trade-off between sensitivity and specificity. Other features that suggest pathological lymph nodes include rounded shape, loss of normal fatty hilum, increased or heterogeneous contrast enhancement, lymph node clustering and sentinel lymph node location [10].

1.4 Magnetic Resonance Imaging (MRI)

MRI provides superior soft tissue definition compared with CT and can often provide information that is complementary to CT [11]. For example, MRI can provide more accurate definition of tumours of the tongue and is more sensitive for superficial tumours. MRI is also better than CT for discriminating tumour from the mucus and in detecting bone marrow invasion. For this reason, MRI can be useful for evaluation of cartilage invasion, particularly for non-ossified cartilage that can pose difficulty for CT. On the other hand, CT scanning is better than MRI for detection of bone cortex invasion since MRI shows no bony detail. Multidetector computed tomography has a high specificity (87–90%), while MRI has a high sensitivity (96–97%) and a high negative predictive value for assessment of bone involvement; however, MRI has a low specificity (54%) for cortical invasion, making it a less valuable imaging tool for assessment of these malignancies. The oblique reformations are helpful for determining inferior alveolar canal involvement. Multiplanar and oblique reformations done on workstations with spatial cursor localisation further enhance the assessment of mandibular canal involvement. It is necessary to provide informa-

tion regarding sparing of the condyloid process and posterior segment of the mandible, which helps plan the reconstruction necessary after segmental mandibular resection [12].

MRI is superior to CT for evaluation of perineural spread, skull base invasion and intracranial extension of oral cancer. MRI may also provide additional benefits compared with CT in the evaluation of the base of the tongue and parotid glands. The most important imaging sequences for head and neck imaging include noncontrast-enhanced T1-weighted images, contrast-enhanced T1-weighted images with fat suppression and fat-suppressed fluid-sensitive sequences, such as T2-weighted images with fat suppression or short-tau inversion recovery (STIR) images. Images in axial and coronal plane are the most useful. For general purpose, slice thickness should be no more than 5 mm. Some applications, such as evaluation of skull base and perineural spread, may require thinner slice thickness, typically around 3 mm. In most studies, CT scanning outperforms MRI for the detection of pathologic nodal metastases. The reported sensitivity of MRI is as low as 57–67%.

1.5 Positron Emission Tomography with Computerised Tomography (PET/CT)

PET is superior to both CT and MRI for detecting regional nodal metastases, as well as distant metastases and second primary tumours. Its main utility is in finding occult distant metastases, unknown primary lesions and synchronous second primary tumours, as well as altering radiation fields and doses for patients who are not undergoing neck dissection.

Contemporary studies suggest that PET/CT may be beneficial and cost-effective for restaging of advanced head and neck cancer. Negative PET/CT findings after chemoradiation may accurately determine early disease response, making further surgical intervention unnecessary. In a prospective randomised controlled trial including patients with initial N2 or N3 disease treated initially by chemoradiation, patients undergoing PET/CT surveillance were shown to have similar survival rates compared with those with post-chemoradiation neck dissection [13].

1.6 Evaluation of Depth of Invasion

Accurate assessment of DOI is challenging on imaging, and it is important to understand that tumour thickness (TT) measured on imaging is not equivalent to DOI. DOI is measured as histologic tumour infiltration below the basement membrane which is equivalent to the endophytic component. CT and MRI may have limited utility in assessing DOI, and ultrasonography has been reported to be a more accurate method for assessing DOI [14]. DOI identified by the MRI has excellent correlation with the pathological DOI [15, 16].

1.7 Evaluation of Extranodal Extension

Extranodal extension (ENE) is a clinical and histopathological finding; imaging can also be used as an adjunct in evaluating macroscopic ENE. Imaging features such as perinodal fat stranding and infiltration into the adjacent fat or muscle have high specificity for predicting ENE. Indirect imaging signs of ENE, especially in a high level II node, include denervation atrophy or signs of dysfunction of muscles innervated by the upper cranial nerves IX–XII [17]. It should be noted that in addition to imaging evidence of ENE, the AJCC staging system also requires the presence of clinical findings of gross ENE for a patient to be upstaged based on ENE.

1.8 Evaluation of Distant Metastasis

A component of the initial staging evaluation for patients with new or recurrent head and neck can-

cer is the search for distant metastases. The reported incidence is between 2 and 26% and varies based on locoregional control, nodal involvement (number and presence of extracapsular extension), primary site, histologic grade and T stage [18].

Distant metastases at initial diagnosis are usually asymptomatic; the most common sites are the lungs followed by the liver and bone. Screening tests such as chest radiograph, ultrasound abdomen, serum alkaline phosphatase and liver function tests are insensitive to the presence of distant metastases [19]. CT scan was the most sensitive method to screen for distant metastases in patients with head and neck cancer, identifying malignant findings in between 4 and 19% of newly diagnosed cases [20]. Although chest CT detects distant metastases more frequently than chest radiograph, it fails to detect the 2–5% of patients who will have distant metastases outside the chest [21].

PET/CT has greatly replaced other tests for detection of distant metastases and synchronous second primary tumours. However, false-positive findings are common, underscoring the need to undertake histologic confirmation of any sites of abnormal uptake. PET/CT usually does not pick up a subcentimetric tumour [22].

2 Principles of Management of Tongue Cancers

Multiple surgical approaches have been described for resection of primary oral cavity tumours, including peroral, mandibulotomy, lower cheek flap, visor flap and upper cheek flap approaches. The selection of a particular approach depends on several factors, including the tumour size and site, depth of infiltration and proximity to the mandible or maxilla. The principles of an adequate oncological resection should not be compromised by efforts to minimise the extent of the procedure:

- Small tumours in easily accessible locations of the oral cavity are safely excised via an open mouth approach. Thus, small primary tumours of the oral tongue, floor of the mouth, gum, cheek mucosa and hard or soft palate are suitable for peroral excision. However, in patients with trismus or restricted oral apertures to provide access to the tumour, alternative approaches are required to allow for satisfactory resection.
- Peroral partial glossectomy: All T1 and most T2 lesions of the oral tongue—that is, the anterior two-thirds of the tongue—are suitable for a partial glossectomy through the open mouth.
- Tumours involving the lateral aspect of the oral tongue are amenable to a wedge excision oriented in a transverse fashion.
- Longitudinally oriented excision of a large tumour results in an elongated narrow tongue that often impairs speech and interferes with mastication. In contrast, transversely oriented wedge resections foreshorten the tongue, resulting in functionally and cosmetically superior results.
- The procedure is best performed by electrocautery or ultrasonic coagulation device (harmonic scalpel).
- First, the cutting current is used to incise the mucosa of the tongue on both its superior and inferior aspects. Following the mucosal incision, the coagulating current is used for resection of the underlying musculature of the tongue. Minor bleeding points in the musculature of the tongue are electro-coagulated, while major branches of the lingual artery are ligated with ties.
- The excision should include a generous margin of the mucosa around the tumour and a full complement of the (third dimension) thickness of the musculature of the tongue surrounding the palpable tumour.
- Frozen sections are taken from the mucosal margins and the depth of the surgical defect to ensure adequate excision of the primary tumour.
- Repair of the surgical defect follows confirmation of negative margins by frozen section and attainment of complete haemostasis.

- The patient can take clear liquids by mouth within 24–48 h and is allowed a pureed diet on the third postoperative day. Most patients will be able to tolerate a soft diet by the end of the week.

The wide variation in the presentation of the lesion over the tongue is depicted in Figs. 1, 2, 3, 4, 5, 6, 7 and 8.

Fig. 1 Homogeneous leukoplakia over the right lateral border of the tongue

Fig. 2 Ulceroproliferative growth on the ventral surface and tip of the tongue

Fig. 3 Small growth on the left lateral border of the tongue

Fig. 4 T2 lesion on the right lateral border of the tongue with grade II trismus

Fig. 5 Ulceroinfilterative growth on the lateral border of the tongue: one must be very careful while resecting the base; palpating the base in every step is a must to ensure adequate margin in the third dimension

Fig. 6 Growth on the left lateral border with good mouth opening, a good candidate for peroral resection

Fig. 7 Growth on the dorsum of the tongue, such lesions entail anterior second/third glossectomy

2.1 Types of Glossectomies

This was proposed by Ansarin and colleagues in 2019 [23]:

- Type I glossectomy (mucosectomy)—The mucosa and submucosa are included up to the intrinsic muscle fibres of the tongue. The deep resection margin should include a thin layer of the intrinsic muscles because of a possible invasion of the submucosa. Generally, the wound is left to heal by secondary intention, although the defect may be partially closed primarily:
  - Indication: Precancerous, superficial suspicious lesions, limited to the epithelium of the tongue without previous biopsy
- Type II glossectomy (partial glossectomy)—It includes the lesion and adjacent normal mucosa, submucosa and the intrinsic muscles up to the surface of the extrinsic muscles (when the directions of the muscle fibres change), with appropriate safety margins:
  - Lesions infiltrating the submucosa and superficially into intrinsic muscles, but not extrinsic muscles, or infiltration less than 10 mm deep
- Type IIIa glossectomy (hemiglossectomy)—The specimen includes the mucosa, submucosa and intrinsic and extrinsic muscles ipsilateral to the lesion. The mucosa is resected up to the healthy tissue with appropriate safety margins (at least 1.5 cm); the lingual artery must be ligated and removed en bloc with the lingual and hypoglossal nerves, in the speci-

Fig. 8 Posteriorly based growth on the left lateral border of the tongue: the posterior margin is not visible adequately. A cheek flap approach may be required in such cases

men of the primary tumour and neck nodes. The base of the ipsilateral tongue is preserved. The tip of the tongue can be preserved or not:
  - Lesions infiltrating the intrinsic and minimally extrinsic muscles or infiltration greater than 10 mm but confined within the ipsilateral tongue
- Type IIIb glossectomy (compartmental hemiglossectomy)—The specimen includes the mucosa, submucosa, intrinsic and extrinsic muscles ipsilateral to the lesion, genioglossus, hyoglossus and styloglossus muscles and the inferior portion of the palatoglossus muscle. Medially, the midline raphe is included in the resection. The lingual nerve is resected as far cranially as possible. The hypoglossal nerve is removed after the ansa, the lingual artery and vein are ligated in proximity to the horn of the hyoid bone and removed en bloc with specimen and neck nodes:
  - Lesions massively infiltrating the intrinsic and extrinsic muscles but confined to the ipsilateral tongue
- Type IVa glossectomy (subtotal glossectomy)—This is an anterior subtotal glossectomy with preservation of both sides of the base of the tongue, posterior hyoglossus muscle and hypoglossal and lingual nerves, from the less involved side:
  - Lesions that arise in the anterior portion of the mobile tongue and exceed the hemilingual area of origin involving the contralateral genioglossus muscle but limited to mobile tongue
- Type IVb (near-total glossectomy)—Type IVa glossectomy with extension to the ipsilateral base of the tongue. The following contralateral structures are preserved: hyoglossus and styloglossus muscles, hypoglossal and lingual nerves and lingual artery (functional unit of the base of the tongue):
  - Massive lesions that exceed the border of the hemilingual area of origin infiltrating the ipsilateral base of the tongue and the contralateral genioglossus muscle
- Type V glossectomy (total glossectomy)—The specimen includes all of the mobile tongue and the base of the tongue transected at the level of the vallecula; it includes intrinsic and extrinsic muscles, both lingual arteries, hypoglossal, lingual nerves and the floor of the mouth:
  - Massive infiltrating lesions, for instance, those of the anterior ventral surface of the tongue, dorsum of the tongue or the tongue base, which bilaterally involve the extrinsic genioglossus, hyoglossus and styloglossus with impairment of the mobility of the tongue.

2.2 Indications for Peroral Approach

- Good mouth opening.
- All of the tumour margins are visible.
- Anteriorly placed tumours.
- Able to perform satisfactory reconstruction.

2.3 Indications for Lower Cheek Flap Approach

- Trismus.
- Deep infiltrative tumours.
- Posteriorly placed tumours.
- Tumours reaching the base of the tongue.
- Need for composite resection.

2.4 Indications for Visor Approach

- Trismus.
- Posteriorly placed tumours.
- Total or near-total glossectomy.

Follow-up photographs of surgical excision of tongue malignancy are depicted in Figs. 9, 10, 11, 12, 13 and 14.

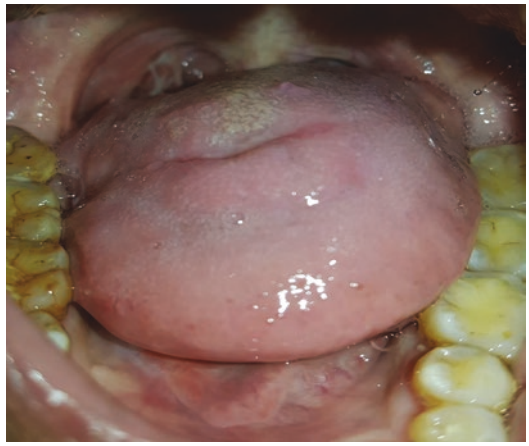

Fig. 9 Wedge primary closure following tongue-wide local excision

Fig. 10 Longitudinal closure helps in maintaining the tip and tongue length

2.5 Reconstruction Options for Tongue Defects

- Partial glossectomy defects:
  - Primary closure:
    - Wedge closure.
    - Longitudinal.
  - Healing by secondary intention.
  - Pedicled flaps:
    - Facial artery myomucosal flaps.
    - Nasolabial flaps.
    - Submental flaps.
    - Infrahyoid flap.
    - Supraclavicular flap.
    - Pectoralis major myofascial flap (PMMF).
  - Free Flap.
    - Radial free forearm flap.

Fig. 11 Tongue defect left to heal by secondary intention

Fig. 12 Follow-up after 6 months

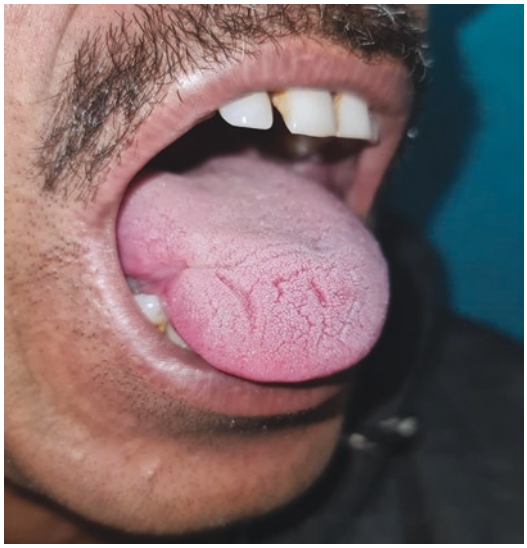

Fig. 13 One-year follow-up after wedge closure

Fig. 14 One-month follow-up following Trotter's procedure for the base of tongue tumour excision

- Hemiglossectomy defects.
  - Pedicled flaps.
    - Facial artery myomucosal flaps.
    - Nasolabial flaps.
    - Submental flaps.
    - Infrahyoid flap.
    - Supraclavicular flap.
    - Pectoralis major myofascial flap (PMMF).
  - Free Flap.
    - Radial free forearm flap.
    - Anterolateral thigh flap.
    - Free fibula osseocutaneous flap (FFOCF) for composite defects.
- Total or near-total glossectomy.
  - Pedicled flaps:
    - Pectoralis major myocutaneous flap.
  - Free flaps:
    - Free radial forearm flap.
    - Free anterolateral thigh flap.

The specimen of tongue composite lingual plate marginal mandibulectomy is shown in Fig. 15.

Surgical steps for islanded nasolabial flap for reconstruction following tongue-wide local excision are depicted in Figs. 16, 17, 18, 19, 20 and 21.

Patient underwent tongue-wide local excision with free radial forearm flap reconstruction that is shown in Figs. 22 and 23.

Follow-up photos are depicted in Figs. 24, 25, 26, 27, 28, 29 and 30.

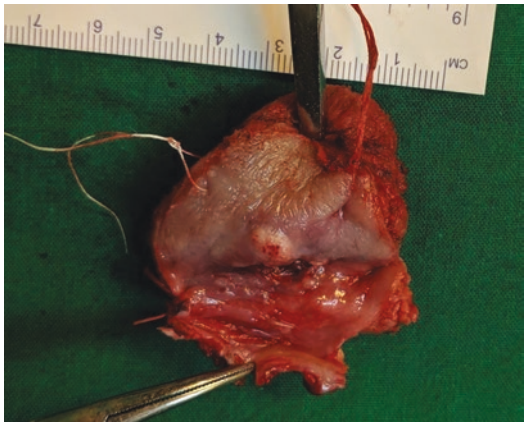

Fig. 15 Specimen of tongue composite lingual plate marginal mandibulectomy

Fig. 16 Marking for islanded nasolabial flap reconstruction for tongue defects

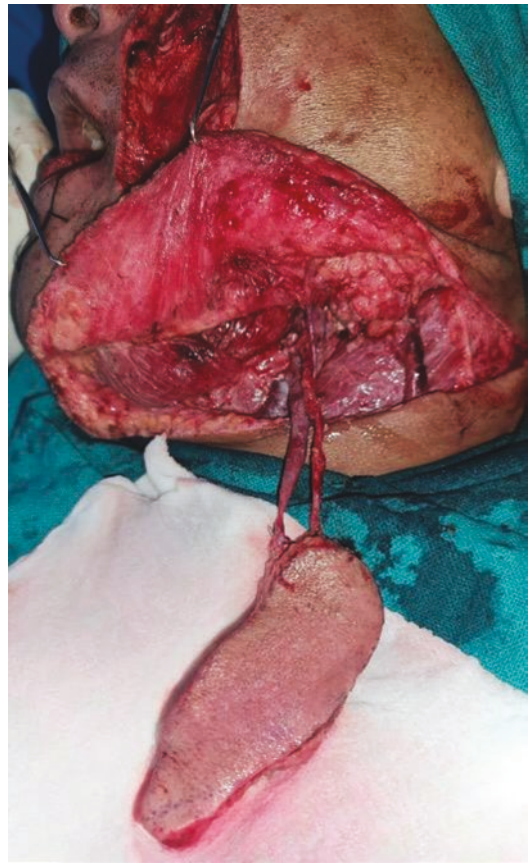

Fig. 17 Islanded nasolabial flap with its intact vessels

Fig. 18 Islanded nasolabial flap with its intact blood vessels

Fig. 19 Following islanded nasolabial flap inset

Fig. 20 Intraoral photo showing the nasolabial flap fitting into the defect like a jigsaw

Fig. 21 Hair growth on the flap indicates good flap vascularity. Depilation may be required if hair growth is troublesome

Fig. 22 McGregor incision and cheek flap approach for the tongue composite resection

Fig. 24 Excellent scar following nasolabial flap reconstruction

Fig. 23 Postoperative picture showing the free radial artery forearm flap reconstruction following hemiglossectomy composite segmental mandibulectomy

Fig. 25 Well-healed scar following tongue composite resection

Fig. 26 En face view shows negligible scar following reconstruction

Fig. 27 One-year follow-up showing good graft uptake in the left radial forearm

Fig. 28 Excellent mucosalisation of the islanded nasolabial flap, fourth month of follow-up

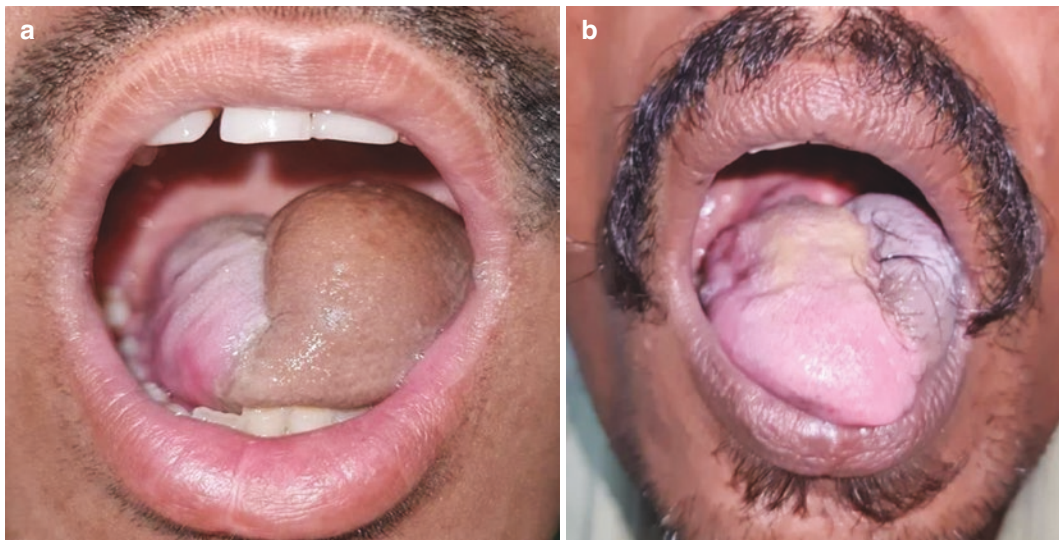

Fig. 29 (**a**, **b**) One-year follow-up after hemiglossectomy with free radial forearm flap

Fig. 30 Total glossectomy with free radial forearm reconstruction

3 Principles of Management of Buccoalveolar Cancers

Carcinoma of the buccal mucosa represents 5–10% of all oral cavity malignancies and has a 4:1 male predominance; the typical patient is in the sixth decade of life. An association between smokeless tobacco use and buccal carcinomas has been noted, and an increased incidence of these tumours is seen in the western population. In India, betel nut use is associated with a high incidence of buccal carcinoma.

The site of the buccal mucosa where squamous carcinoma is most frequently noted is adjacent to the third mandibular molar. Patients with advanced-stage disease may present with trismus as a result of direct invasion of the tumour into the muscles of mastication, and parotid duct involvement may produce obstructive sialadenitis. Anterior lesions metastasise regionally to submandibular nodes, whereas posterior buccal lesions metastasise to upper jugulodigastric lymph nodes:

- Small lesions can be excised transorally.
- Intermediate-stage primary tumours may be resected transorally or through a lip-splitting incision. With the exception of superficial lesions, the buccinator muscle should be resected in continuity to provide adequate margin depth.
- Adequate subcutaneous fat must be left behind to ensure viability of the skin, whenever oncologically safe. Palpation must be done at every step to ensure the button hole of the buccal skin is not created.
- Local intraoral spread may necessitate resection of the alveolar ridge of the mandible or maxilla. Deep invasion into the cheek may require through-and-through resection.
- Peroral: Small anterior lesions are accessible without an external incision. However a peroral approach should be avoided in posterior lesions even if they are small. It is pertinent to resect an adequate base with wide margins as the base is the commonest site of compromised margins with peroral excision. Occasionally an anterior lesion requiring marginal mandibulectomy can be approached perorally.
- Cheek flap: Most lesions of the buccal mucosa (with above exceptions) are approached via a cheek flap with straight lip split, angle split or McGregor incision.
- Midline lip split: When a lesion is located away from the oral commissure, the lip is split in the midline. This incision maintains better oral competence. The incision is continued over the mentum, curving towards the hyoid up to the mastoid process along a suitable skin crease at least two finger breadths below the mandible. The midline lip split incision can be modified for better cosmesis.

The wide variation in the presentation of the lesion over the buccal mucosa is depicted in Figs. 31, 32, 33, 34, 35 and 36.

3.1 Management of the Mandible

- Periosteum is a robust barrier to bone invasion. The mandible is generally invaded via its occlusal surface in alveolar lesions with intact dentition. In the retromolar trigone or in eden-

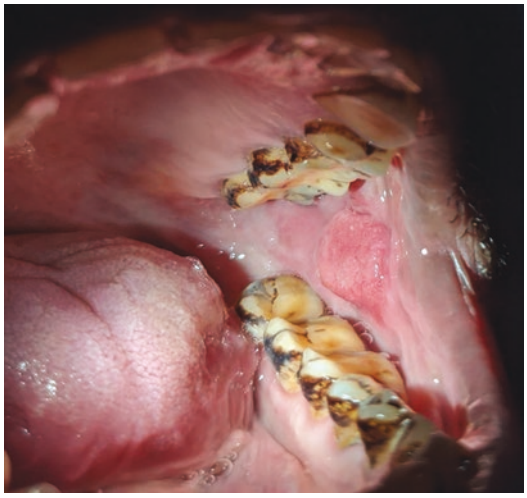

Fig. 31 Growth in the left buccal mucosa

Fig. 33 Verrucous growth in the left buccal mucosa

Fig. 32 Hypertrophic leukoplakic patch in the right buccal mucosa

Fig. 34 Ulceroproliferative growth in the left buccal mucosa

tulous mandibles, the occlusal surface corresponds to the junction of the attached and reflected mucosa (point of abutment).

- In irradiated mandibles and in large tumours, the mandible can be invaded at multiple points due to multiple breaks in the periosteum.
- With cancers of the floor of the mouth, the mandible is infiltrated at the point of abutment. Hence a horizontal marginal mandibulectomy can be attempted in early gingivobuccal complex cancer lesions to include invasion at the occlusal surface; the floor of mouth cancer requires vertical or oblique marginal mandibulectomy to completely excise the lingual plate due to invasion occurring directly at the point of abutment.
- *Marginal mandibulectomy*—Marginal mandibulectomy is indicated when tumour abuts the mandible without gross invasion or when there is only superficial bony invasion (Figs. 37, 38, 39, 40, 41 and 42):

Fig. 35 Growth in the left buccal mucosa close to the lower gingivobuccal sulcus and alveolus. Marginal mandibulectomy may be necessary to achieve adequate margins

Fig. 36 Growth in the right retromolar trigone

Fig. 37 Defect following left buccal mucosa composite marginal mandibulectomy

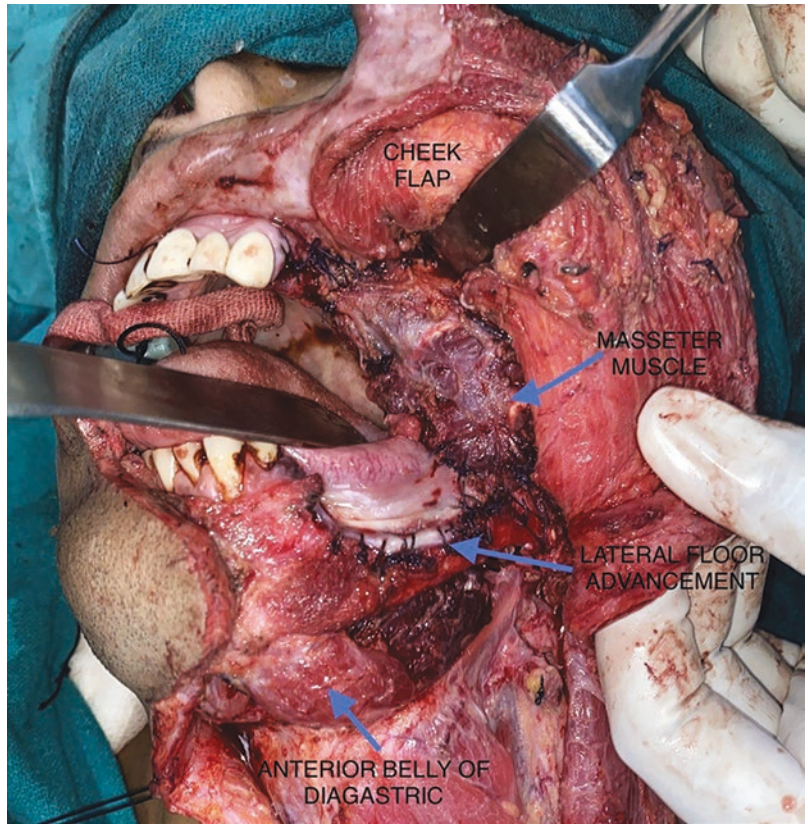

Fig. 38 The floor of the mouth can be advanced laterally to close the marginal mandibulectomy

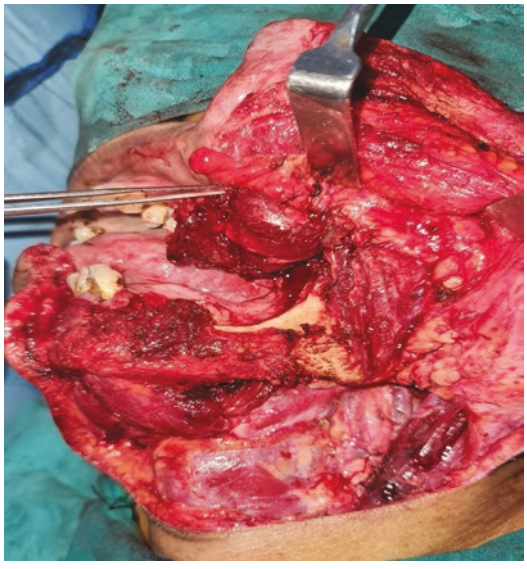

Fig. 39 Masseteric flap for the marginal mandibulectomy defect

Fig. 40 Marginal mandibulectomy defect closure with the masseteric flap

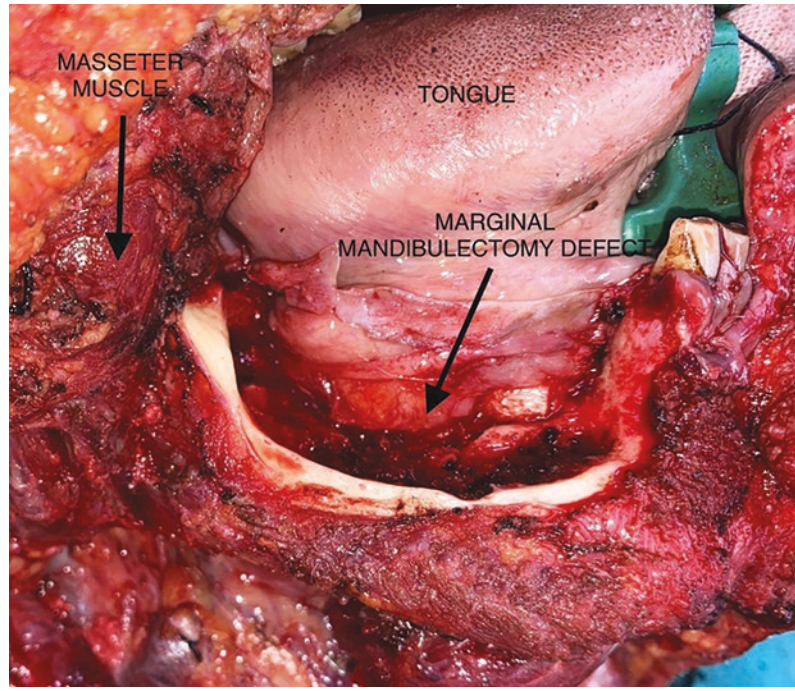

Fig. 41 Marginal mandibulectomy defect

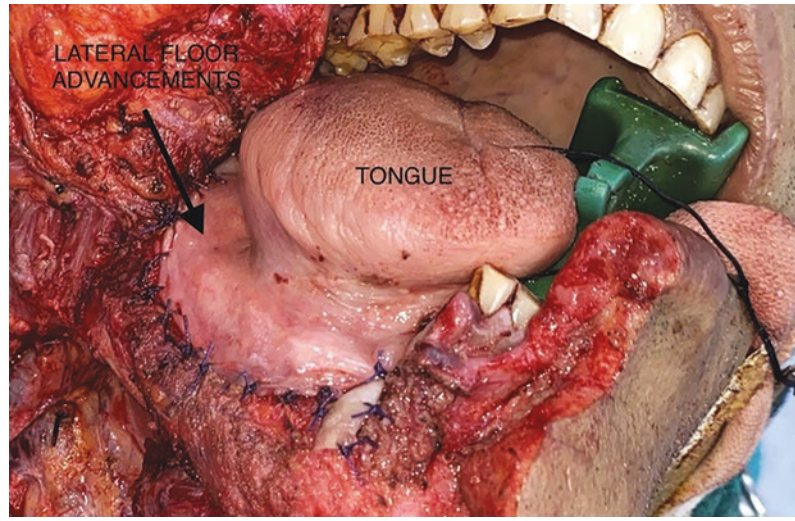

Fig. 42 Marginal mandibulectomy defect closure with the lateral floor of mouth advancement

- Three types of marginal mandibulectomy are described, i.e. horizontal, vertical and oblique.
- As the lingual plate is weaker than the buccal plate, an isolated buccal plate excision may not withstand subsequent weight bearing, and the bone may fracture. Hence isolated buccal plate excision is risky in buccal mucosa lesions.
- The theory of preferential route of tumour entry through the inferior alveolar nerve has been refuted by multiple studies; hence it is no longer advocated to include the inferior alveolar nerve up to the skull base with a rim resection.
- Adequate exposure is achieved with a lower cheek flap. Occasionally, a small anterior lesion can be approached perorally.

  - The edges of the bone should be "canoe shaped" to avoid sharp corners, and angled cuts predispose to stress fractures and hence are avoided. Inferiorly a bony bridge of >1 cm in height should be retained to avoid a stress fracture.
  - The bone is cut with sharp bone-cutting instruments. Finally the corresponding gingivolingual sulcus is divided to deliver the specimen.
  - With marginal mandibulectomy for retromolar trigone cancer, the anterior aspect of the ascending ramus of the mandible is excised in continuity with the coronoid process, as releasing the attachment of the temporalis muscle avoids postoperative trismus.
- Segmental and hemimandibulectomy—This is indicated when there is gross bone erosion, either clinically or radiologically, for significant paramandibular disease, for postradiotherapy recurrence due to the multiple routes of tumour entry or with a pipe stem mandible (inadequate bony remnant of <1 cm in height) (Figs. 43, 44, 45, 46, 47 and 48):

Fig. 43 Skin marking for the complex composite segmental mandibulectomy with skin excision and bilateral neck dissection

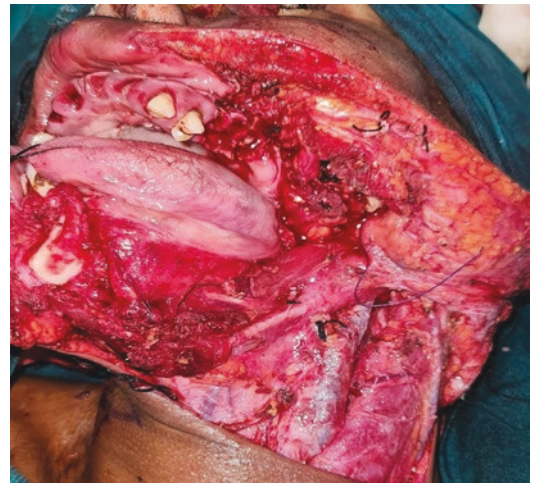

Fig. 44 Defect of complex bite resection

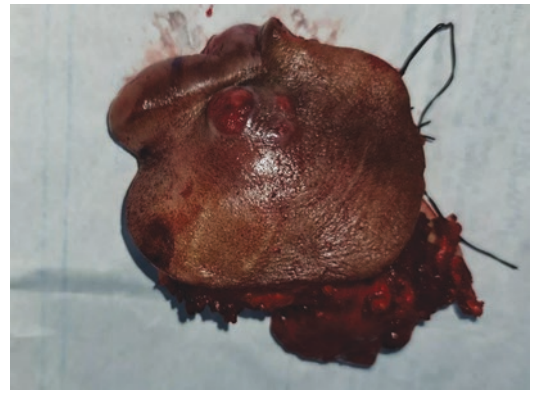

Fig. 45 Specimen of bite composite segmental mandibulectomy

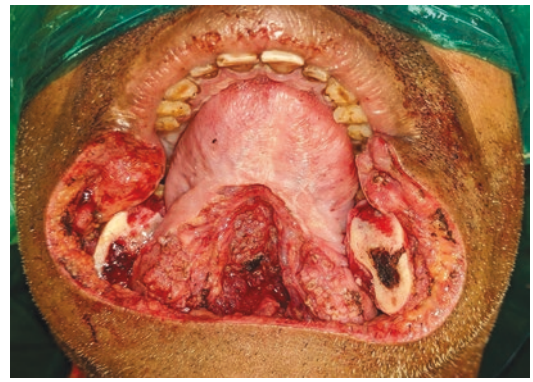

Fig. 46 Composite middle segmental mandibulectomy defect

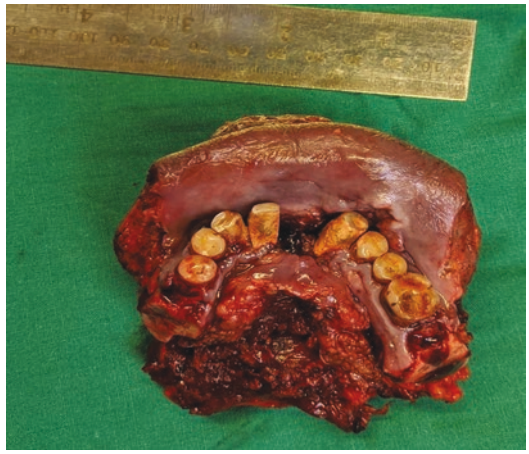

Fig. 47 Specimen of the central arch of the mandible with the lower lip and skin

Fig. 48 Nerve anastomosis with the lateral antebrachial cutaneous nerve of the arm with greater auricular nerve

- Segmental mandibulectomy may encompass the mandibular arch (mid-third segment) or may be arch-preserving (lateral segment).
- Mandibulectomy that includes the entire ascending ramus, condyle and coronoid can either be a posterior segmental mandibulectomy (posterior to mental foramen) or hemimandibulectomy.

3.2 Resecting the Primary

- After incising the mucosa around and soft tissue using diathermy or a knife, bone cuts are marked adjacent to the soft tissue.
- The posterior mucosal cut is made according to the extent of tumour. Attention should be paid mainly to the third dimension, i.e. deep resection margin.
- This margin must contain at least one layer of the normal tissue beyond the tumour. With this in mind, it should contain the buccinator muscle with superficial lesions and the buccal fat pad or the zygomaticus major muscle with deeper lesions.
- With lesions deeper than that, e.g. adhering to the skin or causing peau d'orange, the overlying skin is excised to achieve an adequate margin.

Surgical specimen following buccal mucosa-wide local excision with marginal mandibulectomy or bite resection is depicted in Figs. 49, 50, 51, 52, 53 and 54.

The surgical steps of the modified Estlander flap to reconstruct the angle of the mouth are depicted in Figs. 55, 56 and 57.

The surgical steps of buccal fat pad grafting and split-thickness skin grafting are depicted in

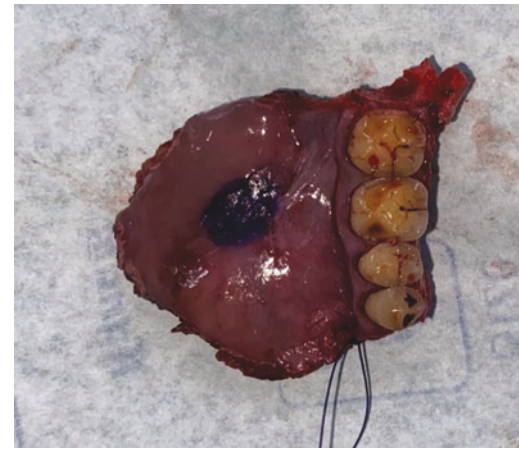

Fig. 49 Specimen showing buccal mucosa composite marginal mandibulectomy

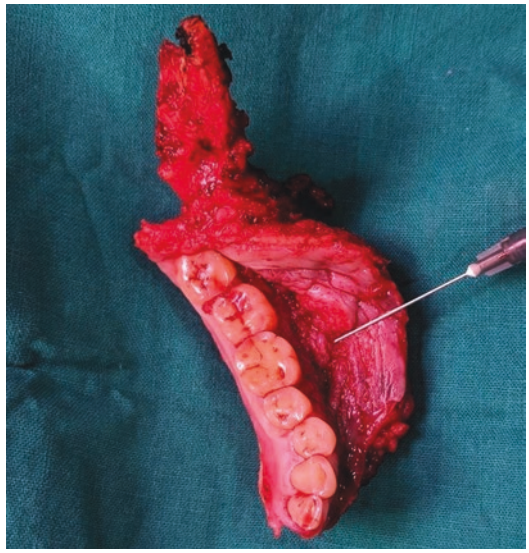

Fig. 50 Specimen of buccal mucosa composite marginal mandibulectomy with the pin at the tumour

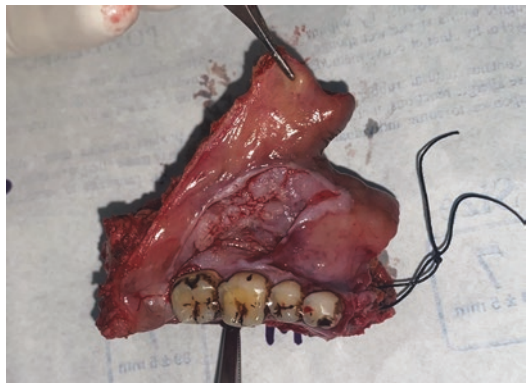

Fig. 51 Composite buccal mucosa with segmental mandibulectomy with oral commissure specimen

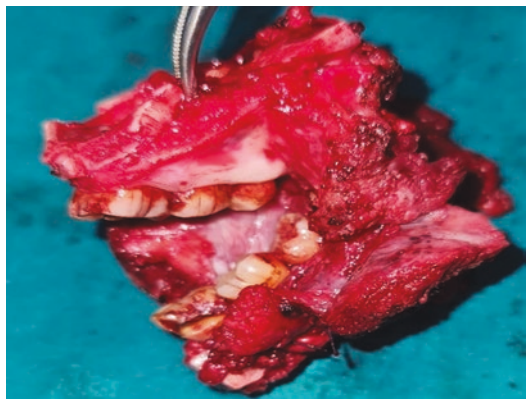

Fig. 52 Bite resection specimen (infrastructure maxillectomy with marginal mandibulectomy with intervening buccal mucosa). * upper jaw and ** lower jaw

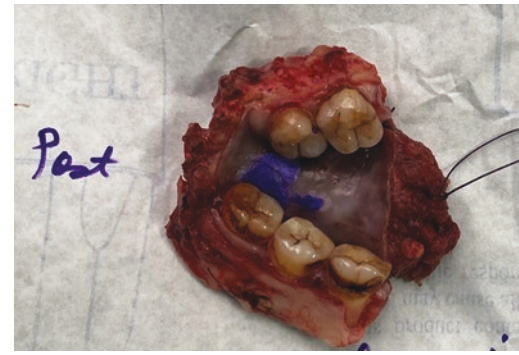

Fig. 53 Specimen of bite marginal resection. * upper jaw and ** lower jaw

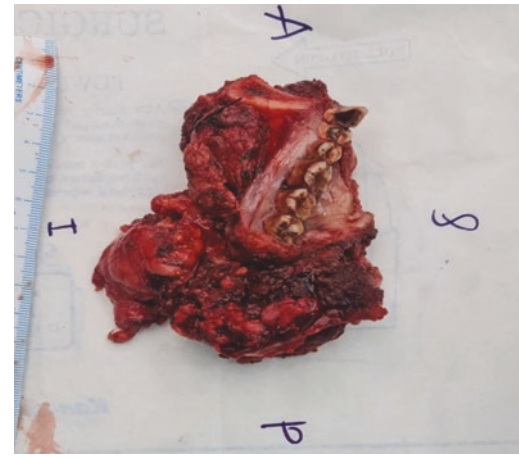

Fig. 54 Posterior segmental mandibulectomy with level IB with the primary resection specimen; this ensures that paramandibular disease is not cut through

Fig. 55 Modified Estlander flap harvesting

Figs. 58, 59, 60, 61, 62 and 63, and composite buccal mucosa resections with segmental mandibulectomy are shown in Figs. 43, 44, 45, 46, 47 and 48.

Fig. 56 Estlander flap creating the oral commissure

Fig. 57 Following the flap inset

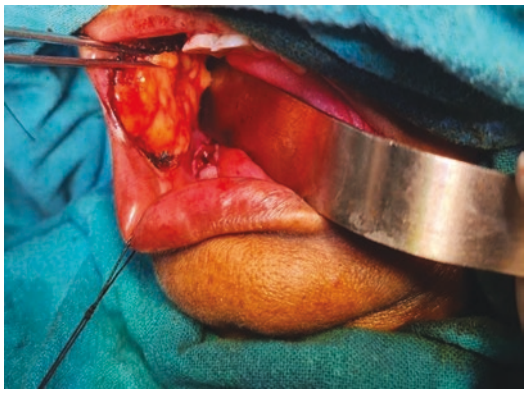

Fig. 58 Buccal fat pad can reconstruct small- to medium-sized buccal mucosa defects

The surgical steps of PMMC flap reconstruction of oral cavity defect are depicted in Figs. 64, 65, 66, 67 and 68.

Fig. 59 Buccal fat pad after inset. A bolster may be placed to secure the fat pad graft

Fig. 60 Buccal fat pad grafting for left buccal mucosa defect

Fig. 61 Buccal mucosa composite defect (following marginal mandibulectomy), reconstructed with the split skin grafting and floor of mouth advancement

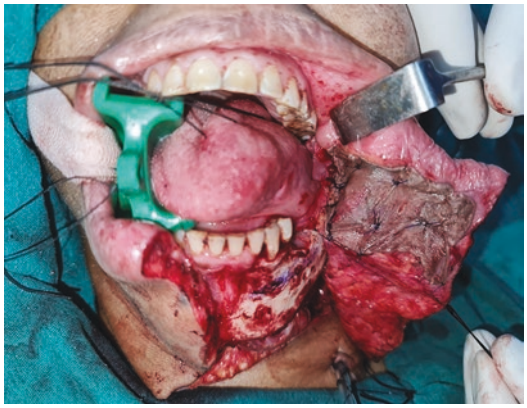

Fig. 62 Floor mouth advancement to close the marginal mandibulectomy defect

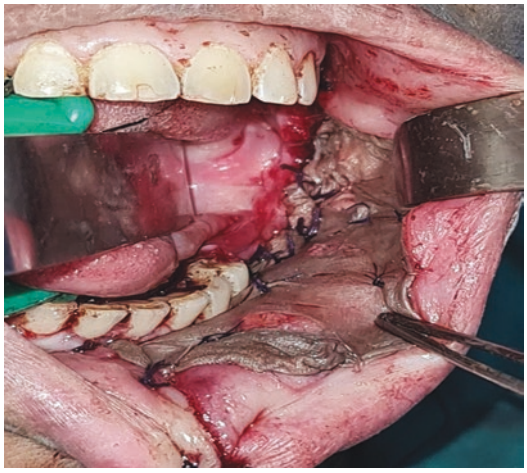

Fig. 63 Defect following complete closure

3.3 Bite Resection

- Also known as composite bialveolar resection, this entails excision of the superior and inferior alveoli with the intervening interalveolar tissue (like a bite).
- It is indicated for lesions involving the retromolar trigone extending to the superior alveolus.

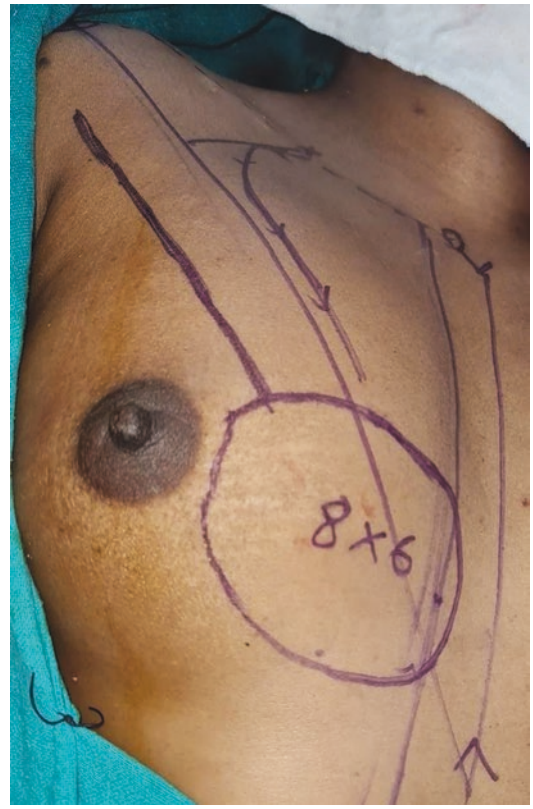

Fig. 64 Marking for the PMMC flap

- The specimen consists of the inferior as well as the superior alveolus in continuity with the overlying retromolar trigone mucosa and soft tissue formed by the pterygoid muscles.

3.4 Infratemporal Fossa

- If the lesion does not involve the ITF, then the bone cut is made anterior to the pterygoid plates. However, if the lesion involves the medial pterygoid muscle, the pterygoid plate is included in the specimen to ensure adequate soft tissue resection that includes the pterygoid muscles.

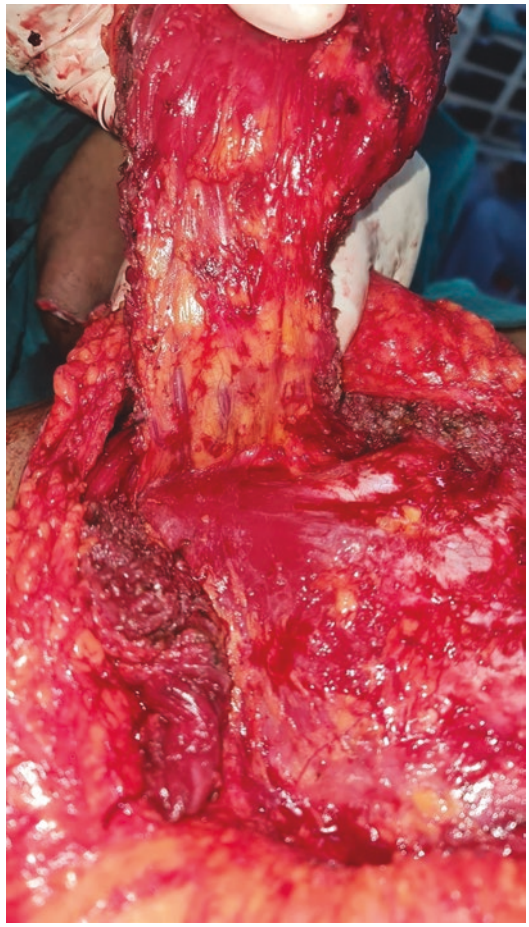

Fig. 65 PMMC flap pedicle

- "Bite excision" with resection of the entire medial pterygoid muscle is performed when the lower ITF is involved. If resection of the higher ITF is warranted, then "bite excision" encompassing the pterygoid plates is performed to include the entire medial and lateral pterygoid muscles.
- The temporalis muscle below the temporal fossa is resected in continuity with the coronoid up to the roof of the ITF.
- The ITF is divided into infra-notch and supra-notch at the level below and above the mandibular or sigmoid notch. The ITF is further sub-compartmentalised as described by Mahajan and colleagues [8].

Fig. 66 Reconstruction with the bipaddle PMMC flap

Fig. 67 Intraoral view of PMMC reconstruction

Follow-up of various reconstructions of oral cavity defects is shown in Figs. 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100 and 101, and the complications following the postoperative adjuvant radiotherapy are depicted in Figs. 102, 103, 104 and 105.

Fig. 68 PMMC donor site closure

Fig. 69 Bipaddle PMMC flap to close the defect

Fig. 70 Propeller nasolabial flap for the reconstruction of commissure defect

Fig. 71 Follow-up of nasolabial flap reconstruction for the marginal mandibulectomy defect

Fig. 72 Contour of the PMMC flap after reconstruction of posterior segmental mandibulectomy defect

Fig. 73 Tongue flap for the buccal mucosa defect

Fig. 74 Follow-up photo showing slight trismus with well-inset free radial forearm flap for the oral commissure and lower lip defect

Fig. 75 Upper lip and commissure defect reconstructed with the FRAFF

Fig. 76 One-year follow-up photo showing good epithelisation of FRAFF

Fig. 77 One-year follow-up of the floor of mouth advancement with buccal fat pad grafting for marginal mandibulectomy defect

Fig. 78 Bipaddle FRAFF for the buccal mucosa and cheek skin defect

Fig. 79 One-year follow-up of the floor mouth advancement with the skin grafting

Fig. 80 Six-month follow-up showing good mouth opening in FRAFF reconstruction

Fig. 81 Six-month follow-up of buccal fat pad grafting for buccal mucosa malignancy

Fig. 82 Deltopectoral flap reconstruction for the osteoradionecrosis debridement of the mandible

Fig. 83 Forehead flap reconstructions are usually not done commonly as the first line of management. This was done for patients with a second primary in the oral cavity and previously operated neck

Fig. 84 Tongue flap for marginal mandibulectomy defect

Fig. 85 Contractures may form within the oral cavity if a large buccal mucosa defect is not reconstructed with a flap

Fig. 86 Chest wall wound showing well-healed PMMC flap scar

Fig. 87 Intraoral view of PMMC flap

Fig. 88 Postoperative view of FRAFF: flap monitoring can be done by colour, skin turgor and scratching the flap with a 22G needle, checking the flap blood sugar and correlating it with the peripheral blood sugar

Fig. 89 Radial free forearm flap defect closed with the split skin grafting

Fig. 90 Excellent contour following mid-segmental mandibulectomy defect reconstruction with free fibula osteocutaneous flap

Fig. 91 Intraoral view of free fibula osteocutaneous flap

Fig. 92 Free fibula osteocutaneous flap for mid-segmental mandibulectomy

Fig. 93 Mid-segmental mandibulectomy with deltopectoral flap reconstruction after 1 year

Fig. 94 Follow-up of free fibula osteocutaneous flap for recurrent ameloblastoma resection with skin involvement

Fig. 95 Excellent contour following free fibula osteocutaneous flap

Fig. 96 Contour following the right segmental mandibulectomy reconstruction with free fibula osteocutaneous flap

Fig. 97 Intraoral view of free fibula osteocutaneous flap for the posterior segmental mandibulectomy

Fig. 98 Primary closure was done to close the buccoalveolar defect

3.5 Reconstruction Options

The aims are to restore the form and function. Whether to repair a defect depends on its size and depth, and whether there is a through-and-through defect into the neck:

- Buccal soft tissue defect only: Small defects can be closed primarily or left to granulate and heal by secondary intention like a tonsillectomy defect. Larger defects that include resection of the buccinator muscle that are left to granulate may lead to significant trismus due to scarring and contraction of the scar tissue.
- Marginal mandibulectomy: A marginal mandibulectomy defect can be strengthened with an onlay fibula or radial forearm osseocutane-

Fig. 99 A dental step prosthesis can be used to prevent the upper dentition impinging on the lower alveolar reconstruction

Fig. 100 The prosthesis prevents the upper dentition impinging on the FFOCF, especially while taking adjuvant radiotherapy

ous flap or a vascularised soft tissue flap may be used to close the exposed bone.

- Segmental mandibulectomy: If the resection crosses the midline and includes the mandibular arch, e.g. mid-third mandible segment, reconstruction with an osseocutaneous free flap is mandatory to avoid a debilitating Andy Gump deformity:
  - For lateral and posterior segmental or hemimandibulectomy defects, reconstruc-

Fig. 101 Reconstruction with the anterolateral thigh flap for composite defect

Fig. 102 Heister's mouth gag can be used to rehabilitate the patients with reduced mouth opening

Fig. 103 Plate can be exposed due to ORN: such exposed plates must be removed, and a vascularised flap must use to close the ORN wound

Fig. 104 Hyperpigmentation can occur in the field of radiation

Fig. 105 Mucositis can occur as a consequence of radiotherapy

tion with a pectoralis major myocutaneous flap is an acceptable alternative if osseocutaneous free flap reconstruction is not possible or is inadvisable, e.g. in the elderly, frail and unfit.

- Large soft tissue defect: A large soft tissue defect following, e.g. a "bite excision" or infratemporal fossa clearance can be reconstructed with pedicled flaps (forehead, temporalis, pectoralis major, deltopectoral) or free flaps (radial free forearm, anterolateral free thigh).
- Facial skin defect: The skin can be reconstructed by bipaddling a radial free forearm, anterolateral thigh or pectoralis major flap to provide both inner and external skin covers. Else a double flap, i.e. one flap to cover the inner mucosal and soft tissue defect and the other to cover the skin loss, can be used:
  - Commissure defects—primary closure of the lip commissure usually leads to trismus and reduced mouth opening:
    Estlander flap can be modified to feed the oral commissure defects.
    One must be extremely careful while dealing with the commissure defects following midline lip split incision. The intervening lip and skin may be devascularised due to loss of blood supply from the superior and inferior labial arteries.
    The bipaddled flap can be fed into the commissure by deepithelisation of splitting the flap. A small intervening skin may be left at the point of skin paddle fold to create the new commissure.
  - Whenever oncologically safe, the commissure must be left intact as it is the functional unit of the lip, helping in oral competence.
- Split skin graft: Split skin grafts can be employed for superficial defects. The graft is harvested from the thigh, and the mucosal defect is quilted with the skin graft with absorbable sutures. Pressure is applied to the graft by using a bolster, e.g. BIPP impregnated gauze, which is tied over the graft and kept in situ for 5 days. However, using a split skin graft for deeper and larger defects causes fibrosis that may cause trismus.
- Buccal fat pad graft: The buccal fat is conveniently located in the posterior part of the buccal space and can be gently delivered into the defect and used to fill the defect. It subsequently mucosalises. In some cases, it may cause a scar band and trismus.
- Pectoralis major flap: This was the workhorse of oral cavity reconstruction prior to the advent of free microvascular tissue transfer flaps. The pectoralis major flap has a robust blood supply and has a long pedicle length which makes it amenable to reconstruction of a defect as superior as the lower border of the

zygoma; the shape of the flap can be adjusted to the shape of the defect; and a large flap can be harvested. It remains a good choice when free flaps are not available, as a salvage procedure for a failed free flaps or when patients cannot tolerate a long procedure, e.g. the elderly and infirm.

- Nasolabial flap: The nasolabial flap has advantages that the donor site is adjacent to the defect and that the flap is thin and pliable and has a robust blood supply that permits shaping of the flap to precisely fill the defect. A larger defect, particularly when located anteriorly, can be covered with bilateral nasolabial flaps.
- Tongue flap: Advantages are that the flap is harvested adjacent to the defect and that there is insignificant donor site morbidity. Generally a posteriorly based flap is used.
- Floor of mouth advancement can be used to close the marginal mandibulectomy defects.
- Temporalis flap: This is favoured by some surgeons. It is based on the deep temporal artery which courses close to the coronoid process of the mandible which this has to be preserved.
- Forehead flap: It has a robust blood supply and is pliable, and the entire forehead can be utilised. It is however associated with significant cosmetic morbidity and is therefore generally reserved as a second-line reconstructive option.
- Deltopectoral flap: It is quick and easy to harvest, has consistent anatomy and is a pliable flap. For oral cavity repair, it generally requires a second-stage surgery after 3 weeks to disconnect the pedicle.
- Radial free forearm flap: It is a thin, pliable flap with a consistent blood supply and a long vascular pedicle. It is quick and easy to harvest. Donor site morbidity is worse than with anterolateral thigh free flaps. Its pliability makes it amenable to reconstruct a variety of oral defects. It can also be harvested as an osseocutaneous flap.
- Anterolateral free thigh flap: It can be harvested with the muscle and hence used for larger soft tissue defects. Donor site morbidity is insignificant.
- Radial osseocutaneous flap: This can be complicated by fractures of the remaining radius and restriction of forearm range of movement.
- Free fibula flap: Advantages include long bone length, a segmental blood supply making multiple osteotomies feasible and minimal donor site morbidity. Single vascular pedicle is most commonly used for maxillary and orbital defects.

3.6 Postoperative Care

- Airway: Elective tracheostomy is always indicated if mandibular resection crosses the midline with excision of both genial tubercles resulting in the tongue dropping back, extensive composite palatal resection or buccal mucosal resection requiring reconstruction with a bulky flap.
- Position: Maintain the patient at 15 degrees head high to reduce venous congestion and bleeding. Maintain a head position that avoids twisting of the vascular pedicle of a free flap.
- Feeding: Patients are fed by nasogastric feeding tube for at least 5 days, and until that patient is able to tolerate their own saliva.
- Antibiotics: A broad-spectrum antibiotic is administered for 24 h.

3.7 Rehabilitation

- Jaw stretching exercises: Trismus is one of the most common and most disabling sequelae of surgery for buccal cancer because of scarring and shortening of the muscles of mastication, as well as scarring in the tumour bed and of the flap or skin graft. Jaw stretching exercises should commence after initial wound healing, i.e. about 5–7 days after surgery, and should be continued for a long period of time.
- Bite guide prosthesis: Unopposed contraction of the opposite muscles of mastication results in deviation of the lower jaw following hemimandibulectomy. A bite guide prosthesis prevents this deviation and should be used for at least 6–8 weeks following surgery until complete healing has occurred.

4 Principles of Management of Palatal Tumours

Small lesions may be treated with transoral-wide local excision with resection performed to the level of the bony portion of the hard palate with anticipated reepithelialisation. The periosteum of the palatal bone acts as a barrier to spread; excision with bony preservation may be adequate for small lesions. Involvement of the periosteum requires removal of a portion of the bony palate, and partial or subtotal maxillectomy is required for larger lesions and for those that involve the maxillary antrum. For malignancies that extend along the greater palatine nerve, biopsy of this nerve is important for identifying neurotropic spread. Small- to medium-sized defects may be reconstructed with the aid of a temporalis, palatal island, facial artery musculomucosal or buccal fat pad flap. For lesions that result in a palatal defect after resection, prosthetic rehabilitation can offer the patient an excellent functional result. In this circumstance, a maxillofacial prosthodontist may construct a splint preoperatively for use at the time of resection. A split-thickness skin graft is used to cover edges of the bone from the osteotomy and surrounding soft tissue. The cavity is then packed with petroleum gauze, and a screw or wire fixation can be used to secure the prosthesis to the maxilla and to provide support to the underlying repair. The temporary prosthesis is removed approximately 1 week later, and the packing is evacuated; an interim prosthesis is worn until a more permanent option can be tailored to the resultant defect:

1. Approaches to the surgical management of tumours limited to the hard palate and alveolar ridge include:
   (a) Wide local excision—palatectomy—fenestration procedure.
   (b) Partial lateral maxillectomy: Surgical approach for tumours of the hard palate that involve the maxillary sinus and nasal cavity.
   (c) Inferior maxillectomy: Surgical approach for tumours of the hard palate that do not involve the floor of the maxillary sinus and nasal cavity.
2. Approach to benign mucosal tumours of the palate (biopsy-confirmed benign tumours only involving the mucosa (i.e. pleomorphic adenoma, etc.))
   (a) Wide excision 0.5-cm margins of the lesion.
   (b) The deep margin should be the periosteum of the hard palate.
   (c) If there is a concern for tumour involvement deep to the periosteum, the surface of the hard palate is drilled with a burr to remove the superficial layer of the bone.
3. Reconstruction—A palatal island rotational flap or rotational flap based on either the greater palatine or ascending palatine arteries is used to cover the site of the defect.

4.1 Technique of Infrastructure Maxillectomy

- The approach can be either intraoral or by upper cheek flap approach.
- Intraoral approach: Retract the lips with a hands-free cheek retractor, a bite block or a side-biting mouth gag to gain access to the oral cavity.
- Intraorally, mark the margins of the resection with a marking pen or electrocautery.
- Margins should be a minimum of 1 cm for any lesion suspicious of malignancy or biopsy-proven malignancy.
- The maxillary sinus is entered, and the inferior aspect of the maxillary sinus is thoroughly evaluated.
- A sagittal saw is used to make precise osteotomies:
  – The osteotomy is usually completed with the osteotome at the level of the pterygoid plates.
- The nasal cavity is entered, and the septum is transected with heavy curved Mayo scissors 1–2 cm superior to the floor of the nose.
- The soft palate is transected with electrocautery, and any residual soft tissue attachments are transected with scissors.
- After the mucosa of the maxillary antrum is removed as previously described, the inferior turbinate is also removed to prevent infection

and oedema, which would interfere with the application of a palatal prosthesis.
- More extensive tumours limited to the hard palate and alveolar ridge can be removed by simply extending the osteotomies.
- Once the specimen is removed, it is sent to the pathology laboratory for frozen section diagnosis and assurance of clear surgical margins.
- A split skin graft can be placed to cover the cheek, and an obturator is planned.

The surgical steps of infrastructure maxillectomy and its follow-up are depicted in Figs. 106, 107, 108, 109, 110, 111, 112 and 113.

Fig. 106 Marking for the intraoral infrastructure maxillectomy

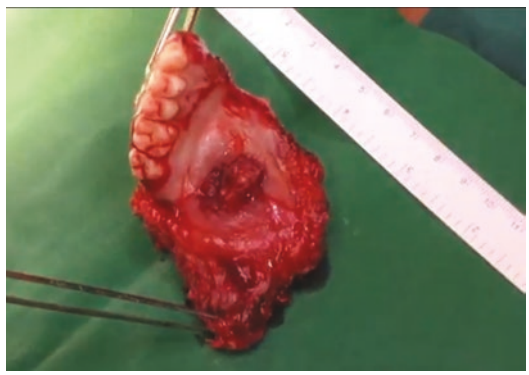

Fig. 107 Infrastructure maxillectomy defect

4.2 Rehabilitation

The four major means of rehabilitation include:

1. *Dental prosthetic management*: A patient with a limited lesion of the alveolar ridge alone or the alveolar ridge and palate that does not involve more than half the hard palate can be rehabilitated relatively easily. Preoperative dental impressions are made, and a surgical splint is fashioned and inserted at the time of resection as described earlier. Prostheses are designed according to the Aramany classification. The permanent prosthesis will have teeth

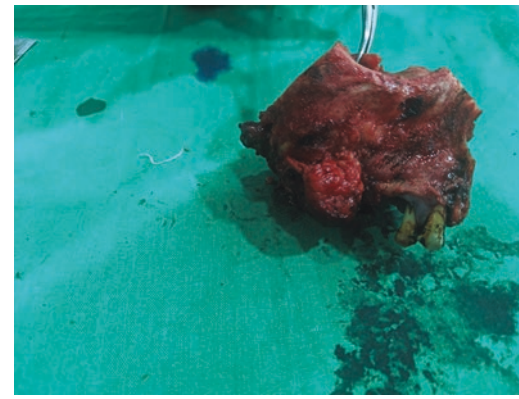

Fig. 108 Specimen of infrastructure maxillectomy

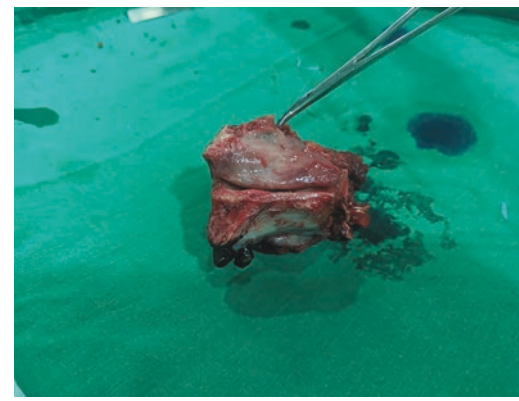

Fig. 109 Medial view of the infrastructure maxillectomy defect

Fig. 110 Palatal fenestration defect reconstructed with FRAFF

Fig. 111 Skin changes following radiotherapy

like a denture; it can be removed and cleaned by the patient. It is essential for the surgeon and prosthodontist to coordinate their efforts in restoring the patient's voice and ability for adequate oral intake. Patient with a removable denture that restores good oral-nasal separation and provides for a good cosmetic appearance.

2. *Local flaps*: The palatal island flap for reconstruction of palatal lesions is a single-stage mucoperiosteal flap that is a reliable source of regional vascularised soft tissue that obviates the need for prosthetic rehabilitation.
3. *Regional flaps*: The temporalis flap allows for immediate reconstruction with minimal morbidity for patients who have undergone total

Fig. 112 Intraoral infrastructure maxillectomy defect

inferior maxillectomy. The temporalis muscle is an attractive option for reconstruction. Microvascular free tissue transfer: Reconstruction of defects after inferior maxil-

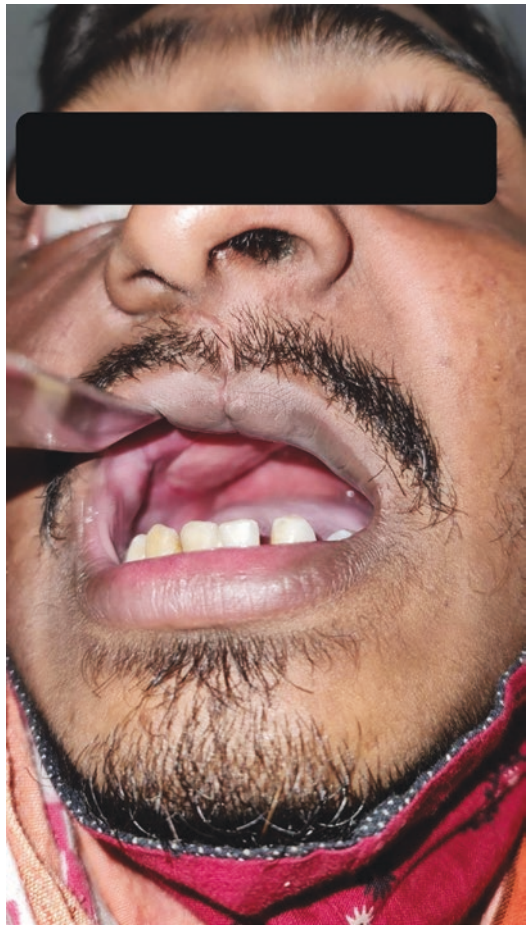

Fig. 113 Maxillectomy defect reconstructed with the temporalis muscle flap

lectomy is well summarised under the theme of functional palatomaxillary reconstruction. Larger defects (classes III to VI) require reconstruction with composite free flap reconstruction to close off the defect and restore natural facial contour.

4. *The free flap*: must include vascularised bone and soft tissue from the radial forearm, fibula, iliac crest or scapula. Free tissue transfer is superior to prostheses in larger defects with regard to improved speech and swallowing; however, placement of dental implants may be delayed or impossible based on the type of flap used.

5 Principles of Management of Lip Tumours

Small primary lesions may be treated with surgery or radiation with equal success and acceptable cosmetic results. However, surgical excision with histologic confirmation of tumour-free margins is the preferred modality:

- The reconstruction of lip defects after tumour excision requires innovative techniques to provide oral competence, maintenance of dynamic function and acceptable cosmesis.
- With small lesions—that is, defects of up to one-third of the lip's length—simple excision with primary closure is possible.
- When lesions require resection of up to one-third to two-thirds of the the lip's length, reconstructive options include a lip switch (Abbe-Estlander procedure).
- For tumours that require resection of more than two-thirds of the lip, the reconstructive options are the Gilles fan flap, nasolabial flap, bilateral advancement flaps, Karapandzic flap or free radial forearm flap with the palmaris longus tendon.
- The Karapandzic flap is a sensate neuromuscular flap that includes the remaining orbicularis oris muscle. The blood supply for this flap is provided by the corresponding branches of the labial artery.
- Microstomia is a potential complication with these methods of lip reconstruction.
- For large defects, Webster or Bernard procedures using lateral nasolabial flaps with buccal advancement have been described. In addition, for aggressive and advanced-stage lesions, an evaluation for perineural spread should be performed at the time of resection. With extensive PNI, a drill-out of the mental nerve or a mid-segmental mandibulectomy may be necessary.

Reconstruction of various lip defects is showing in Figs. 114, 115, 116, 117, 118, 119, 120, 121 and 122.

Fig. 114 Abbes flap for the upper lip defect, before pedicle division

Fig. 115 Abbes flap following pedicle division

Fig. 116 FRAFF for the lower lip and commissure defect

Fig. 117 Barrel FRAFF for the lower lip defect

Fig. 118 Verrucous carcinoma of the lower lip

Fig. 119 Submental flap reconstruction for the defect

Fig. 120 Six-month follow-up following the submental flap reconstruction

Fig. 121 Lower lip defect closed with islanded nasolabial flap

Fig. 122 Lower lip defect closed with interpolated nasolabial flap at a follow-up of 2 months

6 Principles of Management of the Neck

Management of neck nodes in patients with oral cancers has been a subject of debate for decades. This has in turn generated some good-quality evidence which forms the basis of current treatment guidelines. The two main controversies surrounding surgical management of cervical nodes have been the role of prophylactic neck dissections (PND) in node-negative early oral cancers and the extent of neck dissection for both node-positive (N+) and node-negative (N0) patients with oral cancers.

There have been numerous retrospective, prospective studies and randomised controlled trials (RCTs) addressing this issue. We published the results of our large retrospective cohort of 359 patients, where elective neck dissection (END) did not show a significant improvement in survival as compared to observation [24]. There have been five randomised controlled trials (RCTs) comparing PND to the wait-and-watch policy [25–29]. All trials prior to the trial published by D'Cruz et al. did not show a significant benefit of PND in early node-negative oral cancers. These trials however had very small numbers and heterogeneous methodology. The trial by D'Cruz and colleagues showed a significant improvement in overall survival and demonstrated that PND improved overall survival in these groups of patients by 12.5% (80% vs. 67.5%; $p = 0.01$). This effect of neck dissection was retained across all potential prognostic factors. This study also brought to light the pattern of relapses with patients in the wait-and-watch group. Patients who did not undergo PND presented with far more advanced nodal disease [25]. This could have been the probable cause of significantly poorer survival in patients who were not treated with a PND.

The most recent meta-analysis published by Ding et al. [30] has analysed six prospective studies (five RCTs, one prospective case-matched study), which included 865 patients. Results of this meta-analysis also showed that PND significantly improved the disease-free survival and reduced regional recurrences in the patients with early oral cancers.

In summary, the adequate evidence is in favour of performing a PND in patients with early oral cancers with a node-negative neck. Prophylactic neck dissection has a definite survival advantage in this group of patients avoiding relapses that are advanced and associated with poor prognostic factors. Majority of patients will be node-negative after a PND; however till robust predictive factors are available, prophylactic neck dissection should be considered the standard of care.

6.1 The Conundrum of Extent of Neck Dissection

For patients with an oral cavity malignancy who are clinically and radiographically N0, the procedure of choice is a selective neck dissection encompassing levels I–III [31]. Skip metastases to level IV are rare in oral cancers; however a SND (I–IV) is also an accepted procedure for the above group of patients.

In a N+ neck, there is a debate regarding the use of a more time tested comprehensive procedure of a MND (I–V) versus performing SNDs

avoiding dissection of level V. There is no head-to-head comparison in a prospective RCT looking at these two treatment approaches.

A meta-analysis published by Liang et al. in 2015 analysed five retrospective cohort studies, comparing SND with comprehensive neck dissection (CND) in clinically node-positive patients with oral squamous cell carcinoma. A total of 443 patients were included in this meta-analysis. They concluded that SND followed by adjuvant therapy had similar regional control and survival rates as compared to CND [32].

Andersen and colleagues [33] looked into the feasibility of SND in a node-positive neck. Treatment-naïve patients with mobile neck nodes without clinically apparent extranodal extension were included. Patients with intraoperative findings of tumour infiltrating to nonlymphatic structures were excluded. Postoperative radiotherapy was offered for patients with evidence of extracapsular spread (ECS) or multiple levels of nodal involvement, extensive primary disease or positive margins at the primary resection site.

They reported the results of 129 SNDs in 106 patients, and regional control rate was 94.3%. There were only six recurrences on the side of SND (5%). They concluded that patients could be treated with SND if they did not have massive adenopathy, nodal fixation, gross extracapsular spread, history of previous neck surgery or radiotherapy. As per the latest NCCN guidelines, the ECS would warrant adjuvant chemoradiotherapy [34], but Andersen's trial has provided only radiotherapy, which would be its stark limitation.

A current Cochrane analysis by Bessell and colleagues [35] found no evidence that RND increased overall survival compared with conservative neck dissection surgery. Rodrigo and colleagues [36], in a recent systematic review of current literature, supported the role of SND in HNSCC patients with cN1 and in cN2 patients.

Additionally, postoperative radiotherapy or chemoradiotherapy is routinely delivered in node-positive patients. Although these results are encouraging SND for node-positive patients should be offered with caution and to a subset of patients with N1/N2 nodal disease, no extracapsular involvement and without lower levels being involved. The final answer to the debate will come from a well-powered RCT comparing SND (I–IV) vs. MND with disease outcomes as an endpoint.

7 Technique of Selective Neck Dissection

Neck dissections are defined by the levels that are removed, and the surgical steps for each level are described individually here. The sequence of events for every neck dissection will be different, depending on surgeon preference, the location of palpable lymph nodes and the plan for excision of the primary cancer. General principles for all neck dissections are to define the superficial borders of the dissection and work from the superficial to deep structures to ensure adequate exposure of critical structures:

- Planning the incision—Depends on levels being removed and surgeon's preference. A transverse incision placed in a skin crease or standard utility incision affords good exposure for most SNDs.
- Incise the skin along the entire length of the planned incision. Identify the platysma prior to incising it, as the posterior border provides a reliable landmark for the external jugular vein and great auricular nerve.

- Incise platysma and elevate subplatysmal flaps. Keep the plane of dissection just deep to the platysma; this is an avascular plane that facilitates preservation of the external jugular vein, great auricular nerve and the superficial layer of the deep cervical fascia (e.g. the fascia overlying the SCM). In areas where the platysma is dehiscent, flap elevation is continued in a subcutaneous plane of the dissection that is best defined by the surgeon.
- The extent of flap elevation depends on the levels of the neck that will be removed.
- Expose the most superficial boundaries of the neck dissection according to levels removed:
  - Level Ia—Lateral border of the contralateral anterior belly digastric, mentum and hyoid.
  - Level Ib—Lower border of the mandible, anterior and posterior digastric, facial artery and vein at their junction with the lower border of the mandible.
  - Levels II–III—Posterior belly of digastric, lateral border of sternohyoid and SCM from behind the angle of the mandible to the posterior omohyoid muscle.
  - Levels II–IV—Posterior belly of the digastric, lateral border of sternohyoid, SCM from behind the angle of the mandible to the clavicle and the clavicle anterior to the insertion of SCM.
  - Level V—Mastoid tip, anterior border of trapezius, clavicle and posterior border SCM.
  - Levels of the neck are depicted in Fig. 123.
- Level I dissection:
  - Level Ia—Submental triangle: Dissect the fibrofatty tissue overlying the anterior bellies of the digastric muscles, from the mentum to the hyoid bone, including the lymphatic tissue between and deep to the anterior digastrics.
  - Complete dissection of level Ib includes the investing fascia of the submandibular gland and requires identification, dissection and protection of the marginal mandibular nerve. The course of the nerve in relation to the inferior border of the mandible is variable, and identification is facilitated by opening the investing fascia in the vicinity of the angle of the mandible, where it exits the tail of the parotid.
  - Once the nerve is skeletonised and placed above the inferior border of the mandible, the fascia of the gland is separated from the inferior border of the mandible, and the facial artery and vein are ligated.
  - Level I contents are retracted inferiorly and posteriorly while dissecting the lymphatic tissue from the anterior belly of the digastric and mylohyoid to expose the lateral border of the mylohyoid. After ligating the blood supply to the mylohyoid, an angled

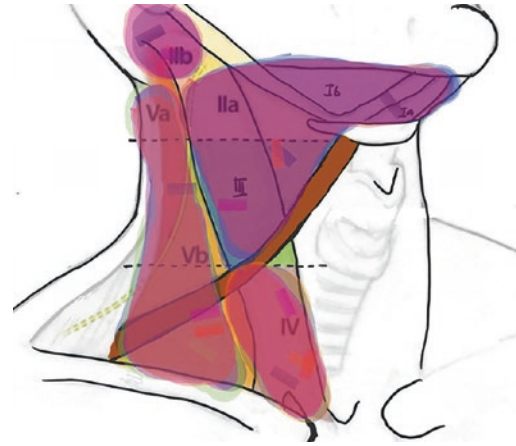

Fig. 123 Various levels of the neck

retractor is used to retract the lateral border anteriorly to expose the lingual and hypoglossal nerves, submandibular ganglion and duct and cross the hyoglossus muscle.
  - The ganglion and the duct are ligated and transected while visualising the lingual nerve and, once accomplished, will allow inferior retraction of the specimen and improved exposure of the hypoglossal nerve inferior and deep to the submandibular duct. Both the lingual and hypoglossal nerves are identified prior to ligating the submandibular duct as anteriorly as possible to ensure removal of the accessory gland tissue along the duct.
- Level II–III and II–IV dissection:
  - Elevate the investing fascia of the SCM along the entire anterior border preserving the great auricular nerve. Ligating the external jugular vein is not required but may be performed to improve exposure to level IIb. If the external jugular vein is ligated, consideration is given to reconstruction needs, and it should be ligated high along the anterior border of the SCM so sufficient length remains for anastomosis.
  - As the fascia is developed, retract the SCM posteriorly to clear the fascia off the deep surface of the muscle.
  - Identify CN XI as it enters the SCM. Avoid vigorous traction or trauma to the nerve. The transverse process of the atlas is a palpable landmark for the lateral border of the jugular in proximity of XI if anatomy obscures immediate visual identification.
  - Skeletonise the inferior border of the posterior belly of the digastric muscle, and retract superiorly to expose CN XI as it crosses the jugular vein.
  - Expose the lateral border of the jugular vein at the superior extent of the dissection, which will be defined by whether IIb is included in the dissection. For IIa, the superior extent of dissection is inferior to the intersection with CN XI.
  - If level IIb is included in the dissection, retract the posterior belly of the digastric/stylohyoid muscle complex superiorly and the SCM posteriorly with angled retractors. Then skeletonise CN XI from the lateral border of IJ to its entrance into the SCM. Expose the lateral border of the jugular vein, and gently retract XI inferiorly to fully expose level IIb.
  - Bovie cautery can be used to clear the fibroadipose tissue from the muscular floor in the superolateral corner of IIb but as the dissection nears CN XI and the jugular vein changes to a dissector and bipolar cautery, or LigaSure is used instead. Sweep IIb contents under CN XI, and continue with level IIa.
  - The posterior border of the SCM and cutaneous branches of the cervical plexus are the lateral extent of II–IV dissection. While retracting the SCM posteriorly, define the floor of the neck by incising the fibroadipose tissue deep to the SCM to expose the muscular floor, while the assistant provides countertraction of the specimen anteriorly using a surgical sponge. This will expose the deep cervical fascia overlying the scalene and levator scapulae muscles.
  - Division of the posterior omohyoid muscle facilitates complete exposure of level III and removal of the jugulo-omohyoid lymph node. This LN group should be removed in both level III and IV dissections.
  - The deep cervical fascia is left intact on the deep muscles of the neck. This protects the phrenic nerve and brachial plexus. The cervical rootlets are also preserved in SNP. The plane of dissection continues anteriorly in the plane superficial to the deep layer of the cervical fascia from the posterior border of the SCM to the carotid sheath. The transverse cervical artery and vein are superficial to the fascia and can be preserved.
  - Prior to dissection of the carotid sheath, identify the inferior most extent of the level III/IV dissection. To prevent inadvertent

torsion of the internal jugular vein (IJV) here, identify the lateral border of the IJV, and open the carotid sheath by using a dissector to expose the lateral border of the IJV. Then clamp and tie the lymphatic tissue lateral to this point for about 2 to 3 cm towards the SCM. Since this is the location of the thoracic duct and its tributaries, the use of Bovie cautery alone for this manoeuvre is avoided.
  - The superior and inferior borders of the carotid sheath dissection are clearly defined by exposing them; the neck contents are retracted anteriorly by the assistant to provide a broad plane of dissection along the entire length of the internal jugular vein. The carotid sheath is incised sharply, while the assistant provides anterior retraction, and the surgeon holds countertraction, both adjusting position as the internal jugular vein is exposed by the surgeon. Tributaries to the IJV are ligated as they are encountered. Traction and countertraction, and a sharp #10 blade, ensure complete clearance of the jugular chain lymph nodes of levels II, III and IV.
  - After dissection of the jugular vein, the contents are freed from the lateral border of the sternohyoid muscle. If the omohyoid muscle was divided, the superior belly is in the specimen and is separated from the hyoid completing removal of the specimen.
- Level V dissection:
  - Expose boundaries of dissection: posterior edge of SCM, clavicle and anterior border of the trapezius muscle.
  - The spinal accessory nerve is identified at the posterior border of the SCM and skeletonised as it traverses the posterior triangle to its point of entry into the trapezius muscle. CN XI is distinguished from other nerves travelling parallel deep to the anterior border of the trapezius muscle for 1 to 2 cm before inserting into the muscle. CN XI is protected as level V is dissected from the posterior scalene and levator scapulae muscle, leaving the fascia on the muscles.
  - Contents of level V are dissected off the splenius capitis, levator scapulae and scalene muscles, leaving a deep layer of the deep cervical fascia intact to protect the brachial plexus.
  - If included with levels II–IV, the contents of level V are brought deep to the SCM and removed with the jugular chain lymphatics.

Various steps of neck dissection are shown in Figs. 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137 and 138.

Fig. 124 Upon skin flap elevation, the marginal mandibular nerve is located by dissecting lateral to the facial artery

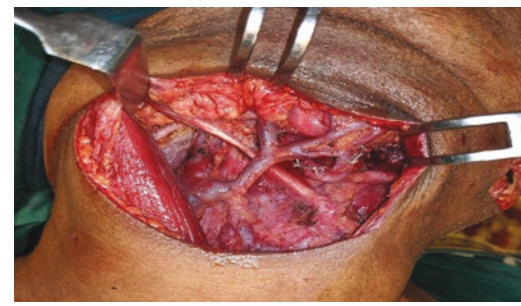

Fig. 125 Facial vein may have a varied course in the neck, but it is posterior to the facial artery

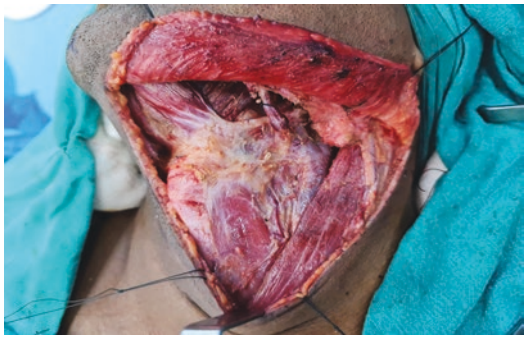

Fig. 126 Facial vessels may be preserved during level IB dissection when the nasolabial flap is planned

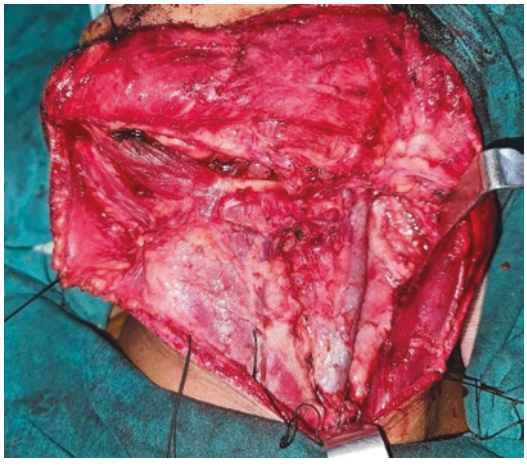

Fig. 127 Following selective neck dissection (I–III)

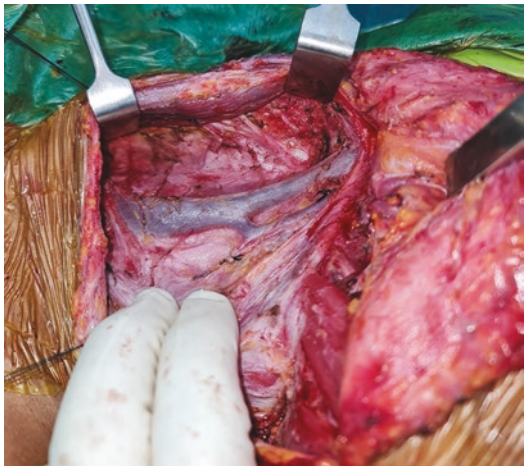

Fig. 128 The spinal rootlets in the neck mark the posterior limit of the selective neck dissection

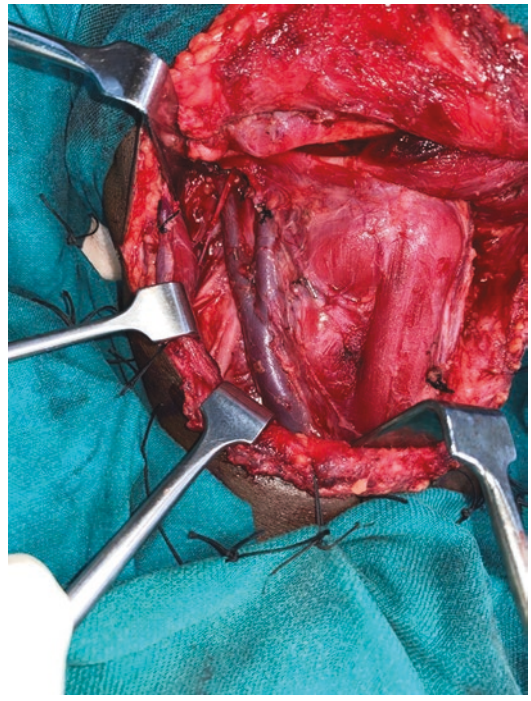

Fig. 129 Spinal accessory nerve and parajugular gutters can be well appreciated

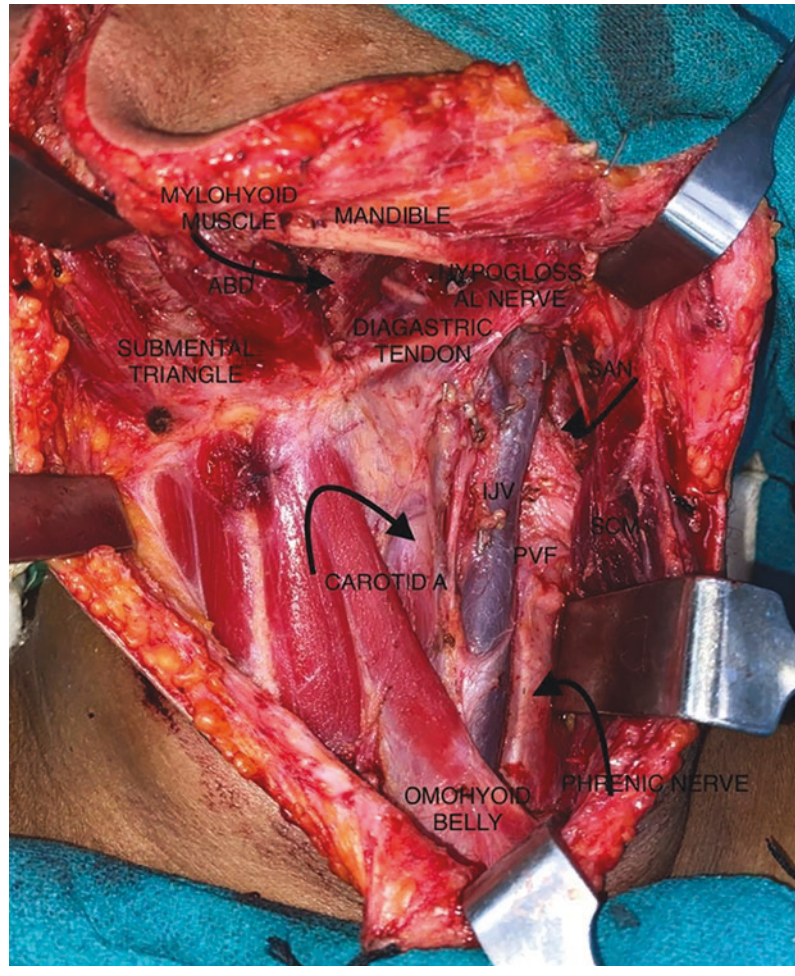

Fig. 130 Surgical bed after selective neck dissection I–III

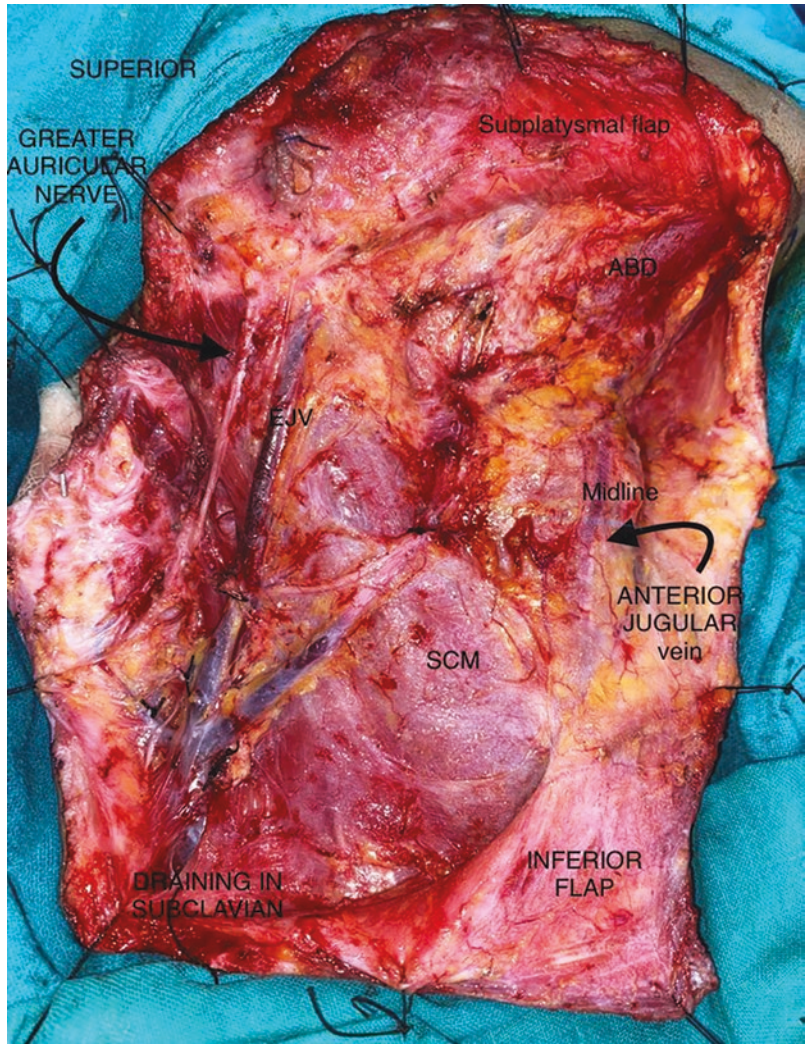

Fig. 131 Upper and lower skin flap elevation for modified radical neck dissection

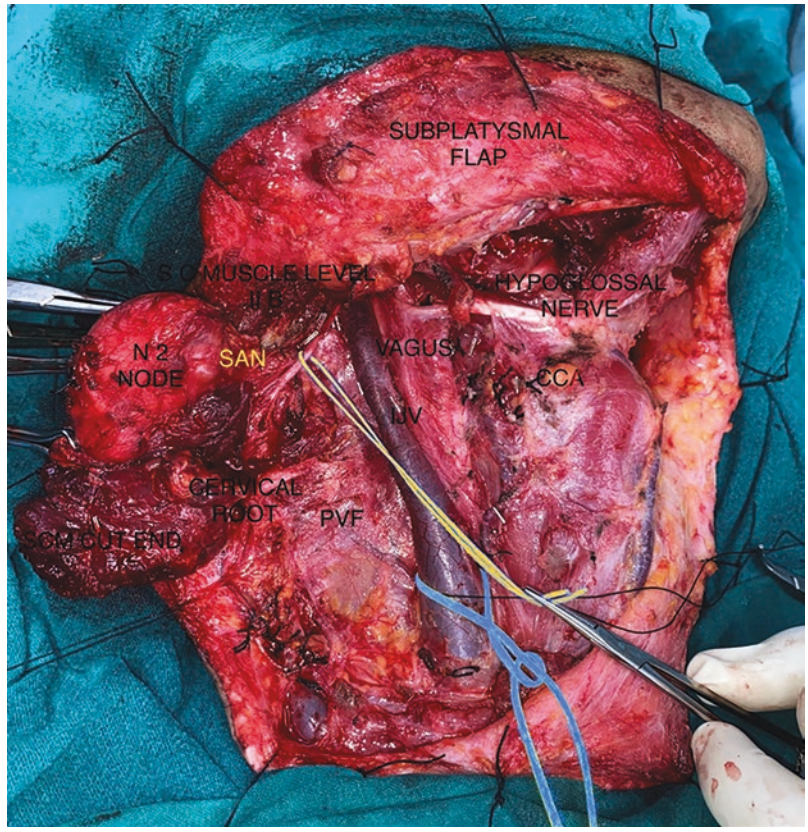

Fig. 132 Modified radical neck dissection

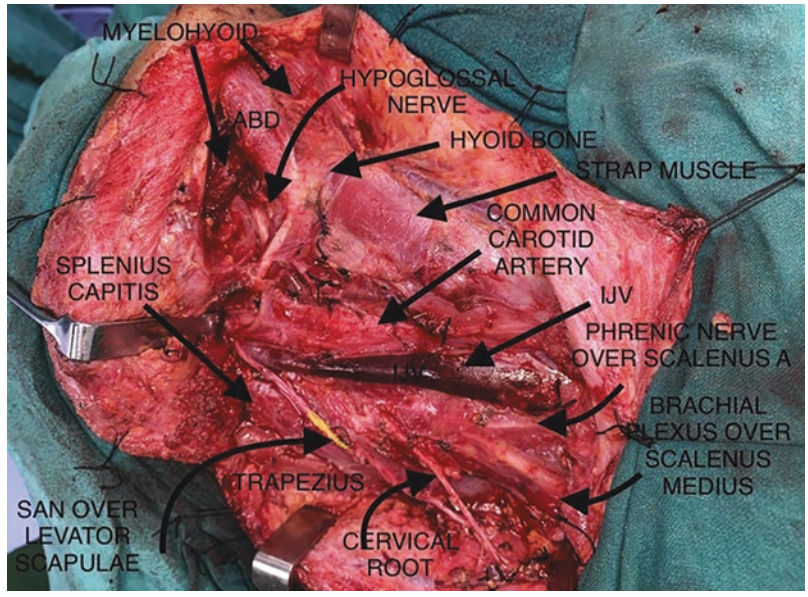

Fig. 133 Modified radical neck dissection showing all structures

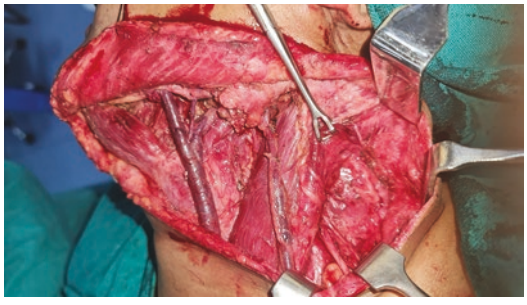

Fig. 134 Spinal accessory nerve in level V can be located 1 cm superior to Erb's point in the neck

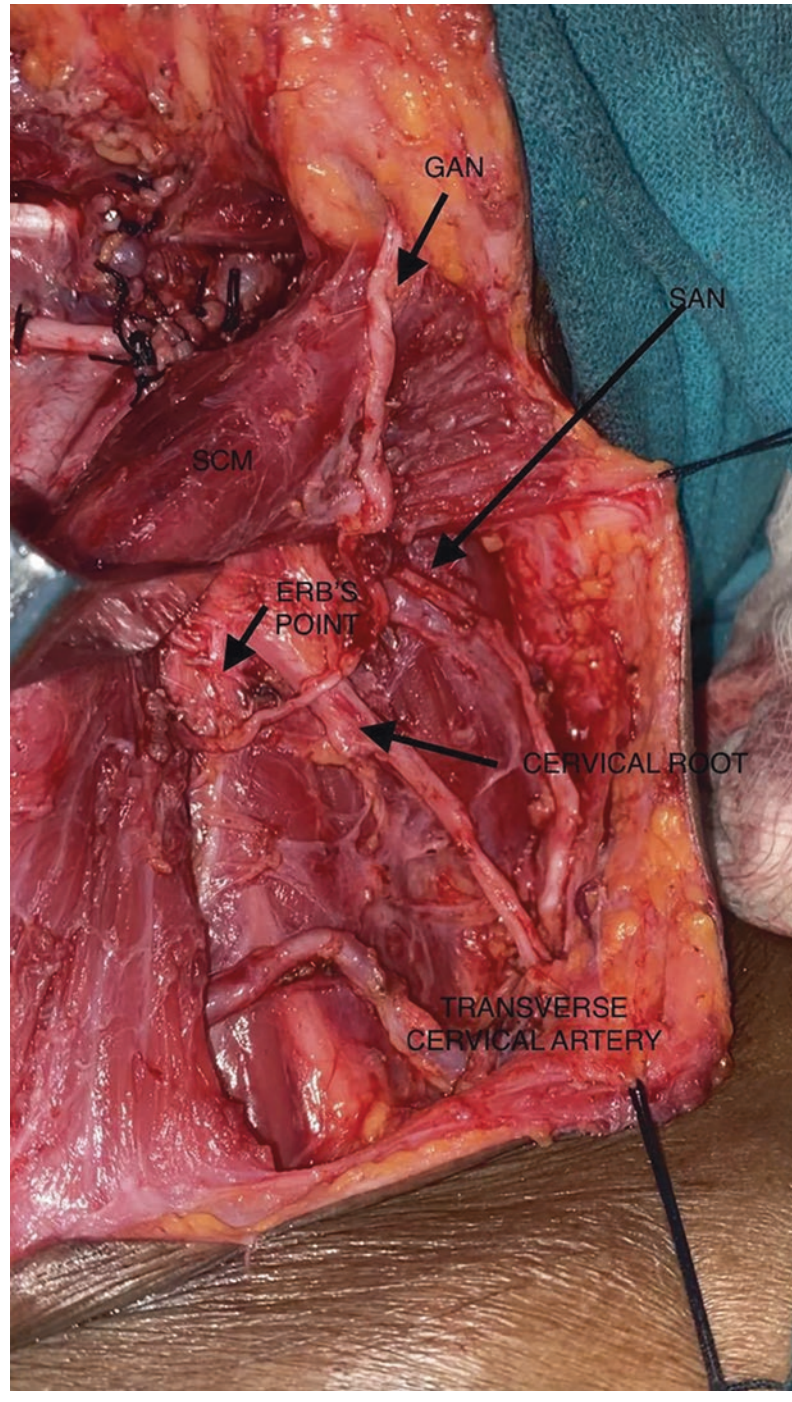

Fig. 135 Erb's point in the neck to localise the spinal accessory nerve

Fig. 136 Well-healed scar of modified Schobinger's incision

Fig. 137 Well-healed scar of apron incision with lateral extension

Fig. 138 Contracture band can occur if vertical drop is not curved

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Nose and Paranasal Sinuses

Nitin M Nagarkar, Ambesh Singh,
and Rupa Mehta

1 Introduction

Nose and paranasal malignancies refer to a wide variety of cancers seen in the nose and adjacent paranasal regions. But less than 1% of the malignant tumours identified in the head and neck area are neoplasms. These tumours typically have mild first signs, making early detection challenging. Because of this, the time between the onset of symptoms and the final diagnosis is often 6 months. Furthermore, these tumours have a variety of histological presentations, making diagnosis difficult. Nasal and paranasal malignancies are grouped by the World Health Organization (WHO) into 44 unique histological categories, which are roughly classified into epithelial and non-epithelial groups [1], as given in Table 1.

Information on surgery and histological distribution of benign sinonasal tract tumours from 1298 patients who had treatment at the university hospitals in Brescia and Varese [2] is described in Table 2.

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Table 1 Paranasal sinus malignancy histologic classification by the World Health Organization [1]

Epithelial malignancies
Squamous cell carcinoma
• Verrucous carcinoma
• Basaloid squamous cell carcinoma
• Papillary squamous cell carcinoma
• Spindle cell carcinoma
• Adenosquamous carcinoma
• Acantholytic squamous cell carcinoma
Lymphoepithelial carcinoma
Sinonasal undifferentiated carcinoma
Salivary gland-type carcinomas
• Adenoid cystic carcinoma
• Acinic cell carcinoma
• Mucoepidermoid carcinoma
• Clear cell carcinoma not otherwise specified
• Epithelial-myoepithelial carcinoma
• Myoepithelial carcinoma
• Carcinoma ex pleomorphic adenoma
• Polymorphous low-grade adenocarcinoma
Adenocarcinoma
• Intestinal-type adenocarcinoma
• Non-intestinal-type adenocarcinoma
Neuroendocrine tumours
• Typical carcinoid
• Atypical carcinoid
Small cell carcinoma, neuroendocrine type
Soft tissue malignancies
Fibrosarcoma
Malignant fibrous histiocytoma
Rhabdomyosarcoma
Leiomyosarcoma
Angiosarcoma
Malignant peripheral nerve sheath tumour
Bone and cartilage malignancies

(continued)

N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_8

Table 1 (continued)

Chordoma
Chondrosarcoma
Mesenchymal chondrosarcoma
Osteosarcoma
Hematolymphoid malignancies
Langerhans cell histiocytosis
Extranodal natural killer/T-cell lymphoma
Diffuse large B-cell lymphoma
Extramedullary plasmacytoma
Extramedullary myeloid sarcoma
Histiocytic sarcoma
Neuroectodermal malignancies
Ewing sarcoma
Primitive neuroectodermal tumour
Olfactory neuroblastoma
Melanotic neuroectodermal tumour of infancy
Mucosal malignant melanoma
Germ cell malignancies
Teratoma with malignant transformation
Sinonasal teratocarcinosarcoma

Accurately staging nose and sinus tumours is difficult despite the advancements in endoscopy and the widespread availability of modern imaging methods such as MRI and CT scans. Surgery and radiation are the two main treatments; however, these treatments can cause significant health issues including facial deformation, difficulty in chewing and in some cases, blindness. Sinonasal malignancies have a worse prognosis compared to other head and neck region malignancies, making quality of life considerations an important factor when assessing treatment options.

Table 2 Benign tumours of the sinonasal tract by histology and surgical approach [2]

	Surgical approach			
Tumour histology	Endoscopic	Combined	External	Total
Inverted papilloma	592	75	12	679
Osteoma	115	54	13	182
Juvenile angiofibroma	147	1	6	154
Lobular capillary hemangioma	68	0	0	68
Fibrous dysplasia	58	3	1	62
Schwannoma	16	3	0	19
Cavernous hemangioma	16	1	0	17
Ossifying fibroma	11	0	0	11
Hamartoma	46	0	0	46
Glioma	8	3	1	12
Pleomorphic adenoma	7	0	0	7
Miscellaneous	33	8	0	41
Total	1117	148	33	1298

2 Surgical Anatomy of the Nose and Paranasal Sinuses

The paranasal sinuses within the nose have extensive and complex architecture. It is essential to have an understanding of the intricate surgical anatomy due to the close proximity of these structures to crucial components such as the orbit and brain. Here is a brief overview.

2.1 Ethmoidal Sinus

- The ethmoid bone is divided into two parts in the midline that looks like a labyrinth. The basal lamina of the middle turbinate further divides this into anterior and posterior cell groups.
- The ethmoidal sinus is made up of a fragile skull base, and lamina papyracea forms its superior and lateral limits.
- The emissary vein, which is anterior to crista galli, and the olfactory fibres in the cribriform region form the path of spread to the anterior cerebral fossa.
- The distance in millimetres from the anterior ethmoidal foramen to the anterior lacrimal crest (24 mm), from the posterior ethmoidal foramen to the anterior ethmoidal foramen (12 mm) and from the optic nerve to the posterior ethmoidal foramen (6 mm) is an established formula for identifying the anterior ethmoidal artery, posterior ethmoidal artery and the optic nerve.
- The cribriform niche can range from 15.5 to 25.8 millimetres in length and from 0 to 15.5 millimetres in depth.
- During a craniofacial resection, the dura should be carefully removed since it is closely applied to the cribriform region.

2.2 Sphenoid Sinus

- The internal carotid artery and optic nerve are clearly visible on the lateral wall of the sphenoid sinus.
- In 20% of cases, the lateral wall has a varying depth, with an opticocarotid recess that is dehiscent.
- Relation:
  - Cavernous sinus, pterygoid canal and foramen rotundum that press into the sinus cavity laterally.
  - The pituitary gland is situated above.
  - The cells of the posterior ethmoidal sinus that extend lateral and superior to the sphenoid sinus are known as the poster superior onodi cell. It is more vulnerable to injury during instrumentation when it is present because the carotid artery and optic nerve are frequently accessible in its lateral wall.

2.3 Frontal Sinus

- Varying in size and form.
- The front ethmoidal cell influences the drainage to the middle meatus, resulting in an hourglass-shaped structure rather than a duct.

2.4 Maxillary Sinus

- The inferior turbinate, the lacrimal bone, the bulla of the ethmoidal sinus, the uncinate process, the perpendicular plate of the palatine bone and the nasal cavity itself form its medial wall.
- The maxillary sinus is related to the cheek anteriorly.
- Moreover, premolar and molar roots connect them inferiorly to the mouth cavity.
- Furthermore, the pterygoid plates, part of the sphenoid bone, form its posterior relation.
- Additionally, the pterygomaxillary fissure, which contains the terminal portion of the maxillary artery, is related posteriorly to the sinus.
- Inside the pterygopalatine fossa is the maxillary nerve, along with the pterygopalatine ganglion in its neural compartment, as well as the maxillary artery's terminal branch and its branches in its vascular compartment.

- Lastly, the infratemporal fossa is located between the pharynx's wall and the mandible's ascending ramus and contains the branches of the mandibular nerve, the lateral pterygoid muscles, the pterygoid venous plexus and the maxillary artery.

2.5 Orbit

- The medial wall comprises a thin bone called lamina papyracea, while the inferior is thin and linked to the maxillary sinus.
- An inferior orbital fissure links the pterygopalatine fossa medially and the infratemporal fossa laterally.
- The superior orbital fissure connects to the cavernous sinus.

2.6 Nasal Septum

- Composed of the ethmoid's perpendicular plate, vomer and quadrilateral cartilage.

2.7 Lymphatic Drainage

- Moderately poor, it drains from the nose and paranasal sinuses to the jugulodigastric and retropharyngeal nodes.
- From the vestibule into the cervical group of lymph nodes through the anterior septum and columella.

3 Pattern of Tumour Spread

The distribution of the nose and paranasal sinus involvement by malignancy is as follows:

- The maxillary sinus is the most common site of nose and paranasal malignancy, accounting for 50–70% of the cases.
- The nasal cavity is the second most common site, accounting for 15–30%.
- The ethmoidal sinus is the third most common, accounting for 10–20%.
- Frontal sinus and sphenoidal sinus are the least common sites, accounting for less than 1%.

3.1 Local Spread

- As the size of the malignant lesion increases, it consumes the sinus in which it is located and erodes any surrounding bone.
- The dura, periosteum and perichondrium act as a barrier to limit the spread of the tumour, but further growth may involve these structures.
- If the dura, periosteum and perichondrium are breached, the tumour may spread to the intracranial and intra-orbital areas.

3.2 Regional Spread

- At presentation, approximately 10% of patients have metastasized lymph nodes.
- In cases where the lesion is centrally situated, both bilateral groups of lymph nodes may be impacted.

3.3 Distant Metastasis

- Adenocarcinoma has a higher chance of distant metastasis compared to squamous cell carcinoma, at 20% and 10%, respectively.
- Common sites for distant metastasis are the bone, brain, liver, lung and skin.

4 Clinical Evaluation

4.1 Endoscopy

It is essential to have a comprehensive endoscopic examination of the nasal cavity if someone is thought to have a malignant tumour in the nose or paranasal sinuses. These tumours may be visible due to their ulcerated form or may be difficult to identify in endoscopy. The tissue around the tumour may also become swollen, resulting

in a polypoid response. However, a biopsy in an outpatient clinic may not yield any information. Therefore, it is recommended to consult with a specialized medical professional and receive additional testing, if needed, to accurately determine the presence of a tumour.

4.2 Imaging

The imaging modality accurately identifies the characteristics of the disease, such as if there is bony erosion, involvement of the skull base, intracranial involvement, perineural invasion and orbital extension, in order to stage the extent of the disease.

4.3 Computed Tomography (CT)

- Bone erosion can be accurately determined by CT scan.
- CT is inadequate for accurately determining the disease's stage.
- It can be hard to tell apart from post-obstructive change, periorbital involvement and dural involvement using a CT scan.
- To gain a better understanding of the anatomy and extent of the disease in the nose and paranasal sinuses, axial and coronal images are necessary.

4.4 Magnetic Resonance Imaging (MRI)

- Precontrast T1-weighted MRI imaging can provide information on the extent of disease involvement in the soft tissue. It is important to assess any periorbital, infratemporal and cerebral space involvement.
- Postcontrast T1-weighted MRI with fat saturation may be useful to darken lipid-rich regions such as the periorbita and can show any perineural invasion of the geniculate ganglion and skull base foramina.
- T2-weighted MRI imaging can be used to re-evaluate the tumour size after CT scans and identify secretions and oedema that may have been overlooked.
- Additional sequences such as FLAIR or diffusion-weighted imaging may be necessary depending on the tumour.

4.5 Positron Emission Tomography (PET)

- Positron emission tomography (PET)/CT enables accurate localization of malignant tumours by combining functional and structural imaging.
- The procedure relies on the uptake of 18-fluorodeoxyglucose (18-FDG) by cancer cells, which have higher levels of glucose transporters and hexokinase than normal cells.
- However, PET/CT is limited in its ability to interpret results due to inflammation in the sinonasal area and is used for pre-treatment staging and post-treatment monitoring for metastases.

4.6 Biopsy

A tissue diagnosis is essential when examining tumours. If a biopsy is performed in an outpatient clinic, it is important to have the necessary resources on hand to address any potential bleeding. Under general anaesthesia, biopsies increase the likelihood of a successful diagnosis and allow for the collection of samples directly from the sinus. If the Caldwell-Luc method has the potential to spread the tumour or complicate a future resection, it should not be used.

5 Surgical Approach

5.1 Endoscopic Approach

- The endoscopic approach was initially introduced for inflammatory conditions in the 1980s, and the technique gradually evolved for skull base and tumour surgery.

- Drills, debriders and bipolar diathermy are used during dissection in addition to the usual endoscopic sinus tools.
- The endoscopic method is of great use when the disease is limited to the nose and paranasal sinus.
- Endoscopic methods are contraindicated when the orbital contents, frontal sinus, dura lateral to the ethmoidal roof or nasal bones are involved; however, these techniques can occasionally be paired with transcranial approaches or orbital exenteration.

5.2 Open Approach

To facilitate the various bony resections, it is necessary to use the appropriate soft tissue access procedure, of which three are commonly described:

- Lateral rhinotomy.
- Weber-Fergusson.
- Midfacial degloving.

5.3 Combined Approach

It involves both the endoscopic and the open approach to facilitate the excision of the locally advanced extensive lesion of the nose and paranasal sinuses.

6 Angiofibroma

Angiofibroma is a highly vascular, histologically benign lesion that can induce severe epistaxis in juvenile nasopharyngeal angiofibroma [3]. It is most common in adolescent male patients [4]. The lateral nasopharynx is where the lesions develop, and they are hormonally sensitive [4]. The internal maxillary artery (IMA) is the source of their blood supply. Despite being a benign tumour, juvenile nasopharyngeal angiofibroma can seriously harm nearby tissues by local invasion.

An examination of 120 cases from the Mayo Clinic characterized the clinical characteristics of juvenile nasopharyngeal angiofibroma [5]. The typical patient was 15 years old (range 7–29 years). The most typical symptoms included nasal obstruction, epistaxis and nasal discharge; OME and reduced hearing can be present [4–6]. Although the tumours may protrude into the nasal cavity, they are frequently only detectable through a nasal cavity and nasopharyngeal examination by an endoscope [5].

CT or MRI with contrast is typically used to confirm the diagnosis and reveals an enhancing mass with involvement and enlargement of the pterygomaxillary fissure [5, 7].

Depending on the extent of the condition, many categories are used to stage it, some of which are shown in Tables 3 and 4.

Different modalities of imaging are shown in Figs. 1, 2, 3, 4 and 5.

Intranasal biopsy of these lesions should be avoided because of the risk of life-threatening bleeding [8].

6.1 Case 1

A 19-year-old male patient presented with a 4-month history of self-limiting nasal bleeding. Contrast-enhanced CT and MRI scan shows an enhancing lesion in the right nasal cavity with

Table 3 Andrew's classification (modified Fisch) of JNAs

Stage	Description
I	Tumour limited to the nasal cavity and nasopharynx
II	Tumour extension into the pterygopalatine fossa, maxillary, sphenoid or ethmoid sinuses
IIIa	Extension into the orbit or infratemporal fossa without intracranial extension
IIIb	Stage IIIa with small extradural intracranial (parasellar) involvement
IVa	Large extradural intracranial or intradural extension
IVb	Extension into the cavernous sinus, pituitary or optic chiasm

Table 4 Radkowski's classification (modified session's classification) of JNAs

Stage	Description
Ia	Limited to the nose and nasopharynx area
Ib	Extension into one to more sinuses
IIa	Minimal extension into the pterygopalatine fossa
IIb	Occupation of the pterygopalatine fossa with or without orbital erosion
IIc	Infratemporal fossa extension with or without cheek or pterygoid plate involvement
IIIa	Erosion of the skull base (middle cranial fossa or pterygoids)
IIIb	Erosion of the skull vase with intracranial extension with or without cavernous sinus involvement

Fig. 1 CT scan in the coronal plane shows the extension of the lesion into the nasal cavity and the maxillary sinus

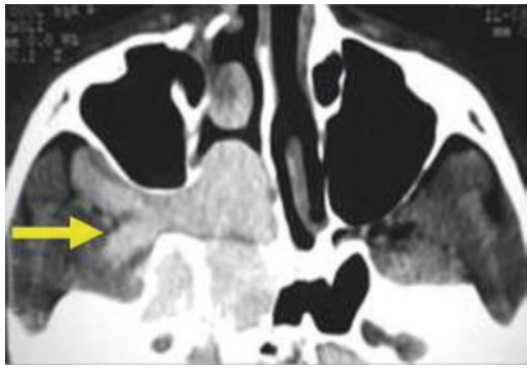

Fig. 2 Lesion originating from the sphenopalatine foramen and involving the nasopharynx, PPF and ITF/stage IIc

limited extension to the pterygopalatine fossa. Patient was planned for excision by endoscopic approach.

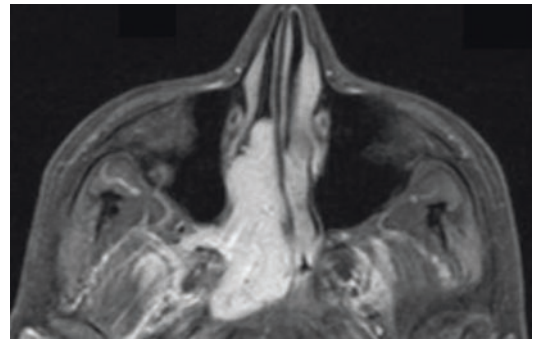

Fig. 3 MRI shows the lesion involving the nose and extending into the pterygopalatine fossa/stage II

6.1.1 Operative Technique

- Cottonoids soaked in lignocaine and mixed with an adrenaline solution should be used to sufficiently clear the nasal cavity due to congestion.
- The inferior turbinate, middle turbinate and axilla of the middle turbinate should all be identified before the zero-degree Hopkins endoscope is inserted into the nasal cavity.
- Good exposure without injuring the surface of the tumour.
- Middle turbinectomy, ethmoidectomy, broad antrostomy or sphenoidotomy are procedures that can be used to accomplish exposure.
- Establishing the proper dissecting plane between the angiofibroma and the surrounding tissue.
- Removing the posterior maxillary wall to reach the infratemporal and pterygopalatine fossae.

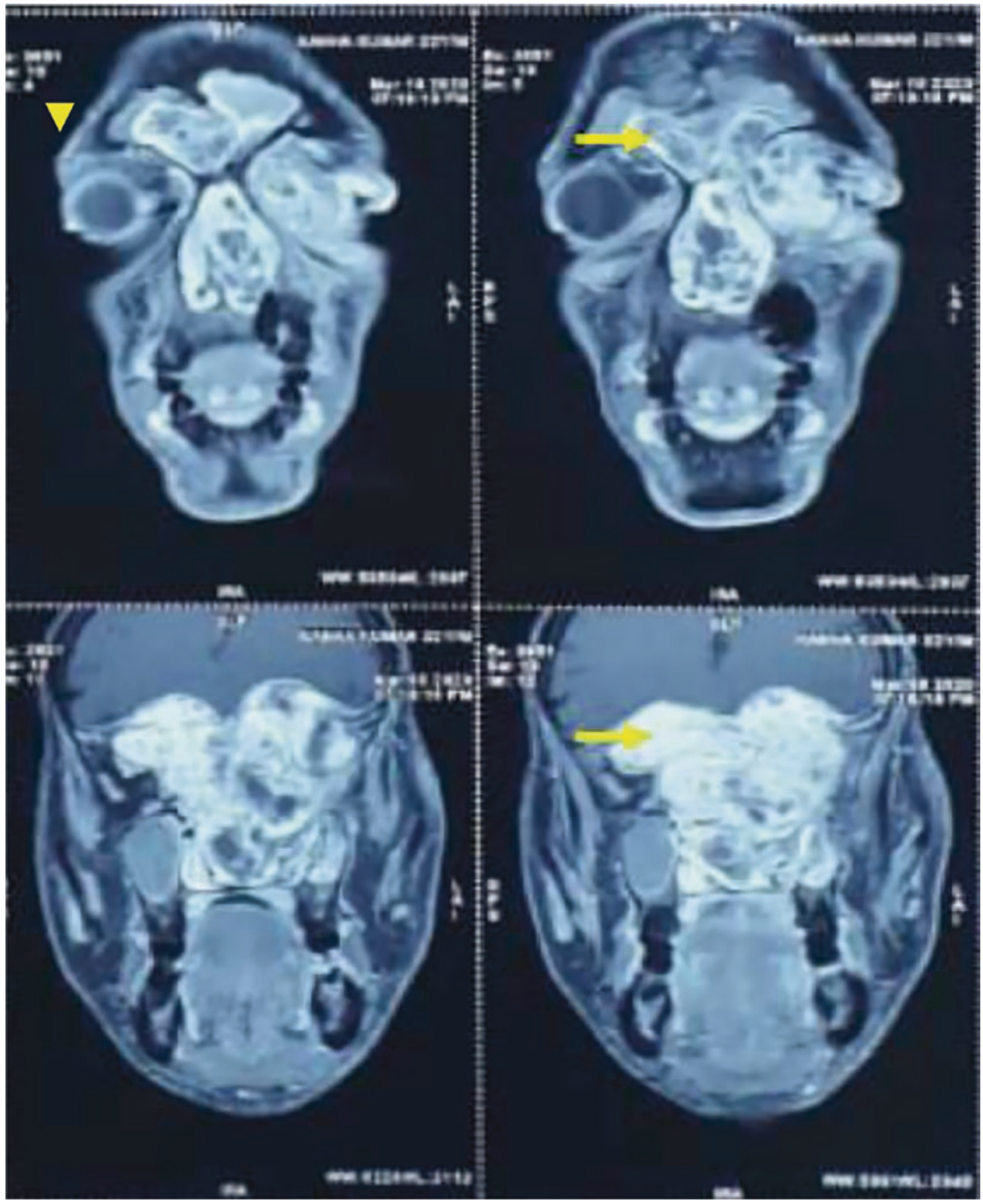

Fig. 4 MRI scan: a coronal plane shows the lesion with intracranial extension

- If a posterior septectomy is necessary to provide for four-hand method exposure to the nasopharynx.
- Identifying and managing the maxillary artery.
- Haemostasis can be achieved by:
  - Irrigation by warm saline.
  - Packing with cottonoids.
  - Using haemostatic material like fibrillar and Surgicel.
- Drilling of the pterygoid root—To prevent recurrence.

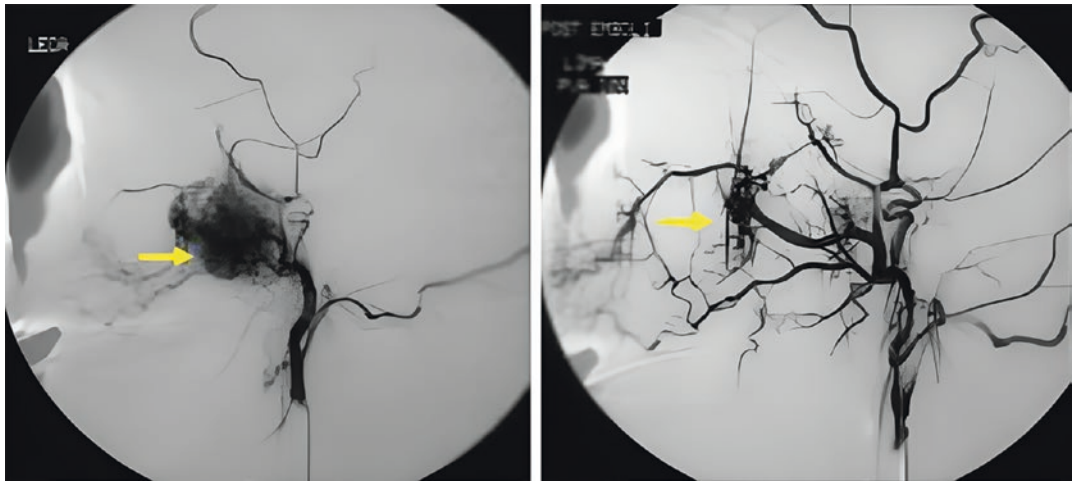

Fig. 5 DSA showing tumour blush, feeder branch from IMA (a), and tumour disappears following embolization (b)

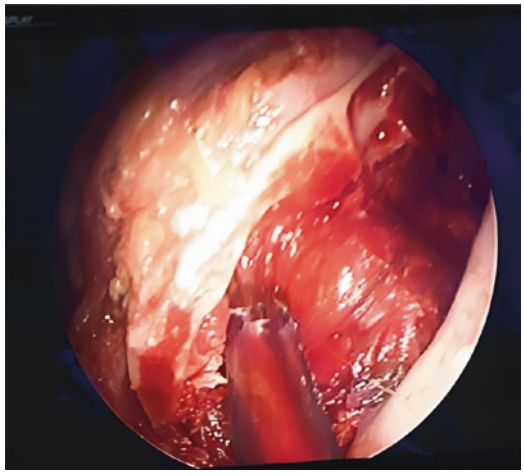

Fig. 6 Creating a plane between the lesion and the surrounding normal mucosa of the nasal cavity by blunt dissection

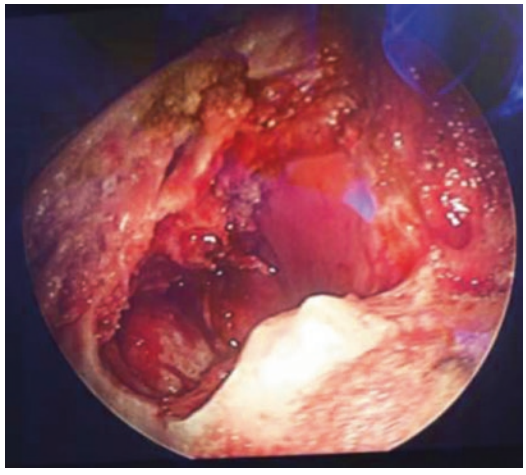

Fig. 7 Endoscopic view of the nasal cavity after the excision of the lesion

Fig. 8 Excised specimen of angiofibroma

- Few steps of endoscopic surgery are depicted in Figs. 6, 7 and 8.

6.2 Case 2

A 17-year-old male presented with intermittent nasal bleeding for 3 months and a mass protruding from the right nostril for 1 month. Contrast-enhanced CT and MRI scan shows an enhancing lesion in the right nasal cavity with extension to the pterygopalatine fossa and no intracranial or intra-orbital extension. The patient was planned for excision by open approach by Weber-Ferguson incision.

6.2.1 Operative Technique: Caveats

- The maxilla is better exposed by the Weber-Ferguson incision.
- To ensure cosmesis, the transverse limb should be positioned near the lid edge, often in the first crease.
- To reduce postoperative oedema, any lateral extension into a crow's foot should travel in an inferolateral orientation.
- It is beneficial to bend the incision forward across the nasal bones in the medial canthal area, where there is the highest risk of skin loss as a result of radiation, for further support.
- The mucosal incision runs anteriorly across the alveolus and turns laterally at the intersection of the hard and soft palates, passing behind the maxillary tuberosity.
- The incisions in the soft tissue are all gradually dissected free when the face skin flap is elevated on a submuscular plane.
- Haemostasis is achieved.
- The nasal and maxillary cavity is packed with ribbon gauze.

Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 9, 10, 11, 12, 13, 14, 15, 16 and 17.

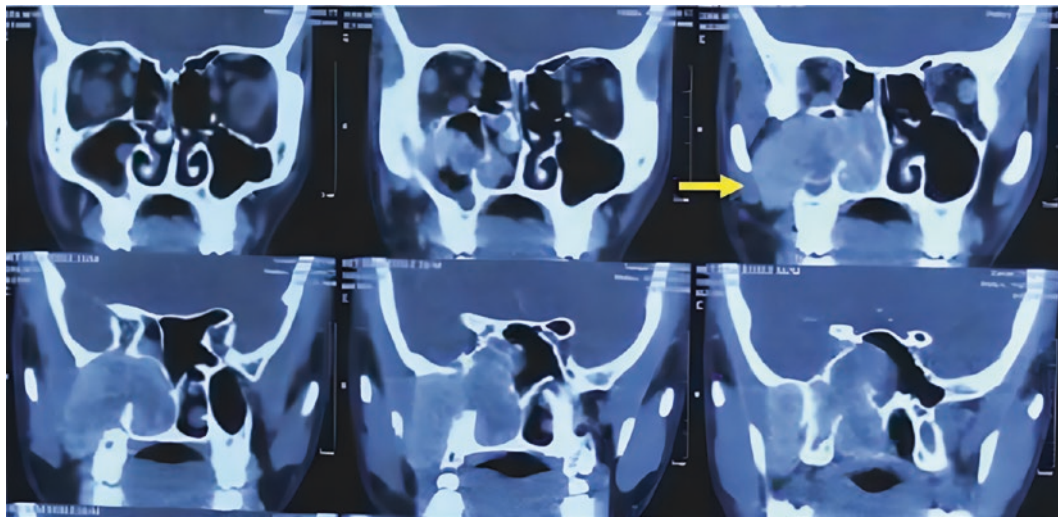

Fig. 9 Hondousa sign widening of the gap between the ramus of the mandible and body of the maxilla because of the lesion

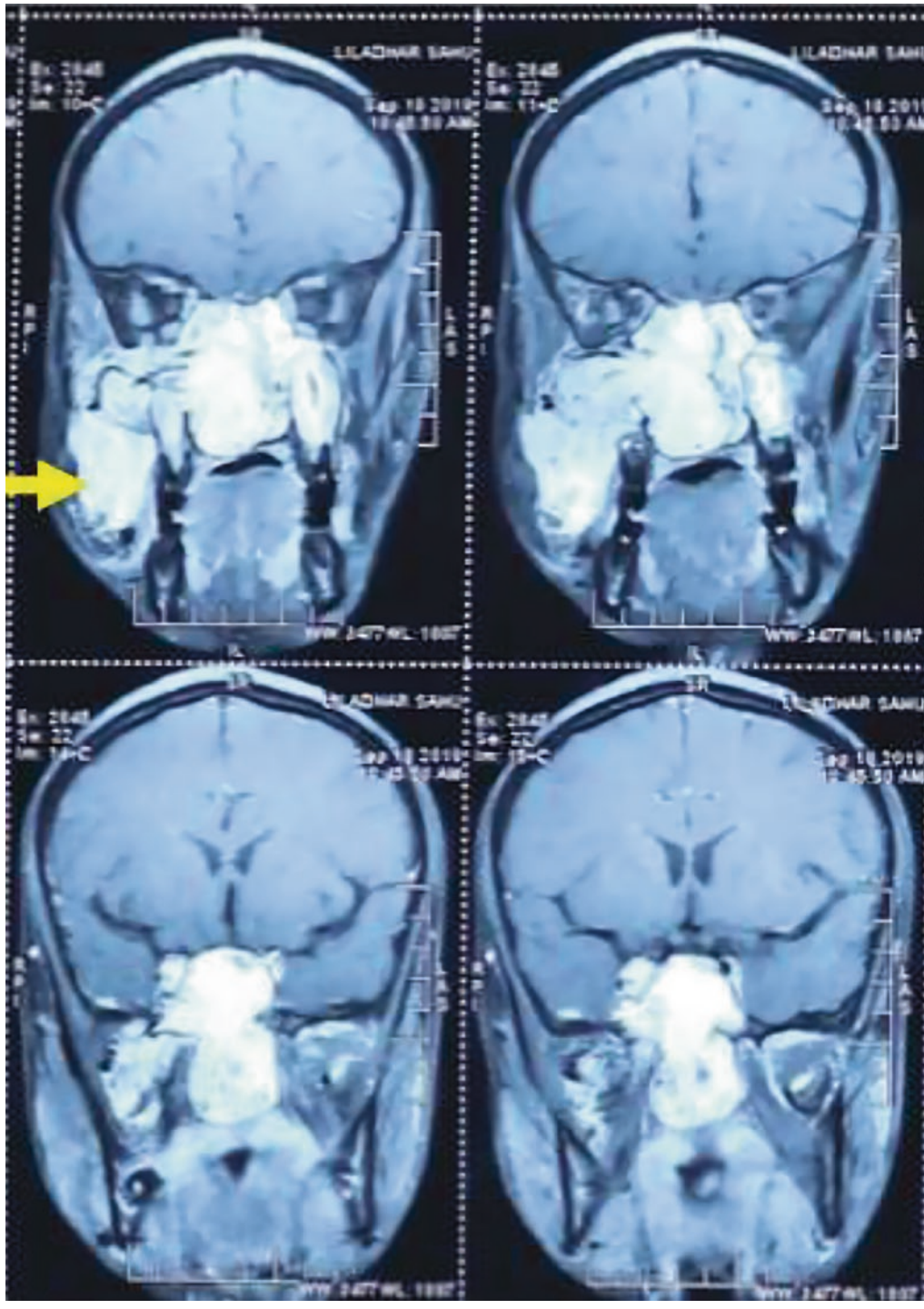

Fig. 10 Hondousa sign as seen in MRI

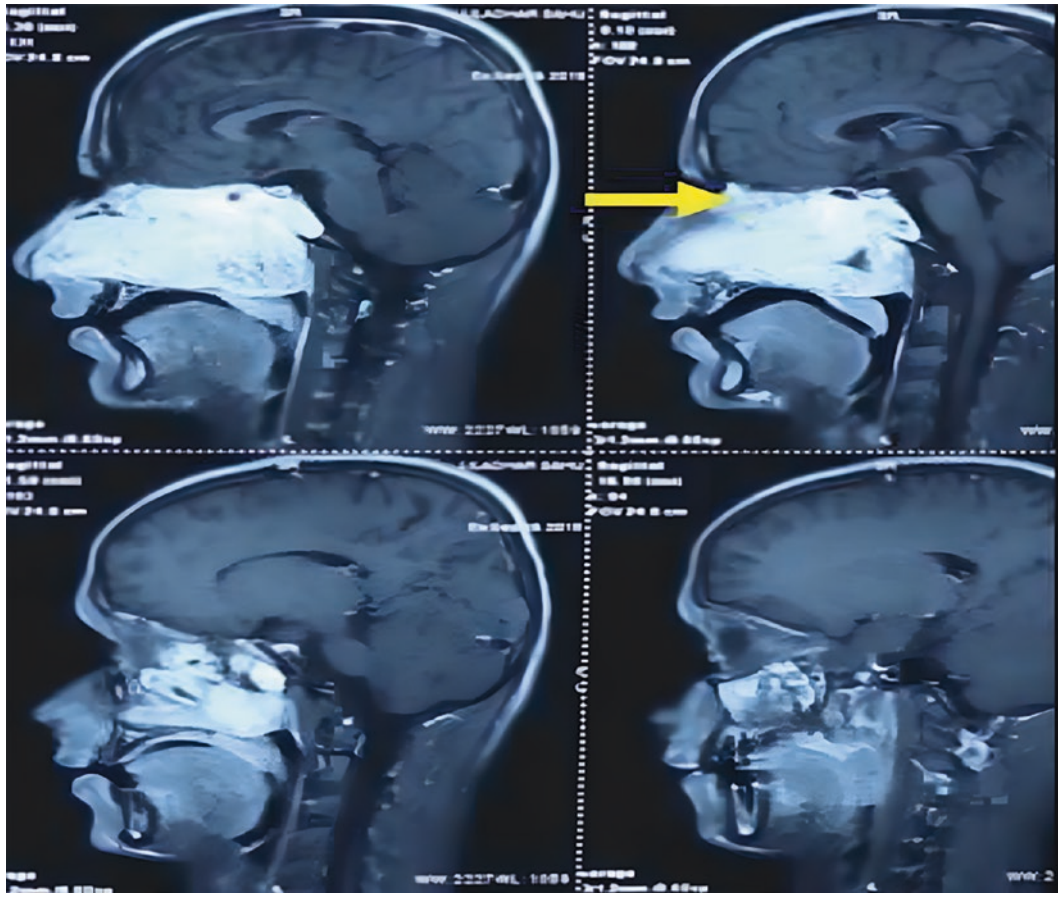

Fig. 11 Para-sagittal view in MRI showing the lesion reaching up to the cribriform plate

Fig. 12 Angiofibroma protruding through the right anterior naris: the lesion can be seen extending through the right anterior naris

Fig. 13 Elevation of the right cheek flap after the modified Weber-Fergusson incision in the subperiosteal plane and exposure of the anterior wall of the maxilla

Fig. 14 Exposure of the tumour after removal of the anterior and medial wall of the maxilla

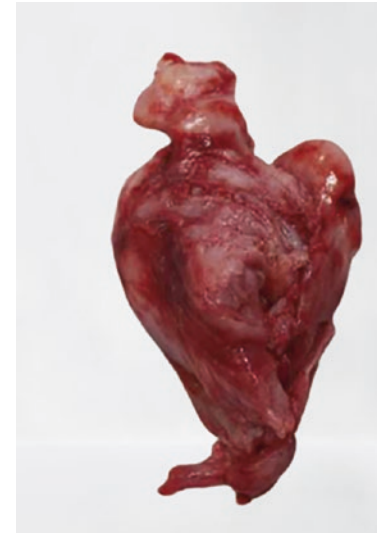

Fig. 15 Excised angiofibroma specimen

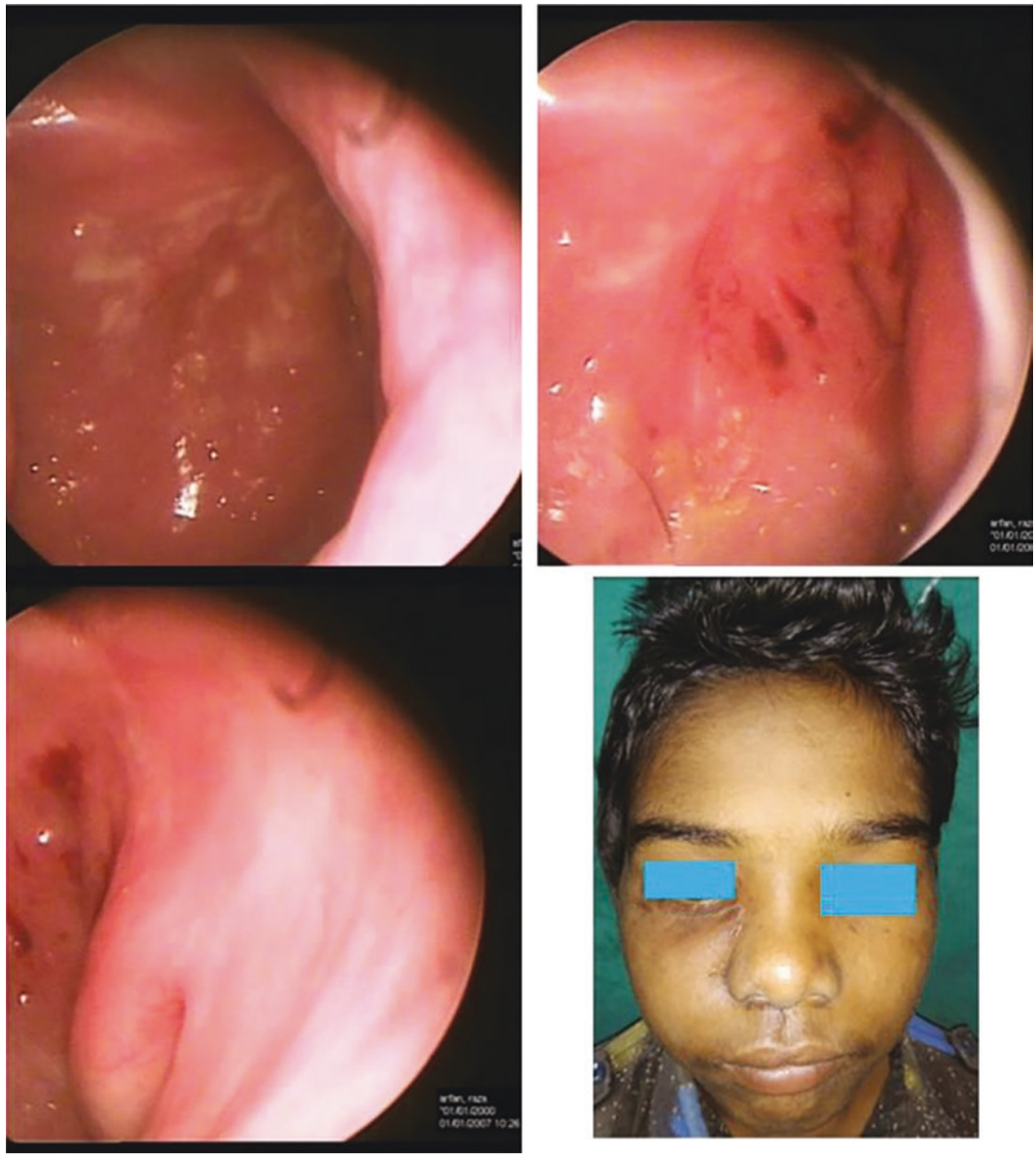

Fig. 16 Follow-up after 6 months: an endoscopic view showing local control of the disease and well-healed scar over the right nasolabial region

Fig. 17 Follow-up after 1 year: the scar is barely visible

7 Inverted Papilloma

Inverted papillomas develop when the epithelium invades the underlying connective tissue stroma, which is more common in adults and rare in children. In the nasal vestibule, middle turbinate, septum or lateral nasal wall, they appear as unilateral red, polypoid lumps. A malignant change may occur in 2% of people [9].

The 2017 WHO classification of sinonasal papillomas are divided into:

- Inverted
- Oncocytic
- Exophytic

7.1 Case 1

A 52-year-old male smoker presented with recurrent inverted papilloma. He was operated thrice outside.

7.1.1 Operative Technique of Medial Maxillectomy: Caveats

- For well-differentiated or low-grade malignant tumours, inverted papillomas and other tumours with a small extent on the lateral wall of the nasal cavity or the medial wall of the maxillary antrum, a medial maxillectomy is suggested.
- Depending on the size and location of the tumour, the surgical procedure involves either a modified Weber-Ferguson incision or a lateral rhinotomy.
- Technically, it is frequently challenging to remove the operative specimen from a medial maxillectomy in a Monobloc method. Mobilization of an ethmoid tumour needs to be done slowly and with great caution due to the sensitivity of ethmoid air cells.
- The anterior bony wall of the maxilla is reached by deepening the skin incision into the soft tissues and muscle of the upper lip and cheek.
- The incision is prolonged superiorly through the soft tissues all the way to the orbit's bony edge.
- The whole medial wall of the maxillary antrum, the side's inferior turbinate, ethmoid air cells and lamina papyracea are all marked for excision.
- The infraorbital nerve close to the orbital rim is carefully protected while the cheek flap is lifted.
- An antrotomy of the anterior wall allows access to the maxillary antrum.
- To provide digital access to the maxillary antrum, a sizable piece of the anterior wall of the maxillary antrum is removed.
- The inside of the maxillary antrum is carefully examined to ascertain the size of the tumour.
- If a medial maxillectomy is feasible, a periosteal elevator is used to lift the medial wall of the orbit's periosteum along the medial orbital rim and then remove it from the lamina papyracea.
- The continuity and shape of the bony rim of the orbit's inferior and inferomedial edges are preserved with extreme care.
- The medial ocular periosteum is dissected as far posteriorly as feasible. The anterior and posterior ethmoidal arteries are transected, ligated or electrocoagulated as they leave the lamina papyracea during this procedure.
- Once the orbital contents have been sufficiently mobilized in an extraperiosteal plane, a dry piece of gauze is placed for haemostasis between the orbital contents and the lamina papyracea.

- The dissection of the nasal ala and its medial retraction, which permits access to the nasal cavity, are the current areas of emphasis.
- The medial wall of the maxillary antrum is divided in a horizontal plane at the base of the nasal cavity using a curved osteotome.
- As observed via the anterior wall antrotomy, this technique is carried out with delicate strokes with a mallet over the osteotome until the posterior edge of the maxillary antrum is reached.
- Similar to this, the medial wall's bony incision is continued cephalad, all the way to the medial wall of the orbit.
- The maxillary orbital surface, the nasal bone and the orbital surface of the frontal bone can all be broken with an osteotome to release the lamina papyracea.
- Finally, the complete surgical specimen encompassing the inferior turbinate and the middle turbinate with the lower ethmoid air cells is removed by transecting the posterior attachment of the surgical specimen close to the posterior choana using angled scissors.
- By electrocoagulating the bleeding spots across the sliced bone surfaces, haemostasis is stabilized.
- With a fine burr, the surgical flaws' sharp bone spicules are smoothed off.
- The nasal cavity and the maxillary antrum are packed using a ribbon gauze roll. The anterior naris is used to remove the packing.
- Using Prolene suture material for the skin and absorbable interrupted sutures for the soft tissues, the remaining incision is stitched close in two layers.

Pre-operative imaging and surgical steps of the same patient are depicted in Figs. 18, 19, 20, 21, 22, 23, 24 and 25.

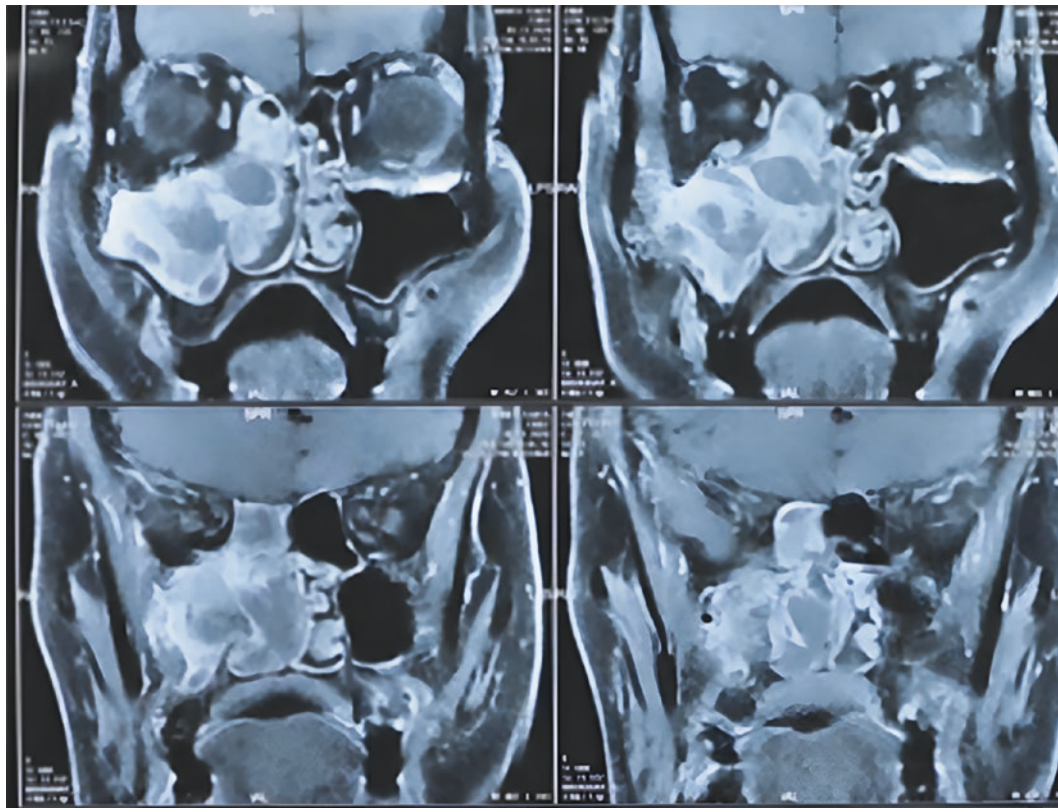

Fig. 18 MRI scan: a coronal plane showing a lesion involving the right medial wall of the maxilla and the lesion extending into the right maxillary, right anterior ethmoid and right nasal cavity

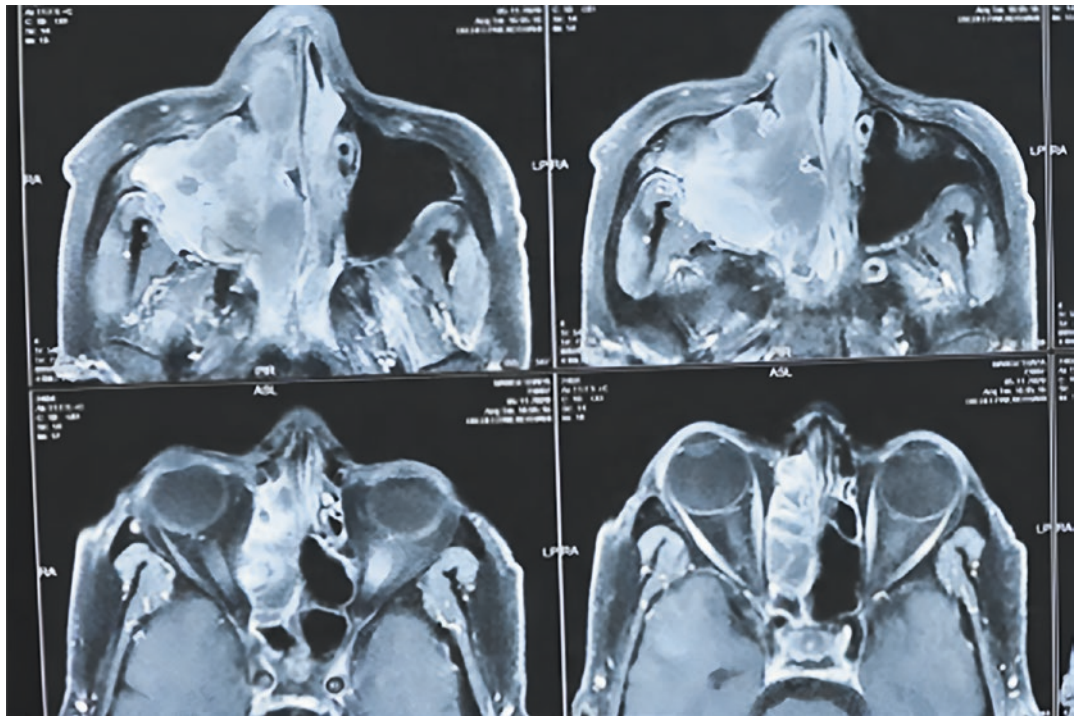

Fig. 19 MRI scan: an axial view showing an extensive lesion involving the right medial wall of the maxilla and the lesion extending into the right maxillary, right anterior ethmoid and right nasal cavity

Fig. 20 Marking of the skin incision

Fig. 21 Elevation of the cheek flap and exposure of the anterior wall of the maxilla

7.1.2 Postoperative Care

- The nasal packing was removed after 72 h.
- However, until the gap has been completely epithelialized, aggressive nasal irrigation and sufficient humidity are essential to eliminate clots and crusts.
- The patient was instructed on nasal irrigation with a catheter, since the anterior nares are the sole route to the maxillary antrum.
- Practically little cosmetic distortion and little functional impairment are the outcomes of the surgery.

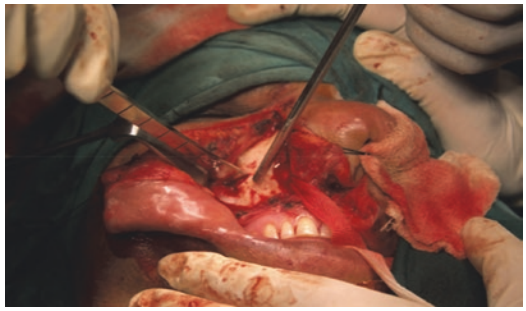

Fig. 22 Opening the anterior wall of the maxilla

Fig. 23 Maxillary cavity after opening the anterior wall of the maxilla and excision of the tumour from the maxillary sinus and the medial wall of the maxilla

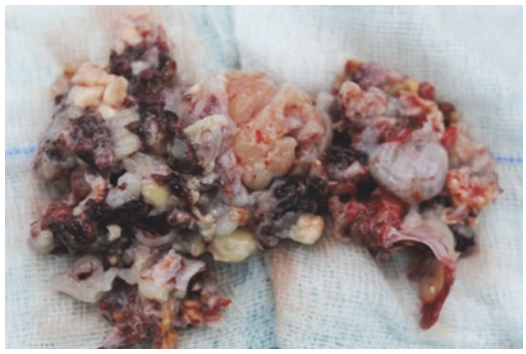

Fig. 24 Specimen of inverted papilloma following medial maxillectomy

Fig. 25 Closure of the skin incision

- The patient binocular vision is normal, and the globe is properly aligned with the opposing side.

8 Giant Cell Tumour

Deep and superficial soft tissue can include soft tissue giant cell tumours, which are uncommon tumours with little malignant potential. These tumours have mononuclear components made up of round, oval or spindle cells as well as equally distributed multinucleated large cells. There are no atypia or obvious signs of mitotic activity. Desmin, cytokeratins, smooth muscle actin and TRAP immunohistochemical stains are quite useful for diagnosis. Since this type of tumour is extremely uncommon to occur in the nasal cavity, soft tissue tumours must be considered in the differential diagnosis of nasal obstruction.

8.1 Case 1

8.1.1 Operative Technique of Infrastructure Maxillectomy: Caveats

- The radiological imaging of giant cell tumour of the right maxilla is seen in Figs. 26 and 27, which includes the upper alveolar ridge and spreads from the area of the second premolar tooth socket to the area of the second molar socket.
- Before beginning an infrastructure maxillectomy, a proper characterization of the extent of the tumour requires appropriate radiographic examination with CT scans in the axial and coronal planes.
- Under general anaesthesia, nasotracheal intubation into the opposing nasal cavity is used to accomplish the surgery.
- Around the visible and palpable tumour, an incision is made in the mucosa of the gingivobuccal sulcus and that of the hard palate, with appropriate mucosal margins.
- If teeth are available, a suitable tooth is pulled far from the tumour's edge through the socket of the bone being removed, protecting the integrity of the neighbouring remaining tooth.
- The mucosal incision is prolonged intraorally into the soft tissues and up to the bone anterior wall of the maxilla.
- Before dividing the bones, this incision is expanded circumferentially through the soft tissue to separate all the soft tissue attachments of the hard palate and the lower part of the maxilla.
- An osteotome is utilized to split the remaining bone attachments and remove the specimen in a Monobloc using bone cuts through the previously described mucosal incision.
- The bottom part of the maxillary antrum, which was taken in one piece together with the surrounding hard palate and the maxilla's alveolar process, is seen in the anterosuperior view of the surgical specimen (Fig. 31).
- It is not necessary to curette out the remaining antrum if the mucosa does not exhibit any chronic inflammatory alterations.
- To keep the packing in place, a dental obturator that had previously been made is now linked to the remaining teeth.
- Nasal packing has to be done.
- The packing may be taken out in 2–3 days, and a temporary dental obturator is made while waiting for the surgical defect to com-

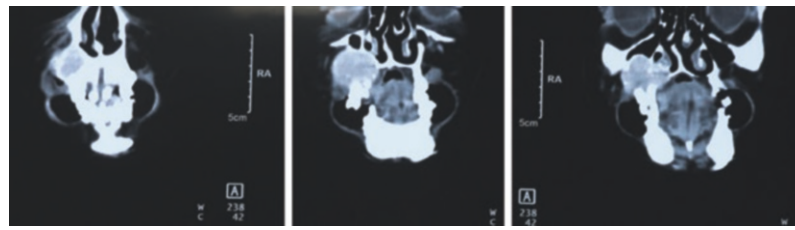

Fig. 26 An expansile radiolucent lesion involving the anterolateral wall of the right maxilla

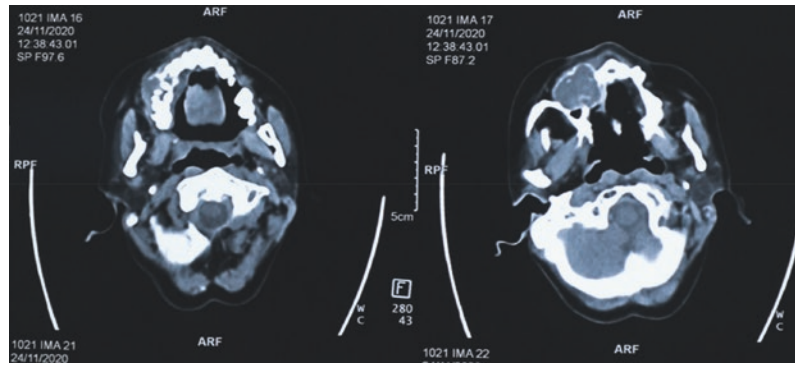

Fig. 27 CT scan: an axial view showing and expansile radiolucent lesion present between the right upper second premolar and first molar tooth

pletely epithelialize. At that moment, maxillofacial prosthodontists create a permanent removable dental prosthesis.

Pre-operative imaging and surgical steps of the same patient are depicted in Figs. 28, 29, 30, 31 and 32.

Fig. 28 Marking of the skin incision

Fig. 29 Elevation of cheek flap exposing the anterior wall of the right maxilla

Fig. 30 Surgical site after infrastructure maxillectomy, maxillary antrum and the cut end of the upper alveolus visualized

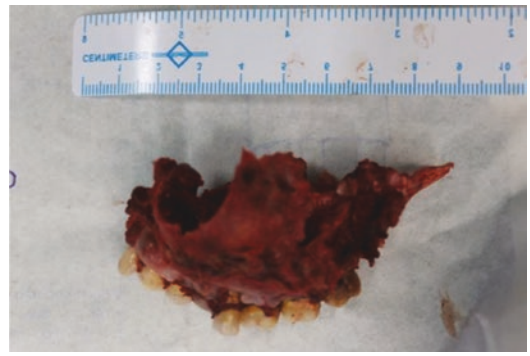

Fig. 31 Specimen showing en-bloc resection

Fig. 32 Final closure

9 Squamous Cell Carcinoma of the Maxilla

The most frequent sinonasal cancer is still squamous cell carcinoma. However, it can be challenging to pinpoint the specific location of the tumour because these areas are frequently afflicted, as well as the nasal cavity and anterior ethmoid. Most often, it starts in the maxillary sinus. Differentiation levels vary and may worsen with time [10], while often, a combination of surgical and chemoradiation therapy is performed. However, chemoradiation alone may be curative in sinonasal carcinomas with poor or undifferentiated carcinoma. Rarely is the columella or nasal septum the main location. Due in part to the potential of bilateral cervical node metastasis, these tumours have a dismal prognosis.

9.1 Case 1

A 51-year-old man presented with a 3-month history of growth on the left side of the maxillary sinus involving the hard palate. A biopsy was suggestive of moderately differentiated squamous cell carcinoma.

9.1.1 Operative Technique: Caveats

- A subtotal maxillectomy effectively eliminates the infrastructure and superstructure of the maxilla together with the whole maxilla, with the exception of the orbital floor.
- It is referred to as a "complete maxillectomy" if the orbital floor is also removed during the procedure. To avoid ptosis of the globe in the situation, the orbital floor rebuilding should be taken into account.
- An orotracheal tube is used to maintain the patient's general anaesthesia when they are put on the surgical table.
- Using the Weber-Ferguson incision, the maxilla was exposed.
- For the elevation of the cheek flap, a skin incision is created with a scalpel, and electrocautery is then performed, which offers good haemostasis.
- The gingival sulcus and the whole thickness of the upper lip are split.
- The superior labial artery has to be tied up if it is bleeding.
- An incision is made in the mucosa of the upper gingivobuccal sulcus, staying close to the gingiva, to raise the upper cheek flap. It is elevated full thickness, staying directly above the maxillary periosteum until its posterolateral side is revealed.
- The infraorbital nerve and its entrance into the soft tissues of the face are exposed by continuing to elevate the cheek flap in this area.
- The infraorbital nerve should be carefully maintained if only the lower part of the maxilla is to be removed in order to maintain the cheek's cutaneous sensations.
- In order to reach the pterygomaxillary fissure, it is crucial to raise the cheek flap all the way back to the posterolateral surface of the maxilla. This will expose the zygoma's underside.
- By slicing the soft tissues along the ala of the nose and through the mucosa of the lateral wall of the nasal cavity, access is gained to the nasal cavity.
- On the other side, a mouth gag is now used, and the oral cavity is opened as widely as possible to reveal the hard palate and alveolar process.
- Just below the infraorbital foramen, the anterior wall of the maxilla is divided using a high-speed power saw with a very tiny blade.
- To produce the desired line of resection through the anterior wall of the maxillary antrum and to enable the excision of the lower half of the maxilla as the surgical specimen, the bone cut is prolonged anteriorly and posteriorly.
- The infraorbital nerve and its entrance into the soft tissues of the face are exposed by continuing to elevate the cheek flap in this area.
- To expose the alveolar process, the hard palate, the zygoma and the area around the posterolateral aspect of the maxilla, the oral cavity is opened as far as feasible.
- The line of fracture is carried between two teeth if there is space between them. It is advised to extract one tooth along the antici-

pated line of transection of the alveolar process since, if the teeth are intact, it is extremely possible that the final tooth on the remaining alveolar process will become loose and maybe fall out.

- The bone that was previously cut between the previously formed transverse line of transection and the alveolar process is connected using the power saw once again.
- The hard palate mucosa is now the centre of attention. A tongue depressor is used to ensure sufficient exposure with the mouth open wide.
- In order to maintain good mucosal margins in all directions, an incision is created in the hard palate mucosa surrounding the primary tumour using a needle-tip electrocautery.
- The tumour is circumvented by anteriorly starting an incision at the maxillary tubercle and curls anteriorly.
- Prior to the alveolar process holding the left-side incisors and canines, the incision is extended behind it.
- To finish the specimen's circumferential mobilization, the mucosal incision comes to a point where it meets the removed first molar tooth's socket.
- The length of the hard palate's mucosal incision is deepened into the mucoperiosteum and up to the bone.
- The incision in the hard palate mucosa and a part of the soft palate is visible in a close-up image of the surgical field.
- Along the line of the mucosal incision, the hard palate is divided using a power saw.
- Due to bleeding from the palatine arteries, internal maxillary artery branches and the posterior wall of the maxilla as well as the pterygoid fossa, there is significant bleeding at this stage of the surgery.
- It is imperative to speed up this procedure, since attempts to stop the bleeding are fruitless until the surgical specimen is removed.
- An osteotome is used to join the fracture lines after all the bone incisions have been performed with the power saw, allowing the specimen to be rocked over the soft tissue attachments.
- The posterior soft tissue attachments of the specimen (the pterygoid muscles) are split with electrocautery or Mayo scissors, and the surgical specimen of the bottom half of the maxilla is removed.
- The internal maxillary artery, sphenopalatine artery and smaller blood vessels of the soft palate are often the sources of bleeding at this stage.
- The internal maxillary artery can be ligated to stop bleeding, or a chromic catgut suture ligature can be inserted through the pterygoid muscle stumps.
- However, bleeding from the sphenopalatine artery is seldom controlled by ligation; as the vessel's stump is typically in a bony cleft, electrocoagulation is the best method for achieving haemostasis.
- Now a burr has been used to smooth out all the jagged spicules on the bony margins.
- Interrupted absorbable sutures are used to approximate the cut margins of the mucosa on the soft palate's anterior and posterior borders.
- Bacitracin solution is used to irrigate the wound, and blood clots are removed.
- Starting at the roof of the antrum, ribbon gauze is applied digitally to the defect and held in place by light digital pressure so that it may fit into the antrum's nooks and corners.
- The dental obturator that was previously made is now applied and fastened with wires. To restore the hard palate section that was removed, the obturator is connected to the remaining teeth. If the patient is missing teeth, drill holes are used to connect the obturator to the remaining alveolus.
- The dental obturator's placement restores the missing hard palate tissue, enabling the patient to swallow liquids and soft meals without any trouble right away after surgery.
- Utilizing non-absorbable sutures for the skin and absorbable interrupted sutures for the subcutaneous tissues, the skin incision is stitched shut in two layers.
- To produce a higher aesthetic effect, careful consideration is given to an accurate approximation of the skin margins.

- In that sense, precise realignment of the skin borders along the ala and the nasal cavity's floor is crucial.
- On the first day following surgery, there is a mild amount of oedema of the lower eyelid and cheek, but this swelling is temporary and often goes down on its own in 2 to 3 days.
- To lessen postoperative oedema, place ice packs over the cheek.
- The majority of patients can handle a soft diet a day following surgery.
- Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 33, 34, 35, 36, 37, 38 and 39.

9.1.2 Postoperative Care

- Following a partial maxillectomy, the postoperative care of the patient focuses on maintaining excellent oral hygiene and caring for the face incision until sutures are removed.
- Because they act as a nidus for infection and can occasionally induce wound separation,

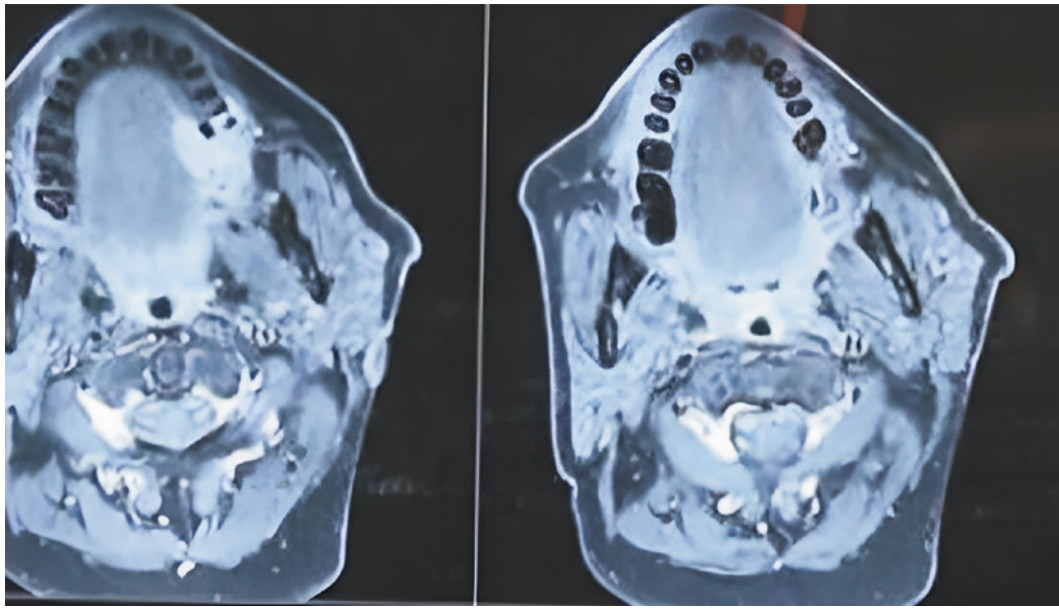

Fig. 33 MRI scan showing lesion

Fig. 34 Marking of the skin incision

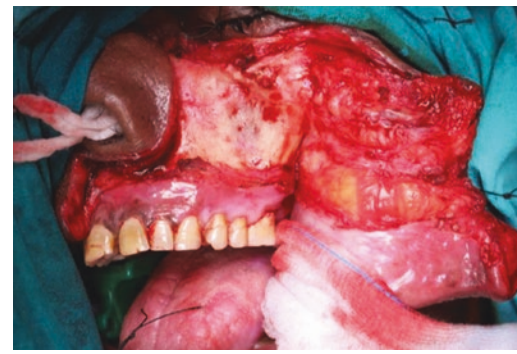

Fig. 35 Elevation of the skin flap and exposure of the anterior wall of the left maxilla

Fig. 36 Surgical site after orbital plate preserving subtotal maxillectomy

Fig. 37 Surgical specimen showing the ulceroproliferative growth with adequate margin

Fig. 38 Closure of skin incision with obturator in situ

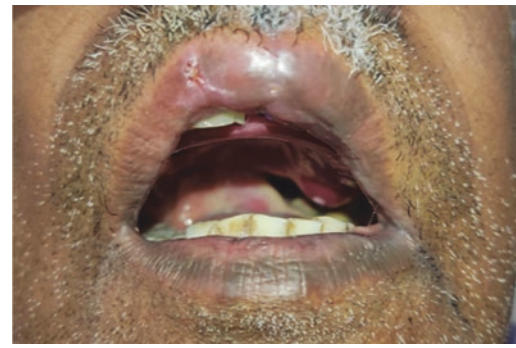

Fig. 39 Two weeks post-surgery with obturator in situ

suture line clots and crusts are carefully removed from above the suture line.

- Sometimes it's important to apply ice pack compresses to the cheek if there is chronic swelling or an inflammatory reaction.
- On the second postoperative day, the patient is instructed to regularly rinse and irrigate their mouths with a warm water and baking soda solution to maintain their mouths free of debris and secretions.
- On the third postoperative day, the packing is delicately removed.
- The dental obturator is removed by cutting the wires 1 week following surgery.
- The prosthodontist now creates an interim obturator, which is secured to the remaining teeth using clasps.
- In edentulous individuals, early retention of a prosthesis can be challenging and frequently disappointing.
- Up until the surgical defect's skin graft has completely healed, oral and nasal irrigations are maintained.
- Approximately 6–8 weeks later, a permanent dental obturator is made.
- The patient's ability to talk normally and consume all sorts of food is restored thanks to this permanent obturator.

10 Odontogenic Tumours

According to the WHO, ameloblastic fibroma (AF) and associated diseases are neoplasms made up of proliferating odontogenic epithelium embedded in cellular ectomesenchymal tissue that resembles dental papilla and with variable degrees of inductive transformation and creation of dental hard tissue [11].

Ameloblastic fibroma is an uncommon mixed odontogenic tumour that makes up about 2.5% of all odontogenic tumours [12, 13]. It typically affects those under 30 (70% of patients are younger than 20 years at presentation). There are no racial or sexual preferences [14–16]. Most often (approximately 80% of the time), AF devel-

ops in the mandible, with the remaining occurrences developing in the maxilla [14, 15, 17, 18]. AF patients typically experience discomfort, tooth eruption failure and/or jaw oedema [12, 14]. It is difficult to identify AF from a straightforward ameloblastoma radiographically because it resembles a radiolucent multilocular or unilocular cyst [12, 14, 17].

10.1 Case 1

A 4-year-old female child presented with a growth in the right upper alveolus for 4 months, with a biopsy suggestive of ameloblastic fibroma.

Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 40, 41, 42, 43, 44, 45, 46, 47 and 48.

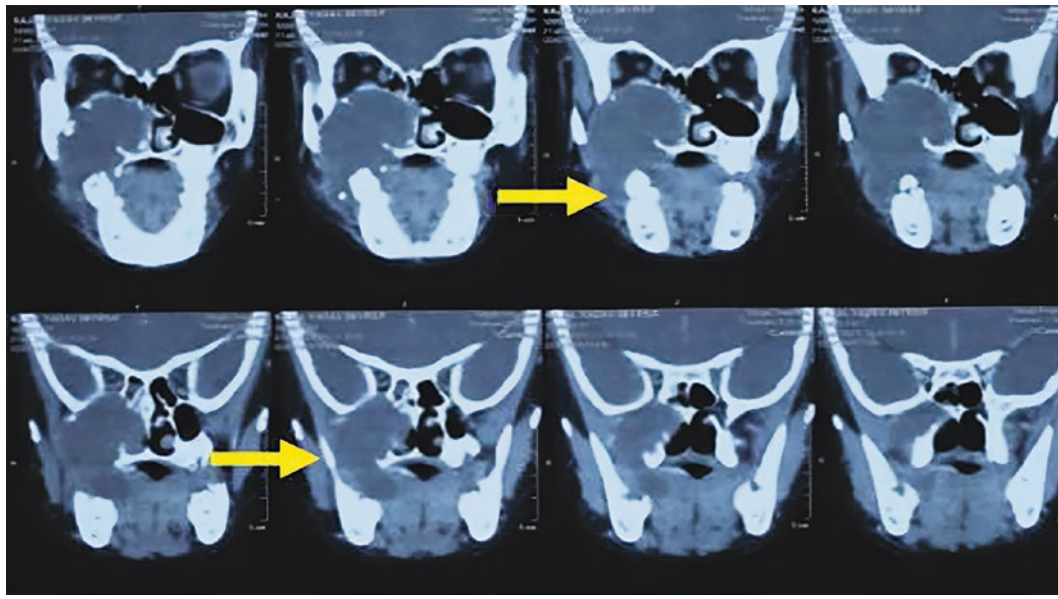

Fig. 40 CT scan: coronal cuts showing the lesion in the right maxilla with the erosion of the anterolateral wall of the maxilla

Fig. 41 Skin incision and elevation of cheek flap

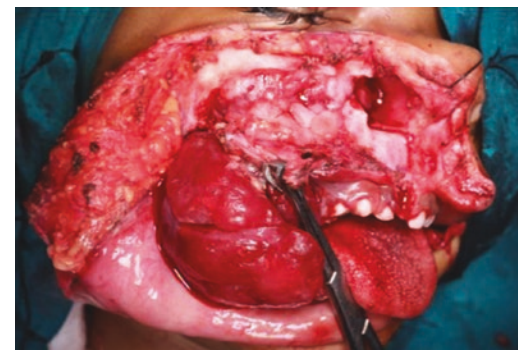

Fig. 42 Elevation of the cheek flap, which shows the lesion eroding the anterolateral wall of the maxilla and extending to the oral cavity at the level of the upper gingivobuccal sulcus, smooth surface of the lesion in the oral cavity can be appreciated

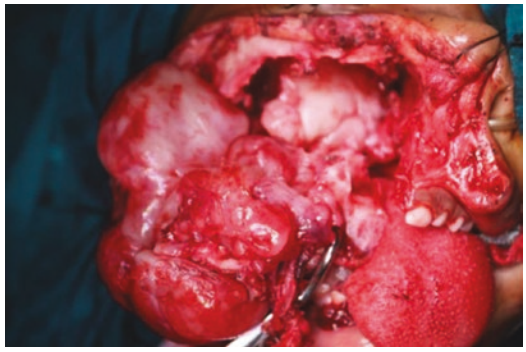

Fig. 43 Tumour exposed in the right maxillary sinus after removal of the anterior wall of the maxilla

Fig. 44 Surgical defect after orbital plate-preserving subtotal maxillectomy

Fig. 45 Excised en-bloc specimen

Fig. 46 Surgical defect after orbital plate-preserving subtotal maxillectomy with an obturator in situ

Fig. 47 Postoperative follow-up after 6 months

Fig. 48 Follow-up after 1-year, locoregionally controlled disease

11 Osteosarcoma

The majority of instances of osteosarcoma (OS), a malignant mesenchymal tumour that seldom affects the maxilla, show as painful swelling of the area around the maxilla as their initial clinical manifestation. The most significant determining variables of prognosis are early diagnosis and extensive surgical excision of the tumour. To hasten the diagnosing process, careful consideration should be given to OS's atypical clinical manifestations.

11.1 Case 1

A 25-year-old man presented with a recurring tumour in the right nasal cavity had right partial maxillectomy with complete palatectomy and temporalis muscle rotation flap restoration after the first undergoing surgery for a giant cell lesion.

Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59 and 60.

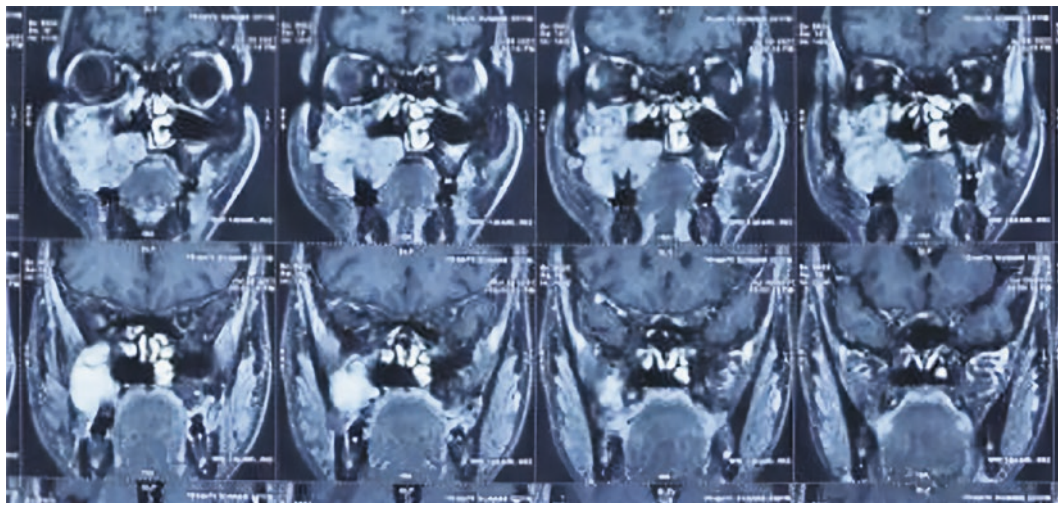

Fig. 49 MRI scan showing the lesion in the right maxillary sinus and involving the palate: lesion over the palate is seen crossing the midline anteriorly

Fig. 50 Marking the skin incision

Fig. 51 Elevation of the cheek flap over the anterior wall of the right maxillary in the submuscular plane

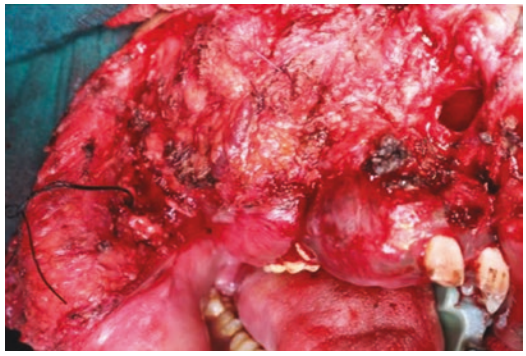

Fig. 52 Complete exposure of the tumour

Fig. 55 Skin marking for temporalis muscle rotation flap

Fig. 53 Surgical defect following extirpation of the tumour in the right maxilla by subtotal maxillectomy with total palatectomy

Fig. 56 Elevation of the scalp skin to expose the temporoparietal fascia

Fig. 54 Surgical specimen

Fig. 57 Rotation of the temporalis muscle onto the right maxillary defect

Fig. 58 In setting the temporalis muscle to completely cover the maxillary defect

Fig. 59 Follow-up after 1 year after the completion of adjuvant radiotherapy with well-healed surgical scar

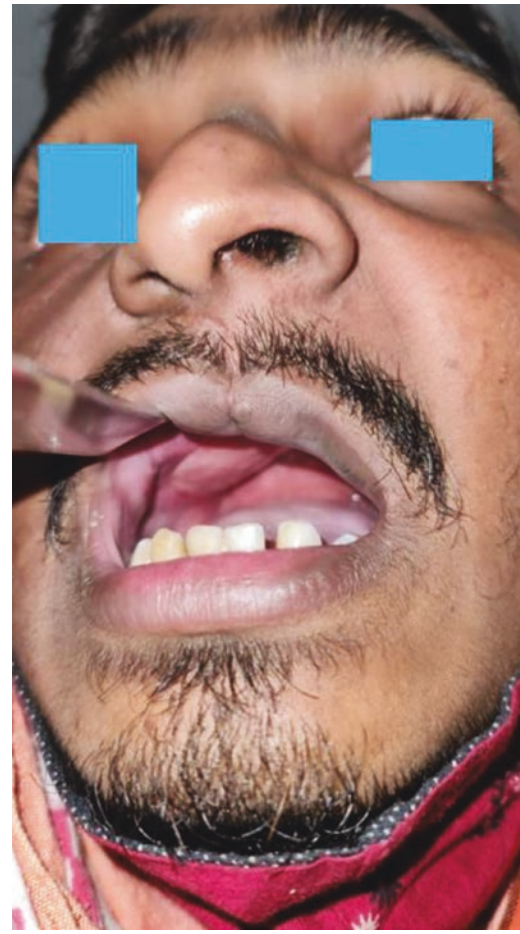

Fig. 60 Follow-up after 1 year showing locally well-controlled lesion

12 Rhabdomyosarcoma

Rhabdomyosarcomas are the most prevalent paranasal sinus malignancy in paediatric patients, with the orbit being the most frequent subsite overall. Rhabdomyosarcomas are among the tumours with tiny, rounded blue cells that may be seen on histology and are generated from the primitive mesenchymal tissue with myogenic differentiation. There are four categories in the current histologic categorization of

rhabdomyosarcoma [19]. The most prevalent kind, known as the embryonal type, often affects newborns and younger children (incidence of 55–65%). Botryoid and spindle cell subtypes are included in the embryonal categorization and are typically thought to have the best prognosis. The alveolar form (20–30% incidence) more frequently affects teenagers and has a worse prognosis potential, necessitating typically more intense multimodality therapy. The undifferentiated type is poorly characterized with no clear histologic myogenesis or differentiation, while the anaplastic type—previously known as the pleomorphic type—primarily affects adults. Due to their quick development and high rate of distant dissemination, both of these varieties have dismal prognosis. Given the proximity of important tissues to the head and neck, only a biopsy is frequently done there, since recurrence might be significant despite rigorous resection. Neoadjuvant chemoradiation is encouraged by certain regimens to make tumours more amenable to excision. Overall, 5-year overall survival is good, notably for orbital rhabdomyosarcoma, which has a 95% survival rate, compared to a 74% survival rate for parameningeal locations [20].

12.1 Case 1

A left nasal mass has recurred in a 3-year-old kid. He underwent the first surgery in April 2021 using an endoscopic technique since his HPR suggested an AC polyp. A second biopsy of the recurring tumour again revealed evidence of an AC polyp, and the final HPE was reported as rhabdomyosarcoma.

Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 61, 62 and 63.

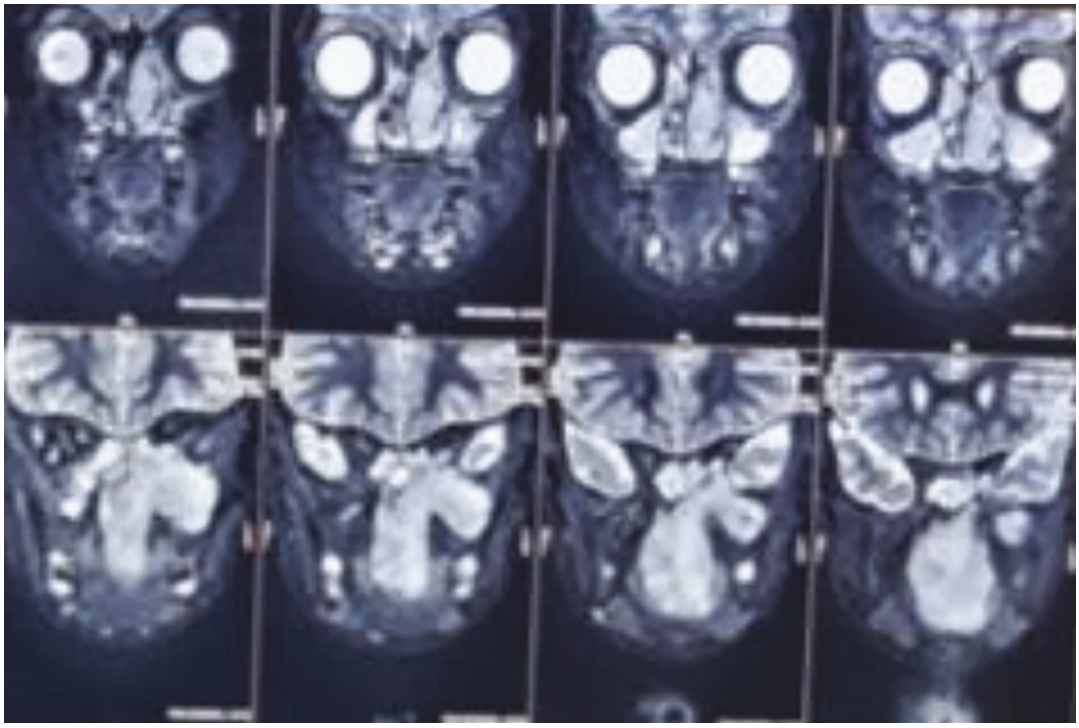

Fig. 61 MRI scan shows a hyperintense lesion present in the left maxillary sinus

Fig. 62 Closure of the skin incision

Fig. 63 Follow-up after 6 months showing a well-healed surgical scar

13 Olfactory Neuroblastoma (OAN)

The basal cells that make up the olfactory neuroepithelium are where OAN comes from. OAN makes up less than 5% of sinonasal cancers [21]. The incidence of this tumour has a bimodal distribution, with peaks at 20 and 50 years of age. Women are more likely to have it. A neuroendocrine tumour called OAN has the ability to create peptides that can lead to paraneoplastic illnesses. In the literature, cases of patients with Cushing's syndrome, OAN-producing vasoactive peptide-induced hypertension or improper antidiuretic hormone production have been reported.

OAN must be distinguished from small cell carcinoma, rhabdomyosarcoma, lymphoma, neuroendocrine tumour and sinonasal undifferentiated carcinoma (SNUC), all members of the category known as "small round blue cell tumours." Therefore, a histopathological assessment by an experienced pathologist is advised. OAN is normally devoid of keratins and exhibits the neuroendocrine markers neurone-specific enolase, synaptophysin and chromogranin. S-100 may only be positive at the tumour's perimeter, which can help distinguish OAN from sinonasal melanoma. Vimentin, actin and desmin negativity rule out rhabdomyosarcoma [22].

Hyams et al. [23], Showed that the degree of differentiation, the tumour architecture, the mitotic index, the nuclear polymorphism, the fibrillary nature of the matrix and tumour necrosis were used to construct a four-point histological grading system for OAN. This tumour may either be indolent (grade 2) or exceedingly aggressive (grade 4). On the use of cytogenetics in the OAN diagnosis, there is minimal information. While some people are lucky enough to live for more than 20 years, others are less fortunate and pass away within a few months from extremely aggressive illnesses with extensive metastases [24]. OAN can penetrate the dura and

anterior cerebral fossa up to 25% of the time. At the time of presentation, cervical node metastases are visible in 5% of patients, and distant metastases are found in around 7% of patients [24]. For OAN, disease-specific staging methods have been developed. The most useful and commonly used systems are those attributed to Kadish et al. [24], Morita et al. [25] and Dulguerov and Calcaterra [26].

13.1 Case 1

A 32-year-old lady presented with a mass in the nasal cavity involving the skin, pre-operative biopsy form the mass showed Esthesioneuroblastoma.

Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 64, 65, 66 and 67.

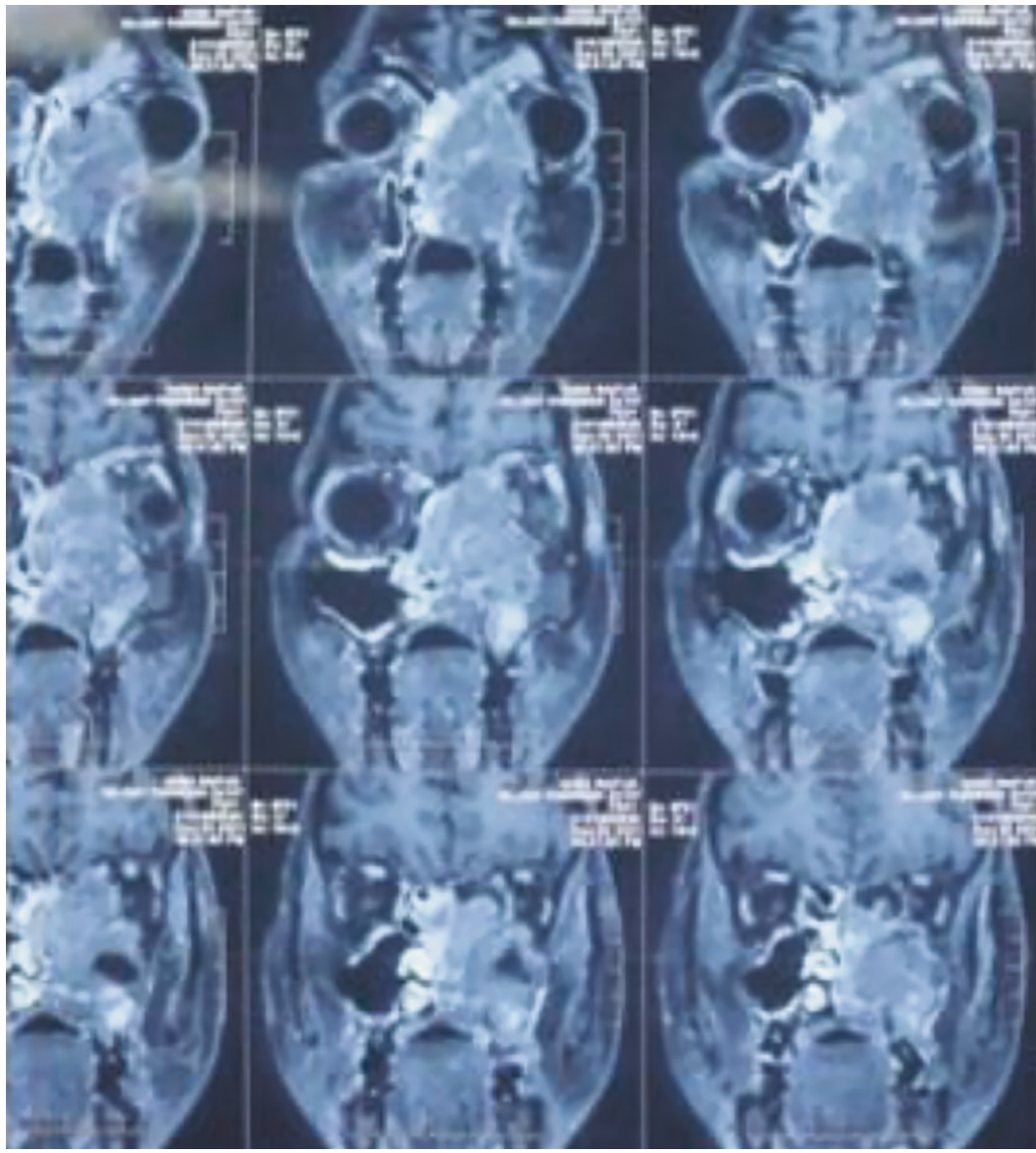

Fig. 64 MRI scan coronal section showing lesion involving the left nasal cavity, lesion seen involving the cribriform plate

Fig. 65 Marking of skin incision

Fig. 66 Elevation of the cheek flap

14 Adenoid Cystic Carcinoma

Nine percent of sinonasal cancers are adenocarcinomas. Similar to SCC, it favours men and typically affects people in their sixth and seventh decades of life. The upper nasal cavity and ethmoid sinuses are the typical locations for adenocarcinomas. They seldom develop metastases and grow slowly. Sinonasal adenocarcinoma has a number of known histological subgroups, including papillary, sessile, mucoid, neuroendocrine, intestinal and undifferentiated. The least aggressive variety is papillary adenocarcinomas. The kind most frequently linked to tumours brought on by wood workers is intestinal. The prognosis for sessile and mucoid adenocarcinomas is the poorest [27].

Fig. 67 Follow-up after 3 months with a well-healed scar over the left nasolabial fold region

14.1 Case 1

A 43-year-old lady presented with intermittent nasal bleed for 3 months and a mass in the left nasal cavity for 2 months; biopsy was suggestive of adenoid cystic carcinoma.

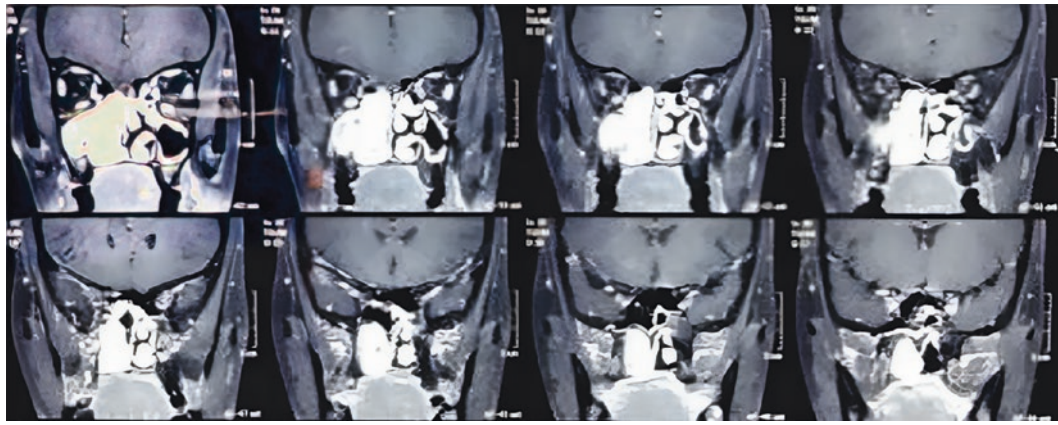

Fig. 68 MRI scan shows a lesion involving the right maxilla and right-sided nasal cavity

Fig. 69 Palate and orbital plate-preserving maxillectomy defect following surgical extirpation of the tumour

Fig. 70 Follow-up after 6 months

Pre-operative imaging, surgical steps and follow-up of the same patient are depicted in Figs. 68, 69 and 70.

15 Postoperative Management

- The conventional/Merocel pack is to be removed after 48 h, preferably in OT, to look for any bleeding at the surgical site.
- Saline irrigation is to be started after pack removal.
- Endoscopic suctioning and cleaning of the nose every week until crusting subsides.
- Regular follow-up.

16 Adjuvant Treatment

- Generally reserved for locally advanced primary tumours of the nose and paranasal sinuses.
- *Radiotherapy:*
  - Positive or close margins
  - Nodal metastasis
  - Unresectable tumours
  - Recurrence (in specific settings)
- *Chemotherapy* is used when previous surgery and radiation have failed.
- *Hormone therapy* has been proposed due to the androgen receptors associated with JNAs in an attempt to decrease tumour size and vascularity.
- *Oestrogen* has been shown to decrease the size and vascularity of the tumour but has feminizing side effects.

17 Mucormycosis

The coronavirus disease 2019 (COVID-19) pandemic has been one of the most significant global health crises in recent history. The World Health Organization (WHO) classified COVID-19, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV2), a global pandemic in March 2020. As the world grapples with the devastating impact of this virus, several countries have witnessed a sharp rise in cases of a rare fungal infection known as mucormycosis. In India, this epidemic of mucormycosis has emerged as a significant concern for healthcare providers. The sudden surge of this deadly fungal infection has put an additional burden on an already overwhelmed healthcare system in India. The fungal invasion of blood vessels results in mycotic thrombosis, ischemic infarction and necrosis of affected host tissues, making mucormycosis a potentially lethal infection.

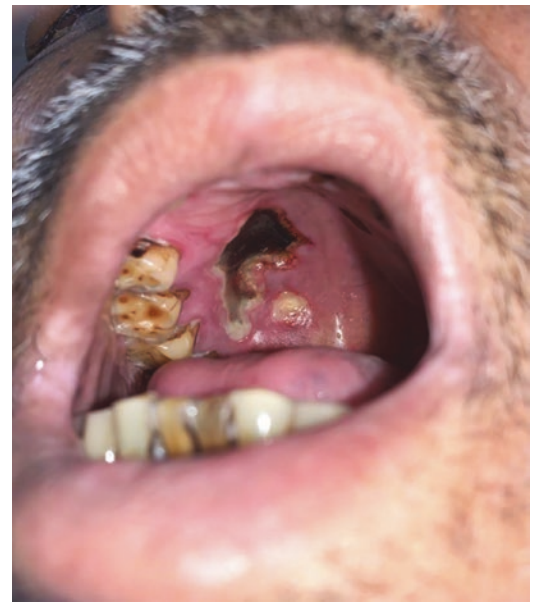

Fig. 71 Mucormycosis of hard palate causing oro-nasal fistula

Fig. 72 Mucormycosis of the alveolar process of the hard palate

Fig. 73 Orbital extension of mucormycosis causing the frozen globe

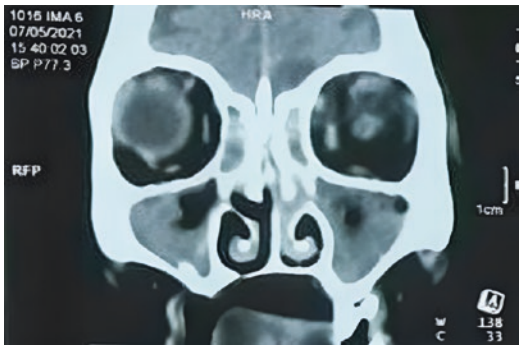

Fig. 74 Contrast-enhanced CT images—coronal section—heterogeneous lesion involving bilateral maxillary sinus, bilateral ethmoidal sinus and extension into the nasal cavity

Fig. 75 Gadolinium-enhanced T1-weighted coronal MRI showing heterogeneous lesion involving bilateral maxillary sinus right more than left with right orbital involvement

Fig. 76 Excision specimen showing the infrastructure maxilla, black necrotic inferior turbinate and middle turbinate

Fig. 77 En-bloc resection of the infrastructure of the maxilla, showing disease involving the hard palate

Fig. 78 Surgical site following left medial maxillectomy and left orbital exenteration

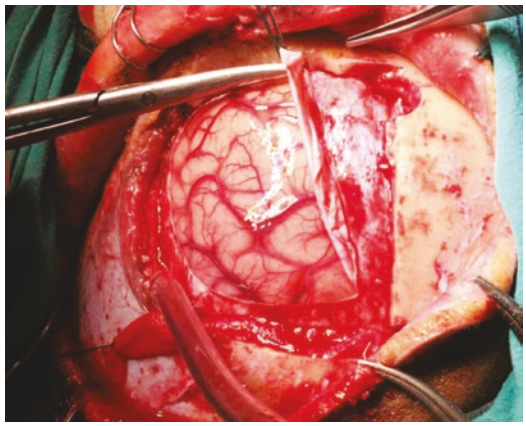

Fig. 79 Intracranial extension of mucormycosis for which craniotomy was performed

Fig. 80 Third month of follow-up of the patient, well-healed orbital cavity following exenteration with a well-healed scar which is barely visible

Various presentations of mucormycosis, intraoperative findings and the rehabilitation prosthesis are depicted in Figs. 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85 and 86.

Fig. 81 Dental obturator lateral view

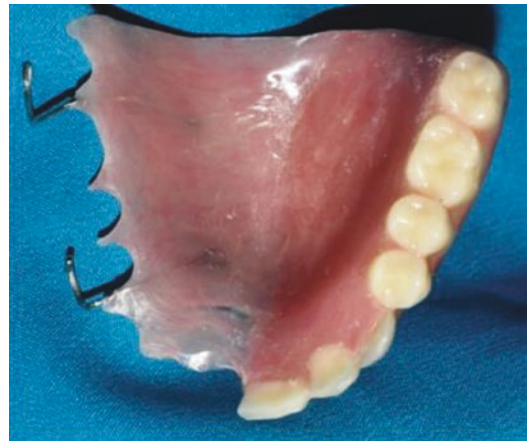

Fig. 82 View of dental obturator from below

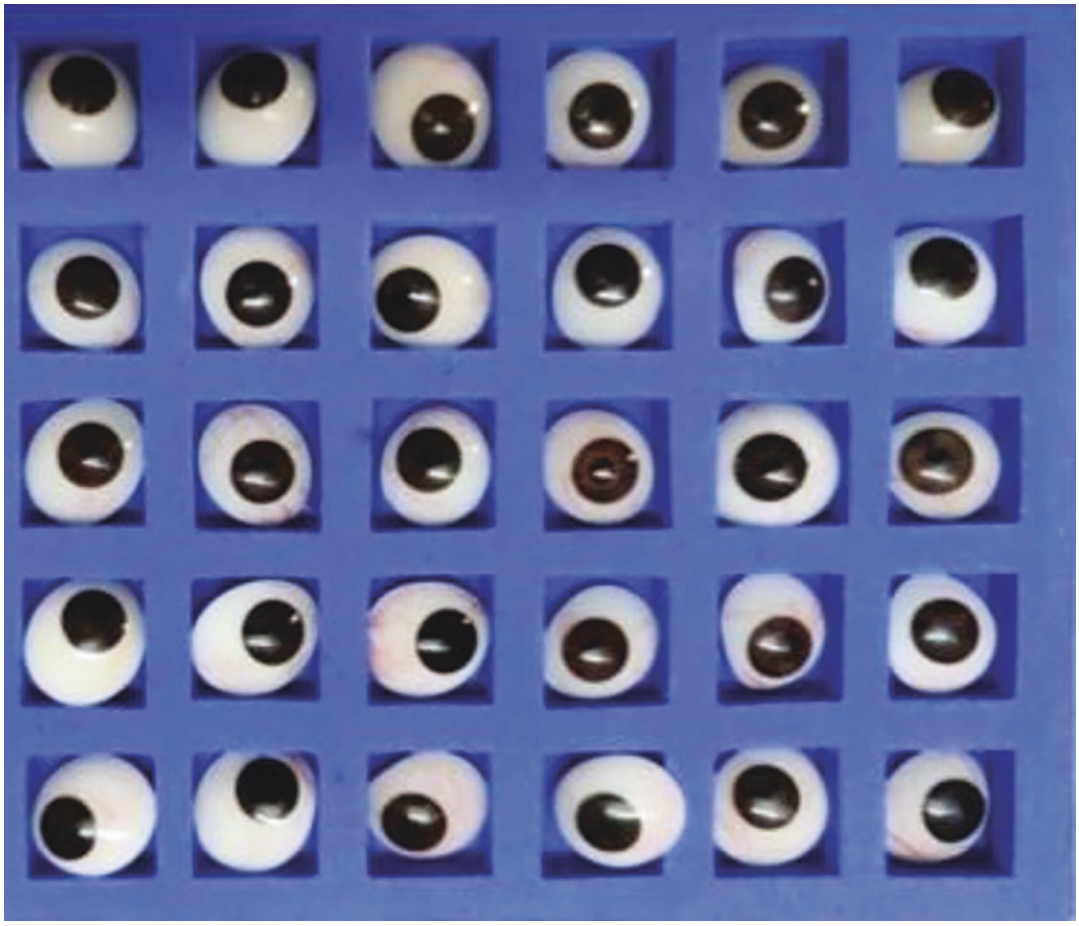

Fig. 83 Ocular prosthesis

Fig. 84 Follow-up case of right infrastructure maxillectomy before placement of the obturator

Fig. 85 Follow-up case of right infrastructure maxillectomy after placement of the obturator

Fig. 86 Before and after use of ocular prosthesis

18 Rehabilitation

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Skull Base

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1 Introduction

Progress in microsurgery, endoscopy, imaging and reconstruction has improved surgical options for cranial base lesions. Adjuvant non-surgical therapies have improved outcomes for certain illnesses, leading to excision being seen as the primary therapeutic modality for many once thought to be incurable. The cranial base is formed by the anterior, middle and posterior skull base; all these structures contribute to the craniofacial junction. They are connected to the orbits, nasal airways and paranasal sinuses and are thus affected by similar pathogenic processes.

Open craniofacial disassembly procedures historically accessed anterior/middle cranial base areas, but endoscopic skull base approaches are considered safe/efficient alternatives to open surgery for skull base issues [1, 2]. The posterior cranial base is unique, and the surgical treatment includes a combination of neurotologic and neurosurgical techniques. The surgical management of lesions affecting the anterior and middle cranial bases is the main topic of this chapter

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2 Surgical Anatomy

2.1 Anatomy of Cranial Base: An Endoscopic Perspective

2.1.1 The Sphenoid Sinus

It is essential to have an in-depth knowledge of the sphenoid sinus architecture to perform successful endonasal skull base surgeries endoscopically. The sphenoid sinus is a complex structure consisting of a number of anatomic components. It is important to recognize the various parts of the sphenoid sinus in order to properly assess the anatomy and perform the procedure safely and effectively. The sphenoid sinus is composed of the body, the anterior wall, the lateral wall, the posterior wall and the roof. A proper pre-operative assessment of the sphenoid sinus anatomy is critical in order to prevent potential complications associated with these approaches.

There may be a range in the sphenoid sinus level of pneumatization:

- Sellar type (the most prevalent): This type of pneumatization is adequate to expose the sellar floor to a varying degree.
- Conchal type: Almost no pneumatization.
- Presellar type: The pneumatization is in front of the tuberculum sellae plane.

N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_9

The sella turcica protrudes into the centre of the sinus. A clival depression between the ICA's paraclival portions results from pneumatization of the clivus beneath the sella [3].

The anterior loop (parasellar carotid) and carotid prominence on each side of the sella are formed by the paraclival carotids as they climb into the cavernous sinus. One to several septations of the sphenoid sinus can result in carotid prominences in around 80% of instances [4].

The pericanalicular section of the optic nerve and canal moves anterior-laterally toward the orbit above the carotid eminence. The depth of the lateral opticocarotid depression, which separates the optic and carotid prominence, indicates the pneumatization of the optic strut [3].

2.1.2 The Pituitary Gland and Suprasellar Space

The pituitary gland is made up of an anterior lobe (adenohypophysis) and a posterior lobe (neurohypophysis). The anterior lobe's precursor is the oral ectoderm. All other pituitary hormones are produced by the posterior lobe, which protrudes from the diencephalon, produces oxytocin and vasopressin and is paler in colour. The anterior lobe and pituitary stalk are supplied with blood via the superior hypophyseal arteries, which emerge from the inferomedial region of the supraclinoidal/paraclinoidal ICA [5–7].

Superior hypophyseal arteries promote the passage of hypothalamic hormone-releasing factors to the adenohypophysis via a portal venous system [5–7]. The inferior hypophyseal arteries, which are outgrowths of the meningohypophyseal carotid trunk, provide blood to the posterior gland [5, 6].

The inner meningeal and outer periosteal layers of the dura, which surround the pituitary gland, combine to create the sphenoidal wall of the cavernous sinus. The meningeal layer, which is still attached to the gland, forms the medial wall. The intercavernous veins that link the two levels allow for venous outflow from the gland to the cavernous sinus [5, 6].

2.1.3 The Anterior Skull Base

The bone in front of the limbus must be removed in order to have access to the frontal lobe. Access to the anterior skull base by expanding the surgical corridor to cover two mid-orbits laterally, the frontal sinus' anterior table anteriorly and the suprachiasmatic region posteriorly after a full ethmoidectomy and frontal sinusotomy. The posterior ethmoid artery is located near the junction of the planum sphenoidale and cribriform plate, while the anterior ethmoid artery crosses the ethmoid roof obliquely from posterolateral to anteromedial. It is essential to recognize these arteries and keep them away from the orbit to prevent a retrobulbar haemorrhage [8]. The foramen cecum is situated anterior to the crista at the frontoethmoidal junction, through which the anterior nasal emissary vein traverses. This could facilitate the growth of tumours and act as an anatomical marker.

2.1.4 The Cavernous Sinus

The cavernous sinus is split into venous compartments based on connection to the carotid when approached endonasally, as the cavernous carotid is met first when operating in the sinus.

The cavernous sinus has compartments that are:

- Superior compartment
- Posterior compartment
- Lateral compartment
- Inferior compartment

The superior compartment is between the roof of the cavernous sinus and the horizontal cavernous segment of the carotid. Superomedially, the interclinoidal ligament is present; posterolaterally, the dura of the oculomotor triangle is present, and the paraclinoidal carotid is anteromedially passing superiorly and posteriorly.

The inferior compartment is between the posterior genu and beneath the horizontal carotid and the anterior wall of the cavernous sinus.

The lateral compartment is present lateral to the horizontal cavernous carotid segment, which contains the abducens nerve, CNs III, IV, and V1. CN V2 borders the cavernous sinus floor.

2.1.5 The Clival and Petroclival Regions

The synostosis of the occipital and sphenoid bones forms the clivus, the middle portion of the skull base. It has a wedge-like form, and the petroclival fissure divides it from the petrous bone on each side. Any transclival endoscopic method requires knowledge of the clivus' anatomy [9]. The inferior petrosal sinus fits at the cranial end of the fissure's groove. Two dura layers—periosteal and meningeal—cover the upper surface of the clivus. The basilar venous plexus connects with the internal/external vertebral plexus inferiorly and with the cavernous sinus rostrally.

The dura in the lateral most region clivus is penetrated by the abducens nerve, creating Dorello's canal. Gruber's ligament, or the petrosphenoidal ligament, runs between the dorsum sellae and the petrous pyramid. Rarely, calcification may create a posterior sphenoidal/clinoid process [9]. During the endonasal approach, this ligament is behind the abducens nerve rather than above it.

The roof of the pharynx's posterior extension is formed by the pharyngeal tubercle, around 1 cm anterosuperior to the foramen magnum. It is flanked by the superior and inferior clival lines, which attach the rectus capitis anterior and longus capitis major. The nasopharyngeal fascia covers the muscle layer; the lower clivus, atlantooccipital joint, occipital condyle and ligaments become visible when the layers and anterior atlanto-occipital membrane are resected. The supracondylar groove is at the level of the pharyngeal tubercle and serves as a landmark to find the hypoglossal canal [10–12].

The ventral clivus subdivisions are based on Rhoton's first categorization of the posterior fossa using the 'rule of three' [13], the connections between the three clival regions and the three cerebellar arteries (anteroinferior, superior and posterior inferior), the related CNs (III, VI, XII), the midbrain, pons, medulla and the cerebellum, the clivus may be divided into three regions: upper, middle and lower (superior, middle and inferior cerebellar peduncle). The following is a presentation of the surgical anatomy of subdivisions of the clivus.

2.1.6 The Petroclival Region and Jugular Foramen

Transpterygoid infravidian approach followed by sublacerum or infrapetrous approach can be used to expand the lower transclival approach laterally to address tumours that extend laterally to the petroclival junction [14, 15]. In order to expose the vidian nerve, the pterygopalatine fossa must first be opened, and its contents must be moved laterally after the posterior wall of the maxillary sinus has been removed. In this procedure, the pterygopalatine and palatosphenoidal arteries must be sacrificed [16]. The palatosphenoidal artery marks the vidian nerve and enters the vidian canal. Drill posteriorly to the foramen lacerum, mobilize and resect the sublacerum and then drill the infrapetrous bone between the Eustachian tube and petrous carotid. Lateral corridor to the jugular foramen is created by transecting the Eustachian tube and drilling the carotid canal. Imaging (Doppler ultrasound) is needed to access the parapharyngeal ICA due to lack of bone markers.

2.2 Transcranial Perspective of the Cranial Base Anatomy

The extent and contents of the anterior, middle and posterior skull bases, which make up the cranial base, are described here.

2.2.1 Anterior Skull Base

Three different bones form the anterior cranial base:

- Frontal
- Sphenoid
- Ethmoid

The orbital process of frontal bone forms the anterior cranial fossa's floor from a lateral view,

while the ethmoid bone is the base of the anterior skull from a central view. The lateral edge of the cribriform plate is formed by the ethmoid bone's vertical lateral lamella. The frontal bone's fovea ethmoidalis, which shields the ethmoid sinus' roof, is attached through the lateral lamella to the cribriform plate [17].

The planum sphenoidale is formed by the sphenoid bone and serves as the roof of the sphenoid sinus. The minor wings of the sphenoid forms the anterior clinoid processes, which mark the ICA and optic nerves. The olfactory bulb and tract are above the planum, and the limbus of the sphenoid divides it from the prechiasmatic sulcus posteriorly [18].

The orbit and paranasal sinuses are very closely connected to the anterior skull base. The orbital apex has three major apertures:

- The superior orbital fissures
- The inferior orbital fissures
- The optic canal

The orbital apex is divided into lateral and medial compartments by the annulus of Zinn, which is formed by the orbital muscles having a common tendinous attachment.

The lateral superior fissure of the orbit transmits:

Nerves: The trochlear nerve
The frontal nerve
The lacrimal nerve
Vessel: The superior ophthalmic vein

Within the superior orbital fissure is the annulus of Zinn, which transmits the superior/inferior divisions of the oculomotor, abducens and nasociliary nerves. The optic canal is superomedial and contains the optic nerve/ophthalmic artery. The ocular artery originates just medial to the anterior clinoid process. Within the orbital apex, the ophthalmic artery gives its branches the supratrochlear/frontal arteries, plus the anterior/posterior ethmoidal arteries. In 7–13% of cases, the optic canal is entirely encircled by Onodi cells, which are posterior-most ethmoidal cells, so caution is needed during endonasal approaches to avoid damaging the optic nerves [19]. At the orbital apex, the superior ophthalmic vein connects with the inferior ophthalmic vein, draining into the cavernous sinus.

2.2.2 Middle Skull Base

The middle skull base is composed of the sphenoid bone's body, which houses the sphenoidal sinus. The ICA grooves to the sides of the sphenoid body, and the petrous ridge and sphenoid ridge restrict its lateral aspect. The floor is made up of the petrous apex of the temporal bone, squamous temporal bone and the sphenoid's greater wing. The medial and lateral pterygoid plates extend down from the pterygoid body and join the palatine bone anteriorly. The anterior clinoid process has two attachments to the sphenoid bone and creates the roof and floor of the optic canal. The prechiasmatic sulcus lies between the two optic canals and is bordered by the limbus of the sphenoid bone and the tuberculum sella. The sella turcica is in between the tuberculum sellae, dorsum sellae and posterior clinoids and the cavernous sinuses and carotids.

The diaphragma forms the roof of the sellar turcica, running from the tuberculum to the dorsum sellae. The pituitary stalk crosses the diaphragma's plane via the pituitary aperture. The pituitary fossa's contents are not often covered in cerebrospinal fluid (CSF) as the arachnoid does not typically extend into the aperture [20]. The diaphragma and cavernous sinus dura are connected laterally.

The middle cranial fossa has a significant number of foramina. It connects to the orbit and its contents via the superior orbital fissure. The maxillary division of the trigeminal nerve travels through the 4 mm foramen rotundum, which is under the fissure and separated by the maxillary strut of the sphenoid bone [21].

The foramen ovale transmits the lesser superficial petrosal nerve (LSPN), emissary veins, accessory meningeal artery and mandibular division of the trigeminal nerve. The foramen spinosum, which passes the middle meningeal artery, is posterolateral. The foramen lacerum, medially located, contains the ICA's lacerum section and petrous carotid canal in its superior

compartment and fibrocartilage in its inferior. The variable foramen of Vesalius, in the mandibular strut connecting the two foramina, sends an emissary vein to the cavernous sinus. The LSPN travels posterolateral to the foramen spinosum and the greater superficial petrosal nerve (GSPN), medially and parallel, merges with the deep petrosal nerve at the foramen lacerum and enters the vidian canal. The GSPN's posterior terminus is the geniculate ganglion of the facial nerve. The horizontal section of the petrous carotid, inferior and medial to this nerve, follows the GSPN's path [22].

CN V follows the petrous ridge from the posterior cranial fossa to Meckel's cave. The trigeminal impression and prominence, followed by the meatal impression, are found medially. Laterally, the tegmen tympani and tegmen mastoideum are thin roofs overlying the superior semicircular canal.

The cavernous sinus encircles the sella turcica and connects to the superior orbital fissure. The periosteal dura forms its anterior wall, the meningeal layer forms the medial wall and the posterior wall is formed by the periosteal layer between the petrous apex and dorsum sella [18]. Its roof is formed by oculomotor and clinoidal triangles, and the floor is where lateral and medial walls meet. The lateral wall is mainly periosteal dura, and the anterior petroclinoidal fold is where periosteal and meningeal layers combine. The maxillary division of trigeminal nerve and posterior Meckel's cave marks the sinus' inferior limit.

The cavernous sinus's drainage reaches the transverse and sigmoid sinuses into the superior and inferior petrosal sinuses, respectively. The anterior genu of the cavernous carotid continues as the paraclinoidal carotid, and it has a brief ascending section, posterior genu, horizontal segment and anterior genu.

The cavernous carotid has two main branches:

(1) The inferolateral trunk
(2) The meningohypophyseal trunk

The cavernous sinus contains the carotid sympathetic plexus and abducens nerve. The oculomotor triangle comprises three dural folds, and the oculomotor and trochlear nerves enter the sinus in its posterolateral corner. The supra- and infratrochlear triangles form the lateral wall of the sinus.

2.2.3 Posterior Skull Base

The petrous bone and clivus form the anterior wall of the posterior fossa. The occipital bone is the roof, and the tentorium cerebelli is the posterior and lateral limit. The floor stops at the foramen magnum.

There are two significant foramina on the petrous bone's posterior surface:

(1) The internal auditory canal, which connects the inner ear to the anteroinferior cerebellar artery and the seventh and eighth nerve complexes
(2) The internal auditory canal's lateral vestibular aqueduct, which transmits the endolymphatic duct

The petrous bone's jugular process connects with the occipital bone to form the jugular foramen. The pars venosa houses the jugular bulb and CNs X and XI, and the anteromedial compartment is the jugular foramen (the pars nervosa containing the glossopharyngeal nerve, inferior petrosal sinus and a posterior meningeal branch from the ascending pharyngeal artery). The hypoglossal canal, with the hypoglossal nerve, is inferior and medial to the jugular foramen. The vertebral arteries ventrally exit the CNs as the posterior inferior cerebellar arteries, and the basilar artery emits the anteroinferior cerebellar artery (AICA) and superior cerebellar arteries. The mastoid tip and suboccipital musculature shield the posterior skull base, with the digastric muscle originating medial to the mastoid groove and the sternocleidomastoid muscle originating from the mastoid tip. The suboccipital triangle contains the vertebral artery and its venous plexus. The transverse foramen, vertebral artery and C1 lateral process (attachment point for oblique muscles) are located near the C1 arch, allowing ventral access to the foramen magnum, lower clivus and craniocervical junction.

2.3 Trans Nasal Transsphenoidal Approach

Diagnosis of pituitary adenomas is now more common thanks to advanced hormonal tests and MRI with gadolinium enhancement. Oscar Hirsch and Harvey Cushing's work in the early 1900s laid the foundation for modern pituitary surgery. Endoscopic transnasal sphenoidotomy, without septal dissection, is a successful method that shortens hospital stays and reduces postoperative discomfort [23–26]. This method has replaced other approaches for sellar lesions [27].

Endonasal transsphenoidal endoscopy can be done via deep-transseptal, paraseptal and middle turbinectomy or middle meatal route. One or two nostrils may be used [28, 29].

Transnasal techniques have been used to access the sella turcica since pituitary surgery began. All strategies are transsphenoidal, crossing the sphenoid sinus [30, 31].

2.3.1 Operative Technique and Caveats

- The patient is made to lie down on their back with their neck slightly stretched and turned toward the nostril through which the endoscope will be passed, with the head end of the bed raised.
- A 4 mm endoscope is employed, depending on the patient's nasal passageway's preoperative evaluation.
- Behind the patient's shoulder and right across from the surgeon's field of vision is the video monitor.
- The 0° endoscope guides the intranasal dissection.
- In the beginning, the middle turbinate and its axilla are recognized.
- The middle turbinate is delicately lateralized and separated from its higher connection to the skull base.
- This gives enough room for instruments and exposure for the sphenoid ostia to be identified.
- Posterior septectomy is done to increase accessibility and create enough room for equipment.
- Sella turcica is located, the mucosa covering it is removed and the sella bone is removed (with a sickle knife/periosteal elevator/drill). Sphenoid ostia are located, and sphenoidotomy is performed (inferomedially).
- A cruciate incision is made while cauterizing the exposed dura mater. Tumour resection is performed utilizing a suction device and ring curettes of various sizes and orientations after tissue is removed for histology.
- A technique using a single or double nostril is used for all procedures. The 0° endoscope is removed, and a 30° endoscope is then placed, depending on whether the tumour has been completely removed or whether any remaining tumour is outside the area of vision.
- This endoscope's tilted lens offers superior exposure to the suprasellar and parasellar areas.
- It is possible to see the expansion of suprasellar and parasellar tumours, as well as any possible cavernous sinus invasion, by rotating the 30° endoscope in both the clockwise and counterclockwise directions.
- Any remaining tumour is removed by resection, removing any possible recurrence sites. After the tumour has been completely removed, the region is irrigated to achieve haemostasis.
- In order to repair the sellar defect and seal it with Surgicel and AbGel, the fat is extracted, while the fascia lata is being removed.
- Merocel is used for nasal packing, which is removed after 72–48 h.

Case 1

A 49-year-old male patient presented with headache and bilateral blurring of vision for 1 month.

Pre-operative imaging, use of navigation system and surgical steps of the same patient are depicted in Figs. 1, 2, 3, 4, 5, 6, 7, and 8.

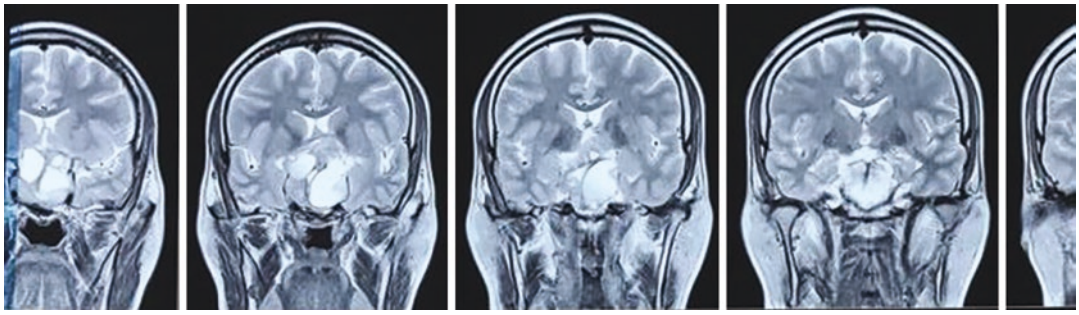

Fig. 1 MRI brain coronal section showing a pituitary tumour

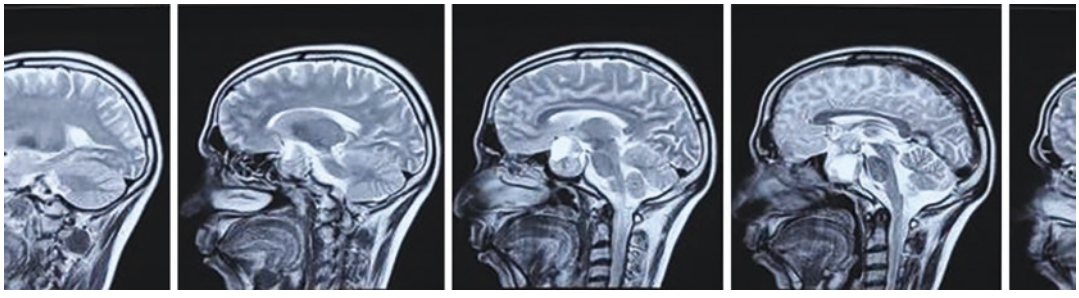

Fig. 2 MRI brain sagittal section showing a pituitary tumour with lesion extension into the suprasellar region

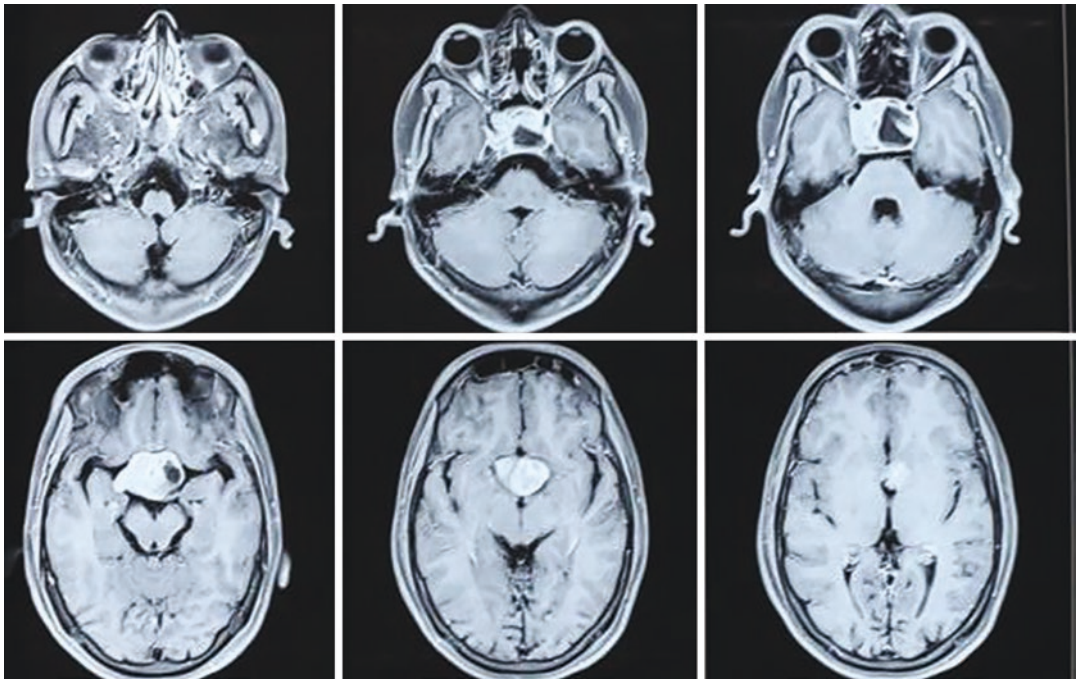

Fig. 3 MRI brain axial section showing a pituitary tumour

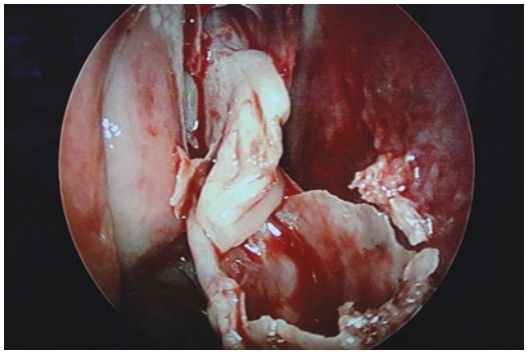

Fig. 4 Endoscopic view of the right nasal cavity: superior attachment of the middle turbinate is cut and is lateralized to create adequate space for instrumentation and visualization of the sphenoid ostium

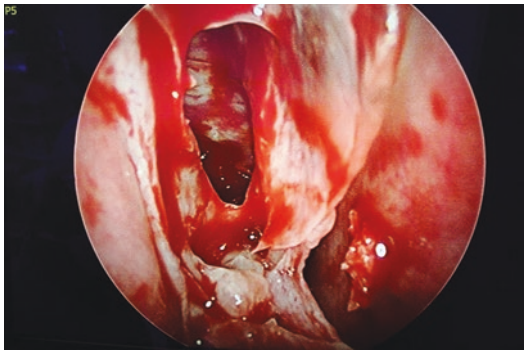

Fig. 5 Sphenoid ostium is visualized and widened

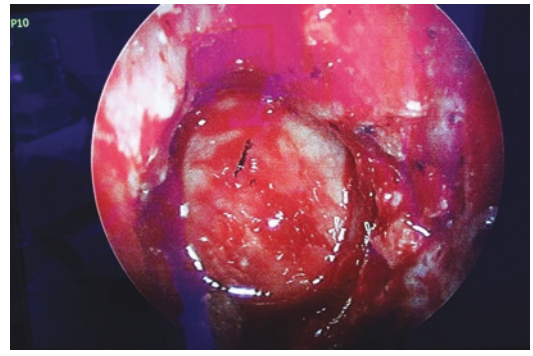

Fig. 7 Sella turcica bone is removed and the dura mater is exposed

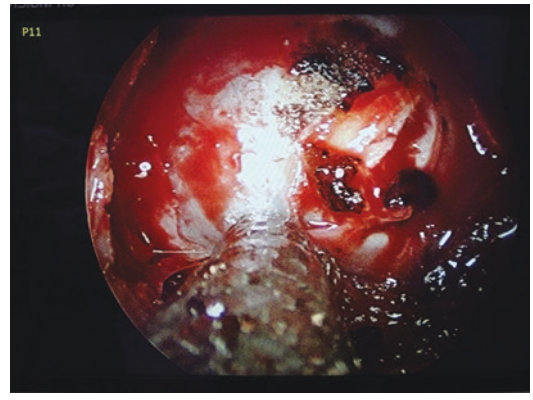

Fig. 8 Surgical site after closure using a tissue glue

Fig. 6 Using the navigation system to know the precise location in coronal, sagittal and axial planes in real time

2.4 Endoscopic Sellar Defect Reconstruction by Modified Gasket Seal Technique

The transsphenoidal method emerged via an evolutionary process as opposed to a revolutionary one. The development of transsphenoidal surgery is a convoluted tale of revolutionary leaps in thought, intensive surgical experimentation and times of outright rejection of the procedure. Skull base tumours may now be surgically removed with a minimum of trauma and with great success thanks to endonasal methods.

The transsphenoidal technique for treating pituitary tumours is where these strategies are most commonly documented [32]. Despite the alleged advantages of endonasal methods, postoperative cerebrospinal fluid (CSF) rhinorrhea is still a common problem that may lead to serious consequences such as meningitis, pneumocephalus and the need for further surgery [33]. Iatrogenic intraoperative rupture of the barrier separating the subarachnoid space from the sinonasal cavity causes CSF leak. In most pituitary adenoma resections, this disruption could be inevitable or unintentional [34]. A watertight repair of the skull base is essential for avoiding postoperative CSF leak, regardless of the reason. For the repair of sellar defects, several local vascularized pedicle and avascular grafts have been reported throughout the years [34–36]. The 'gasket-seal' approach combining a stiff buttress and a fascial graft is often used for dura repair. Both of these procedures utilize autologous fascia lata, which has properties comparable to dura [36–38]. According to reports, the most popular and successful graft material for multilayer repair is autologous fascia lata [39]. Postoperative CSF leak rates after endoscopic TSS treatments vary from 0.5% to 15%, despite the use of contemporary reconstructive methods. Using lengthy, stiff tools in a small working area to position repair materials against the pulls of gravity and overlying dependent intracranial structures is difficult when reconstructing the base of the skull.

Various methods for reconstruction of the skull base based on the defect, technique and material used are shown in Table 1.

2.4.1 Operative Technique and Caveats

Following surgical removal of the sellar tumour, Kelly and colleagues' grading method was used to determine if there was an intraoperative CSF leak [40].

Intraoperative CSF leaks were classified as:

Grade 0—no observed leak
Grade 1—small leak without obvious diaphragmatic defect
Grade 2—moderate leak
Grade 3—large diaphragmatic/dural defect

Barrier-restoring and pressure-diverting methods made up the bulk of the defect restoration. The repair of the dural barrier is as follows:

Table 1 Various methods for reconstruction of the skull base based on the defect, technique and material used

Reconstruction	Barrier restoring	Defect	Technique	Material
		Intradural (inlay)	Layering	Autologous fat
			Buttoning	Fascia lata
		Dural (overlay)	Layering	
			Modified gasket seal	Knitted collagen + fibrin glue
		Bony (skull base)	Layering	Fascia lata + tissue glue
		Barrier		Knitted collagen + absorbable gelatin
		Nasal construct stabilization	Temporary	Sterile nasal sponge (Merocel)
	Pressure diversion	Long term	Shunting procedure	
		Short term	Lumbar drain	

- Filling the sellar cavity with the autologous fat that was also extracted with the fascia lata allowed for intradural (inlay) repair to be completed. The fat was arranged to prevent compression or back pressure on the neurovascular structure.
- The fascia lata is draped over the fat tissue, the fascia is tucked beneath the bone sellae and the dura extends beyond its boundaries in a dural (overlay) repair.
- The fascia lata layer is strengthened with a knitted collagen and fibrin glue to guarantee a watertight closure; this creates a strong barrier that serves as a gasket to seal the dural defect. The initial gasket sealing method was discussed in detail by Weill Cornell University colleagues [37]. Using a bone fragment with a collagen matrix glued with a tissue glue over a vascular pedicled flap overlay, our variation makes use of avascular tissue (modified gasket seal).
- By adding additional layers of the fascia lata and fibrin glue to the bony skull base layer, a thick framework holding the earlier build in place is created.
- The interwoven collagen and absorbable gelatin pieces act as a final barrier.
- A nasal sponge offers temporary stability of the nasal architecture.

In cases of grade 2 and 3 intraoperative CSF leak, the pressure diversion treatments, such as shunting or lumbar drain insertion, were carried out. On the third postoperative day, the nasal pack will be removed under aseptic conditions. Patients are given intravenous antibiotics for surgical infection prevention after surgery and are monitored in the neurosurgical intensive care unit (NICU); if a lumbar drain has been implanted, antibiotics continue while the drain is in place. Patients may be given 2–3 days of bed rest with the head of the bed at 30–45° in instances of CSF leak necessitating the use of postoperative lumbar drainage. During the duration of the hospitalization, sodium and cortisol levels were tested each morning and immediately after surgery to check for changes in sodium and cortisol levels, as well as for evidence of meningeal irritation and eyesight improvement. To check for diabetes insipidus, the patient's salt and hydration status were closely tracked. Within 24 h of surgery, a postoperative computed tomography was performed to assess the pneumocephalus, completeness of resection and hematoma.

Intraoperative surgical steps and follow-up endoscopic images are depicted in Figs. 9, 10, 11, 12, 13, and 14.

2.5 Fronto-Facial Resection

Opportunistic illnesses like mucormycosis were seen as localized outbreaks during the COVID-19 pandemic. Sadly, India has had an abnormally high incidence of mucormycosis patients after

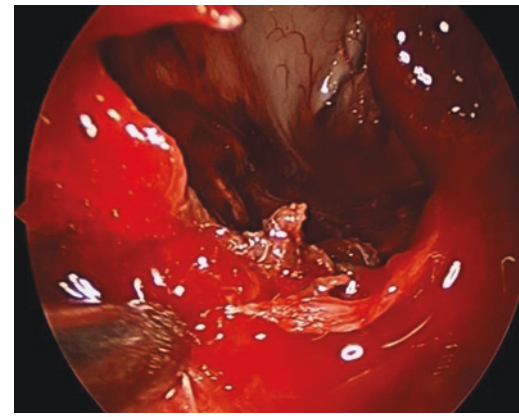

Fig. 9 Sellar defect

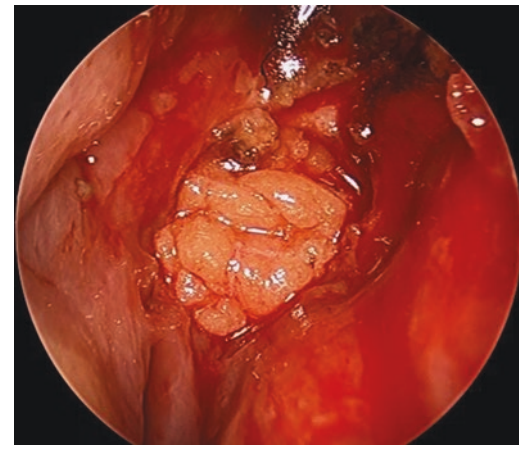

Fig. 10 Intradural fat inlay

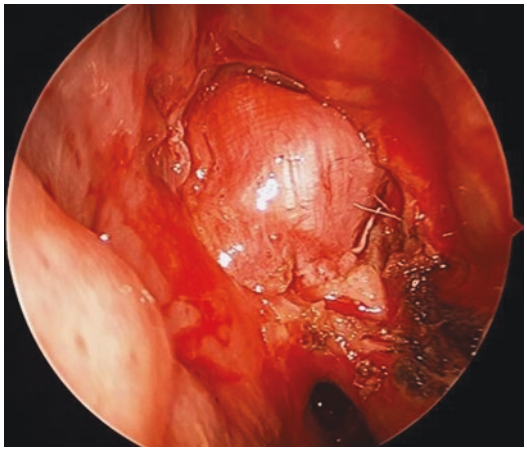

Fig. 11 Layering with the fascia lata

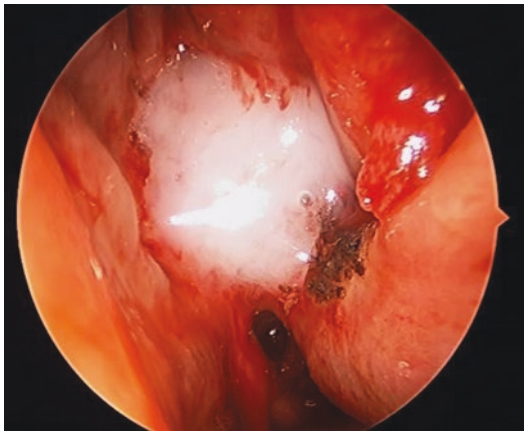

Fig. 12 Layered with knitted collagen and fibrin glue

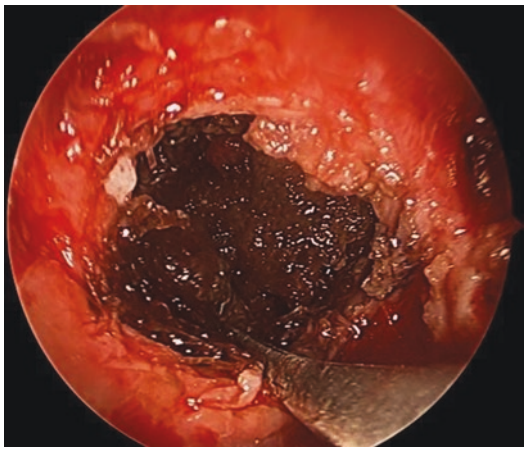

Fig. 13 Modified gasket seal technique

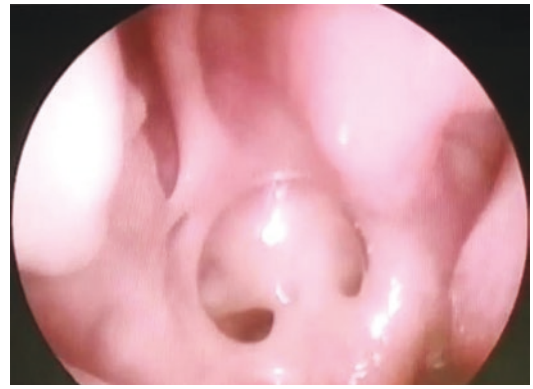

Fig. 14 Follow-up after 6 months with well-mucosalized surgical site with no CSF leak

contracting COIVD-19, particularly during the second wave of the epidemic (March 2021) [41].

Through the use of interdisciplinary hospital teams, surgical debridement and systemic amphotericin B were successful treatments for mucormycosis [42]. A return of the illness, manifested as fungal osteomyelitis of the frontal bone, has been seen after the first therapy for mucormycosis, however. There is no known history of this kind of mucormycosis. This illness' return is attributable to complications from prior surgery, insufficient amphotericin dosage, comorbidities, immunological state and the severity of the disease before the operation [42, 43].

2.5.1 Operative Technique and Caveats

- Due to the recurring nature of the illness and for sufficient disease clearance, open surgical debridement is suggested.
- Bifrontal craniotomy, total removal of the frontal bone, pericranial flap and contralateral supraorbital craniotomy
- Due to bony sequestrum and involucrum along the frontal bone, frank pus became visualized after exteriorizing the frontal sinus.
- Debridement included removing all the sequestrous bone as well as a portion of the involucrum until normal bone could be found.
- Cerebrospinal fluid (CSF) leaks during surgery may be fixed using an inlay pericranial flap as the main closure.

- The likelihood of seeding in the graft and a foreign body response prevents reconstruction using free flaps and alloplastic materials.

Pre-operative photos, radiological imaging, intraoperative photos, postoperative radiological imaging and follow-up imaging are depicted in Figs. 15, 16, 17, 18 and 19.

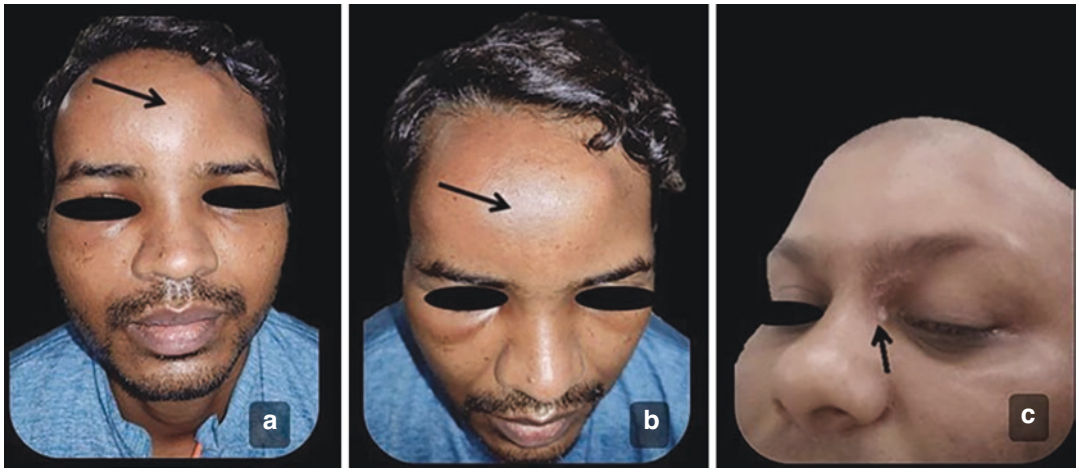

Fig. 15 In panels (**a** and **b**), frontal bossing is present, which resembles a Pott's puffy tumour. The patient in panel (**c**) has a discharging sinus next to the medial canthus

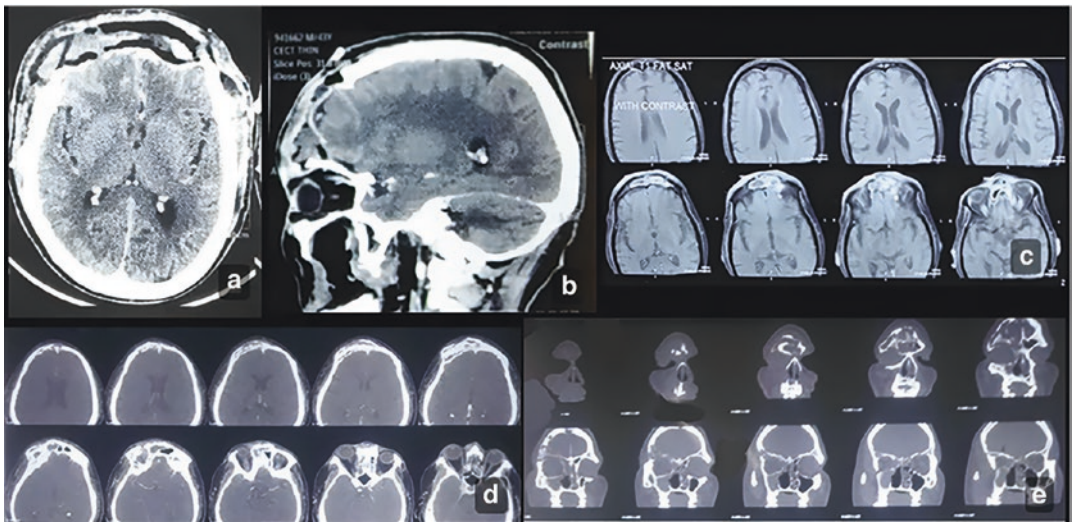

Fig. 16 Imaging before surgery. Axial and sagittal contrast-enhanced CT (CECT) images are shown in panels (**a** and **b**). They demonstrate full erosion of the frontal bone and necrotic soft tissue debris between the skin and dura, with the dura seeming enhanced. Panel (**c**) displays axial T2-weighted MR. Panel (**d** and **e**) are axial and coronal CECT suggestive of frontal sinus erosion with intracranial extension

Fig. 17 In the frontal sinus posterior table, shown in panel (**a**), are the sequestrum and involucrum, with an arrow indicating in their direction. In panel (**b**), the necrotic tissue is removed till the normal bone is reached. Iodoform ribbon gauze is put in the frontal sinus in panel (**c**)

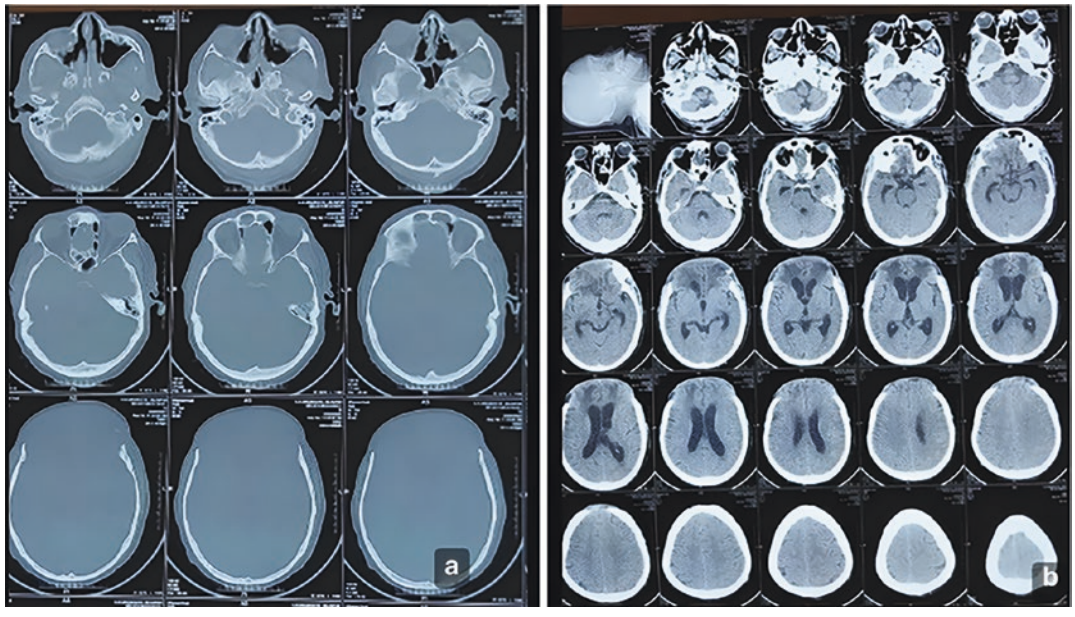

Fig. 18 Ninth postoperative month CT scan. Debrided frontal bone is shown in axial bony window cuts in panel (**a**). Panel (**b**) indicates locoregional disease management since there is no aberrant soft tissue density in the surgical location

Fig. 19 A follow-up image of the patient (panels **a** and **b**) reveals a substantial frontal hollow as a result of thorough debridement. There are no remaining neurological impairments in the patient.

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Larynx and Airway

Ambesh Singh, Prajwal S Dange, and Rupa Mehta

1 Introduction

The larynx coordinates a vital role in the various functions of the upper aerodigestive tract, including speech, swallowing and respiration. Squamous cell carcinoma in the head and neck frequently develops in the larynx due to repeated exposure to carcinogens like alcohol and tobacco.

The larynx is mainly divided into:

- The supraglottic region
- The glottic region
- The subglottic region

These embryologic-based anatomical divides have significant clinical ramifications. Compared to the sparse lymphatic network in the submucosal plane of the genuine vocal cords, the lymphatic drainage of the supraglottic larynx is extremely rich. Laryngeal cancer's patterns of regional spread are thus influenced by the main tumour's local size and place of genesis. The larynx is separated into three different areas or sites. The laryngeal surface of the epiglottis, the aryepiglottic folds, the arytenoids, the ventricular bands or false vocal cords and the ventricles—possible areas between the false and real vocal cords—are the locations in the supraglottic region. The right and left vocal cords, together with the anterior commissure, are the three specified places in the glottic larynx. The right and left lateral walls of the subglottic area, which is commonly regarded as one site, serve as its boundaries.

More than 95% of the primary malignant tumours of the larynx are squamous cell carcinomas; the remaining tumours come from the small salivary glands, neuroepithelial tumours, soft tissue tumours and, in rare cases, the cartilaginous laryngeal framework. The majority of initial malignant tumours in the larynx are found in the glottic area. In contrast to almost 70% of patients with supraglottic carcinoma, who present with advanced disease, nearly 75% of patients with glottic carcinoma had localised disease at the time of diagnosis.

Laryngeal cancer is the tenth most prevalent cancer in India overall, and head and neck cancer is the second most common malignancy after cancers of the lips and oral cavity, according to the GLOBOCAN 2020 statistics. In India, laryngeal cancer diagnoses were discovered in 34,687 instances in the year 2020 [1].

2 Aetiology and Pathology

2.1 Smoking

- Despite alcohol being an independent risk factor for developing laryngeal cancer in the

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_10

absence of smoking, smoking remains the most frequent aetiological cause for malignancies of the larynx.
- Alcohol and smoking seem to increase the risk in different ways [2].
- Smokers are most at risk of getting laryngeal cancer, and that risk rises with cigarette consumption.
- After quitting smoking and drinking, there is a relative risk decrease for laryngeal cancer, with the risk reduction reaching the same level as never smokers after around 20 years [2, 3].

2.2 HPV Infection

- Although they are strongly associated with oropharyngeal tumours, there is less epidemiological evidence connecting them to laryngeal tumours [4, 5].
- Existing retrospective studies have not shown a favourable prognostic factor for HPV-positive laryngeal tumours, in contrast to oropharyngeal cancer [6, 7].

2.3 Other Factors

- Occupational toxicants including asbestos, polycyclic aromatic hydrocarbons, wood dust, coal dust and cement dust are other recognised risk factors [8, 9].
- The chance of having laryngeal cancer is increased for people with lower socioeconomic level [8].
- Certain genetic syndromes, such as congenital dyskeratosis and [10] Fanconi anaemia [11], are also associated with a higher risk of developing laryngeal tumours in addition to tumours in other parts of the body.

The most common type of laryngeal cancer is squamous cell carcinoma; however, many other histological types of cancers can be seen in the larynx. Optimal pathological assessment of specimens requires a coordinated approach between the pathologist and surgeon or oncologist. The Royal College of Pathologists in the UK has published guidelines for the dataset required to report cases. Important factors include the type, size and grade of the primary carcinoma, the pattern of invasion and the proximity of the carcinoma to resection margins. All are important prognostic features [12]. In cases where a total laryngectomy has been performed, whole-organ section analysis is usually performed to consider patterns of spread and resection margins. HPV testing is not considered routine but may be important in a subset of patients.

3 Pathway of Cancer Spread

Depending on where it originates, laryngeal squamous cell carcinoma spreads in several ways. The lymphatic spread is initially controlled by the borders between embryological anlagen (arches III–VI), particularly at the free edge of the glottis, although local spread occurs along tissue planes. But contrary to long-held ideas, Kirchner's painstaking whole-organ dissections from 1969 showed that there is no real physical barrier to restrain the spread between the supra- and subglottis [13].

3.1 Anterior Commissure

Because early invasion through Broyles' ligament straight into the cartilage was believed to be unavoidable, it was formerly common knowledge that glottic cancer might extend to the anterior commissure (AC). This was thought to be caused by the absence of an inner perichondrium there. Recent research has revealed that although the ligament serves as a useful barrier, the prognosis is worsened by expansion superiorly and inferiorly (through the subglottic wedge and, consequently, the cricothyroid membrane). Direct spread may extend posteriorly to infect the arytenoid cartilage or may proceed via the AC to the opposing cord.

3.2 Glottic Tumours

The embryological barrier, located superiorly, and the conus elasticus, located inferiorly, may constrict glottic tumours to Reinke's space. Tumours must cross both the vocal ligament and the vocalis muscle to enter the paraglottic region

and then easily migrate cranially and caudally. Ossified regions appear to be less resistant to invasion than the rest of the laryngeal skeleton. However, direct external extension through the cartilage is frequently observed. Glottic cancer lymphatic dissemination is less frequent than at other subsites. It has been proposed that this is caused by the absence of submucosal lymphatics. Mucosal cell transport has not yet been thoroughly studied to support this, though. When the spread happens, it affects levels II, III, IV and VI. According to estimates, the incidence of macroscopic lymph node metastasis varies depending on the stage of the disease: 5% (T1), 7% (T2), 14% (T3) and 33% (T4) (T4). The so-called "Delphian" node, sometimes referred to as the midline anterior metastatic node or Poirier's prelaryngeal ganglia node, is extremely uncommon but is believed to be connected to T3 or T4 tumours with notable subglottic extension.

3.3 Supraglottic Tumours

Even with pre-epiglottic or nodal dissemination, supraglottic malignancies typically don't spread beyond their local subsite until rather late. As a result, even for large tumours, a supraglottic laryngectomy is frequently a good option. There isn't really an anatomical barrier to inferior spread, though, as was already mentioned. Indeed, the accurate selection of patients who are candidates for supraglottic partial laryngectomy is made possible by the routine use of angled Hopkins rods during staging procedures to examine the cavity of the laryngeal ventricle. This method reveals a steady transmucosal progression of tumours similar to those found in other subsites. According to a number of studies, over 50% of supraglottic malignancies move to the glottic area.

The epiglottic cartilage is less of a barrier to spread than the other cartilages since it has foramina inside it. However, since hyoid invasion is rather uncommon (2–4%), it is frequently possible to keep it during surgical clearing. "Suprahyoid supraglottic" carcinomas often expand mucosally into the pyriform fossae rather than the paraglottic space, invading the pre-epiglottic region and the deep muscles of the tongue.

Additionally, supraglottic tumours are more likely to develop bilateral nodal metastases. Compared to significantly lower rates of 20% in advanced glottic cases, supraglottic carcinoma had a positive nodal rate of over 60% in reports of advanced illness.

3.4 Subglottic Tumours

Caudally and circumferentially extending subglottic cancer is common. By the time a condition is diagnosed, 50% of cases have already invaded the cricoid, and 75% have spread outside. Although a microscopic incidence of one in three places this illness into the category of necessitating elective nodal dissection, clinically apparent nodal metastases is surprisingly rare. The paratracheal/mediastinal nodes should be included in the nodal dissection for subglottic carcinoma due to the tendency for inferior extension.

Transglottic carcinoma is identified by its progression across all three laryngeal subsites, both superficially and into the paraglottic region. Although it is hard to pinpoint the exact origin of true primary transglottic cancer in practice, it is considered to start in the laryngeal ventricle. By definition, it is at least T3 upon presentation; therefore advanced malignancies will be taken into consideration moving forward.

Thyroid gland invasion has been explored in resected tissues, and Kumar et al. studied the incidence of this in a systematic review [14]. This study's analysis of data from 1287 individuals revealed that the overall incidence of thyroid gland invasion is 10.7% [95% confidence interval (CI) 7.6–14.2]. The relative risk of thyroid gland invasion is considerably greater in patients with initial subglottic tumours [relative risk 7.5; 95% confidence interval (CI) 4.3–13.0] and disease extension into the subglottis (relative risk 4.3; 95% CI 2.5–7.2). Other locations did not present this increased danger. Since thyroid gland invasion is uncommon overall, thyroidectomy can be deferred for instances that are thought to be at risk rather than being a standard procedure for all total laryngectomies.

4 Clinical Presentation

Laryngeal cancer, like other malignancies, can exhibit local symptoms as well as those brought on by metastatic dissemination to nodes or elsewhere. Rarely many individuals with laryngeal cancer exhibit general systemic symptoms in the absence of local symptoms, such as anaemia or weight loss. As a consequence, primary care practitioners (and the general public) can have a pretty accurate understanding of the "warning/red flag" symptoms that call for an immediate referral to an otolaryngologist.

4.1 Glottic Cancer

It is fortunate that even the early glottic malignancy modifies the voice by changing the way the vocal cord's wave pattern behaves. Even cancer in situ may cause a considerable alteration in voice since regular voice production depends on the health of a six-cell thick epithelium and a fragile, jelly-like superficial lamina propria.

Therefore, anyone whose hoarseness has persisted for 3 weeks or longer—some even shorter time—should be referred immediately for a checkup with an otolaryngologist and head and neck surgical oncologist.

Maximum phonation time decreases with increasing lesion size, and breathiness with varying degrees of aspiration may be added with the commencement of cord fixation. Progressive dyspnoea and stridor may result from airway obstruction caused by advanced lesions. Larger tumours are typically linked to haemoptysis. Referred otalgia is a dangerous indication that suggests a profound invasion and is detected by the vagal complex. In simple glottic malignancy, dysphagia and odynophagia are quite uncommon. Rarely are neck nodes the initial complaint; when they are, they indicate a severe invasion and extension into the supraglottis.

4.2 Supraglottic Cancer

The quality of the altered voice is distinct from that associated with glottic and subglottic malignancy. Globus or foreign body feeling and paresthesia may be present in small supraglottic lesions that do not extend to the glottis. They may result in haemoptysis if exophytic. Phonation changes as tumour size grows, taking on a "hot potato" sound. As with glottic illness, hoarseness results if tumours spread to the cords.

Lateral extension that is too far may result in genuine dysphagia, referred otalgia and odynophagia. Although tumours may not cause symptoms until they are fairly big, it is normal for cervical nodal metastases to cause a neck lump to be the first symptom to develop.

4.3 Subglottic Cancer

Early symptoms might also be hazy and include a sense of a "globus" or foreign mass in the throat. Hoarseness comes from any glottis or recurrent laryngeal nerve involvement. Diplophonia may occur with paralysis and has a shorter maximum phonation time. Therefore, especially in high-risk situations, this diagnosis should always be taken into account while evaluating the potential alternative causes of "idiopathic" cord paralysis. Progressive dyspnoea and stridor are brought on by circumferential progression, which also causes voice fatigue quickly. A thyroid isthmus lesion may be masked by direct extension into the thyroid.

5 Assessment

5.1 Outpatient Setting

The flexible nasal laryngoscope is the ideal tool for evaluating the larynx in an outpatient situation. Endoscopy can provide significant information that can help with decision-making. If more research is needed, the outpatient endoscopic assessment might also point the examiner in that direction. For instance, the discovery of salivary pooling in the pyriform sinus in the presence of a tiny glottic tumour should raise suspicion and necessitate a checkup of the upper oesophagus and hypopharynx. Laryngeal dysplasia (LD) is a condition that often affects the glottis but can occur elsewhere in the larynx. The larynx has an

erythematous, inflammatory look with leukoplakia or erythroleukoplakia. However, histological signs of LD are not necessarily present in the larynx's clinically aberrant regions, and LD can be seen under a microscope in the epithelium that seems clinically normal. Cancer can manifest as lesions that proliferate, infiltrate or both. These are quickly identified during an endoscopic examination as abnormalities. Malignancies, often non-squamous cancers, can occasionally manifest as submucosal tumours. These lesions might create minor abnormalities that may go unnoticed upon initial observation.

5.2 Imaging

Cross-sectional imaging for all tumour stages should be included as a minimum in imaging for laryngeal mass lesions. Magnetic resonance imaging (MRI) or computed tomography (CT) are two options, with the chest also being scanned. Comparing cartilage invasion, MRI scans are more sensitive than CT scans. Each imaging technique has benefits and disadvantages of its own.

5.3 Assessment Under General Anaesthesia

This should be carried out by the operating surgeon and assessment regarding the feasibility of transoral excision to be done in the same sitting. Complete laryngoscopic examination should be carried out to check for arytenoid fixity, subglottic extension, and to map the extent of the tumor.

6 Management of Laryngeal Cancer

6.1 Surgical Anatomy

Anatomically, the larynx extends from the bottom edge of the cricoid cartilage to the tip of the epiglottis. It has a continuous lower boundary with the cervical trachea. Its anterosuperior border is the base of the tongue, and its posterolateral border is the hypopharynx and cervical oesophagus.

Laryngeal cartilages consist of:

- Thyroid
- Cricoid
- Arytenoid
- Epiglottis
- Corniculate
- Cuneiform cartilages

The height of the anterior midline of the thyroid cartilage varies depending on where the anterior commissure is located. The anterior commissure is often found below the midway in men and at or above the midpoint in women of the anterior midline of the thyroid cartilage. Despite not being a part of the laryngeal structure, the hyoid bone, which is connected to the thyroid cartilage by the thyrohyoid membrane, is crucial to the upper aerodigestive tract's functionality. The laryngeal cartilages can calcify in different ways, which might make it difficult to diagnose tumour invasion from radiographic images.

The larynx's surface mucosa is made up of squamous epithelium with scattered mucous glands. Stratified squamous epithelium lines the vocal cords. The internal branch of the superior laryngeal nerve, which enters the larynx through the thyrohyoid membrane, provides the sensory nerve supply to the supraglottic larynx. The superior laryngeal and recurrent laryngeal nerves provide dual sensory nerve supply to the mucosa of the true vocal cords, whereas the subglottic larynx receives sensory nerve input from the recurrent laryngeal nerve. Except for the cricothyroid muscle, which is innervated by the external laryngeal branch of the superior laryngeal nerve, the intrinsic musculature of the larynx receives its innervation from the recurrent laryngeal nerve. The superior and inferior thyroid arteries' branches feed the larynx with blood. The superior and recur-

rent laryngeal nerves also enter the larynx via these arteries. A robust lymphatic network drains from the supraglottic larynx into first-echelon lymph nodes at levels II and III via the thyrohyoid membrane. The glottic larynx has a relatively scant lymphatic network, notably around the free border of the true vocal cord, which is lymphatic-free. The cricothyroid membrane is the exit point for the lymphatic drainage of the subglottic larynx, which drains into the paratracheal and deep jugular lymph nodes. The larynx also drains the lymph to the parathyroid and Delphian lymph nodes.

6.2 Operative Techniques and Caveats

For patients with:

(1) Advanced laryngeal or hypopharyngeal cancers (T4a) that have invaded thyroid cartilage and extra laryngeal soft tissues.
(2) Tumours that have not responded to the larynx preservation treatment programme of radiotherapy or chemotherapy/radiotherapy.
(3) Extensive tumours of minor salivary origin and other histologic entities not suitable for a partial laryngectomy: a total laryngectomy is indicated as the initial definitive treatment.

Wide-field total laryngectomy is the preferred procedure if a total laryngectomy is being considered for primary laryngeal cancer.

This process includes:

- The lymph nodes in the jugular chain (levels II, III and IV) on the ipsilateral side as well as the lymph nodes in the tracheoesophageal groove on the same side. The whole larynx with its connected prelaryngeal strap muscles.
- To ensure that the ipsilateral tracheoesophageal groove lymph nodes are sufficiently cleared, an ipsilateral thyroid lobectomy should be done for lesions that include the glottic larynx and have extensive subglottic extension.
- Bilateral jugular node dissection (levels II, III and IV) should be carried out if the laryngeal lesion necessitating complete laryngectomy extends on both sides of the midline. If palpable metastatic nodes are present, a modified radical neck dissection with accessory nerve preservation should be carried out.

The operative procedure is described as follows:

- An orotracheal tube is used to administer general endotracheal anaesthesia when the patient is placed on the operating table. The planned permanent tracheostome is a 2.5-cm-diameter circular patch of the skin in the suprasternal notch. If tracheotomy was done before laryngectomy, the stoma will be incorporated in the incision.
- To expose the prelaryngeal strap muscles, the skin incision is deepened into the platysma.
- Elevate the upper neck flap cephalad to expose hyoid bone and suprahyoid muscles. Split and ligate anterior jugular veins. Detach muscles at the superior end of the hyoid to mobilise larynx. Secure the hyoid with Adair clamp, and separate the suprahyoid muscles with electrocautery up to greater cornua's tip.
- The lingual artery is at risk of harm if not exercised with care during operation.
- Note the superior thyroid artery's superior laryngeal branch, which must be identified and handled with care to avoid injury during dissection. Early detection of the superior laryngeal artery reduces haemorrhage during larynx mobility. When thyroid lobectomy and laryngectomy are planned, the superior thyroid artery is split and ligated. The distal stump of the superior thyroid artery is then withdrawn with the specimen and secured with a haemostat.
- Retract the sternomastoid muscle to view the carotid sheath near bulb. Levels II, III and IV deep jugular lymph nodes are directed towards the specimen.
- Split the superior belly of the omohyoid and the sternohyoid/sternothyroid muscles low in the neck, retracting the sternomastoid muscle laterally. Upon division, their stumps will retract cephalad.
- An Adair clamp is used to hold the hyoid bone while gently pulling the larynx towards the chin.

- Split and ligate inferior thyroid artery.
- Detach the isthmus from the trachea with blunt dissection.
- Divide the isthmus with two Kocher clamps.
- Ligate the left thyroid lobe stump with continuous interlocking 3-0 chromic catgut suture.
- The cervical trachea is visible by dividing the thyroid gland's isthmus and the left strap muscles up to the thyrohyoid membrane.
- Preserve superior thyroid arteries when dividing the left lobe from the trachea.
- The left side's superior laryngeal nerves and arteries are separated.
- Electrocautery used to split the inferior constrictor muscle from the thyroid cartilage's posterior margin.
- A knife is used to make the skin incision, and electrocautery separates tissues and haemostasis of the skin's edge.
- An incision is made at the necessary level in the tracheal wall; the trachea is split into long posterior and short anterior wall and the tracheostome has larger circumference due to the trachea's bevel-shaped stump.
- Nylon sutures attach from the distal trachea to tracheostome skin margins, pulling the trachea's stump up from the larynx.
- Direct endotracheal tubes now used to maintain anaesthesia, replacing former orotracheal tubes via tracheostome. The tracheal stump's bevel-shaped creates an oval form for the tracheostome.
- Separate the trachea and larynx from the oesophagus by sharp dissection, apply cephalad tension to the tracheal stump, use electrocautery to reduce blood loss and mobilise the larynx cephalad in the postcricoid area.
- Electrocautery can separate the larynx and oesophageal mucosa with mild traction to the tracheal stump. Entry into the pharynx is achieved through the vallecula when the tumour is endolaryngeal or postcricoid area when the tumour is supraglottic and involves the aryepiglottic fold/epiglottis.
- Make an incision in the mucosa around the larynx to create an opening wide enough for a retractor. Remove the larynx by cutting its mucosal attachments.
- Evaluate the mucosal margins histologically if near the main tumour to rule out a microscopic spread.
- At this point, a nasogastric feeding tube is placed.
- If tracheoesophageal puncture (TEP) is needed, the plane posterior to the tracheostome is dissected. A right-angled haemostat is inserted into the cervical oesophagus and pushed through the anterior oesophageal wall behind the tracheostome at the 12 o'clock position, about 8 mm from the tracheocutaneous suture line. The membranous trachea is cut with a No. 15 scalpel, and the hole is widened by 3–4 mm. A No. 14 red rubber catheter is then inserted into the distal oesophagus. Attach the catheter's outside end to the skin under the clavicle with silk suture. From 7 to 10 days post-op, the red rubber catheter is replaced with a vocal prosthesis.
- A suture is placed at the base of tongue and the oesophageal wall, converting the circular pharyngeal defect into two elliptical ones. When not strained, primary closure of the pharyngeal defect is usually possible.
- Preferably, the pharynx should be closed transversally with a 2-0 chromic catgut suture knotted at the midline of the tongue with the mucosa and muscle of the anterior wall of the oesophagus. Each side of the pharyngeal defect should be sealed with an interrupted inverting suture, beginning at the lateral borders and moving towards the midline. Care should be taken to flip the mucosal edges and avoid inverting and burying them under the suture when tying knots. The pharyngeal closure is complete when done with care.
- Position suction drains lateral to the throat; remove them via skin stab incisions. Seal the skin and platysma with nylon and Vicryl sutures. Avoid tension in closure.

6.3 Case 1

A 56-year-old man who had been a known smoker and alcoholic for the past 25 years presented with hoarseness of voice for 1 month and respiratory problems for 1 week. On video laryn-

goscopic examination, the right true vocal cords showed an ulceroproliferative growth with right-side vocal cord fixation. On CT scan, the right side of the glottic area showed a heterogeneous lesion with expansion into the paraglottic space and erosion of the thyroid cartilage.

The patient was planned for a total laryngectomy.

The operative surgical steps, surgical specimen of laryngectomy and follow-up of the same patient are depicted in Figs. 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, and 11.

Fig. 1 Marking of skin incision from one mastoid tip to the opposite mastoid tip: tracheostoma is included in the incision, and the marking for skin excision is made around the tracheostoma

Fig. 2 Lateral view of the marking of the skin incision

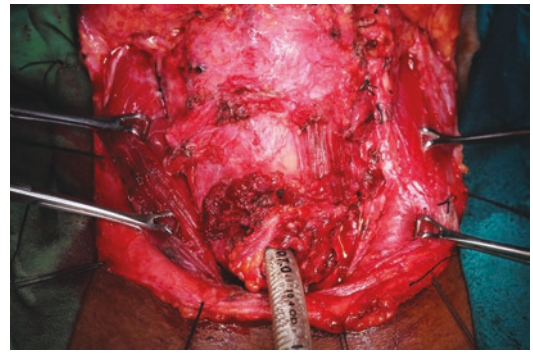

Fig. 3 Anterior view of the larynx after the elevation of the upper and the lower skin flaps with retraction of the sternocleidomastoid muscles on both sides laterally

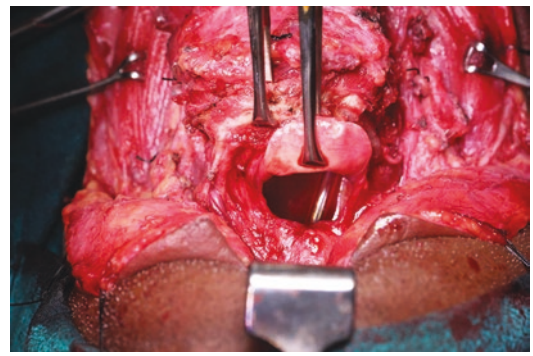

Fig. 4 Entering the pharynx through the vallecula

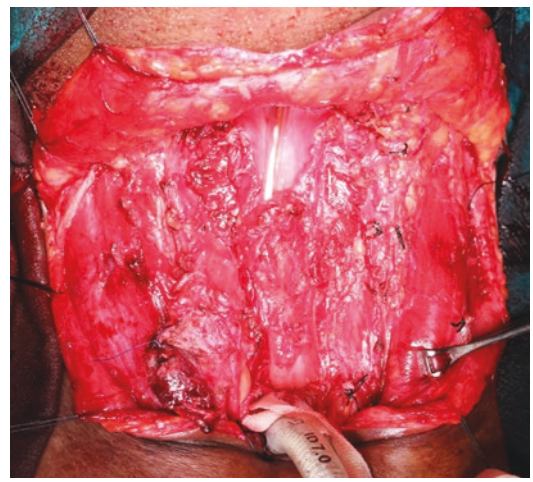

Fig. 5 Surgical site after removal of the larynx and the anterior pharyngeal defect can be visualised

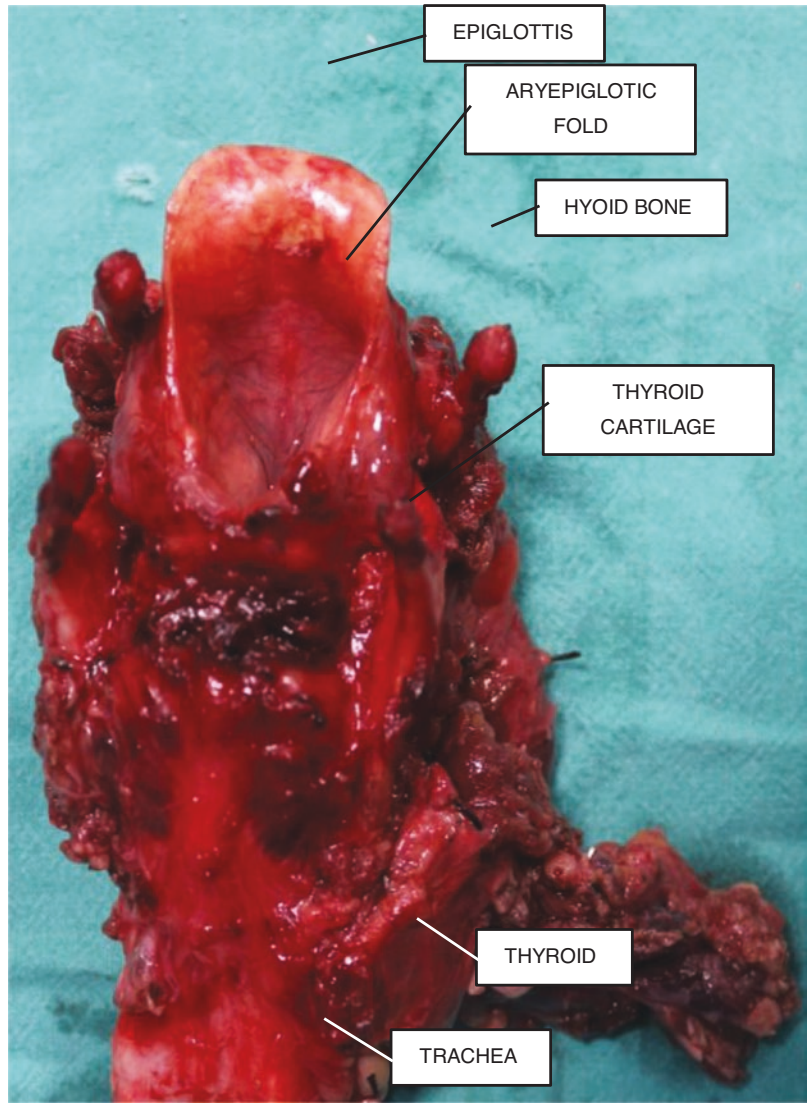

Fig. 6 Posterior view of the surgical specimen of total laryngectomy

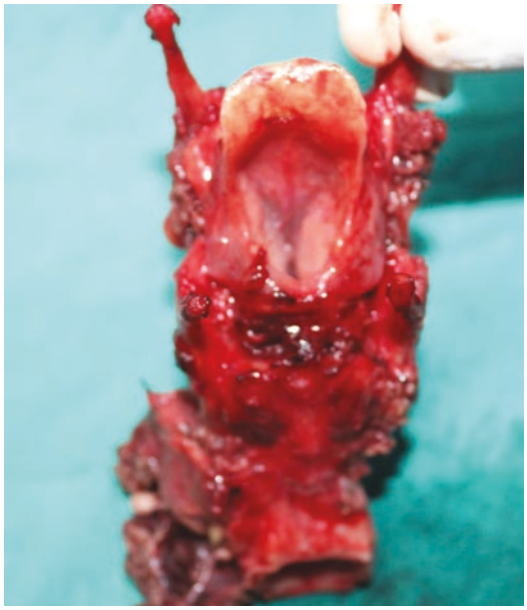

Fig. 7 Posterior view showing the hyoid bone, the epiglottis, the aryepiglottic fold and the cut end of the trachea of the total laryngectomy specimen

6.4 Post-operative Care

- Nasogastric tube feedings are necessary, and they might start within 24 hours.
- When they stop working, suction drains are removed.
- In 7 to 8 days, oral fluids and a puréed food may be introduced if the skin incision and neck flaps are fully healed.
- At 2 weeks, tracheostome sutures are taken out.
- Speech therapy for the oesophagus might start as soon as 3 weeks following surgery.
- The red rubber catheter is withdrawn on the tenth day if an instantaneous TEP is carried out, and a vocal prosthesis is put in its place (Blom-Singer or Provox).
- As soon as 3 weeks following surgery, TEP speech therapy can start.
- To prevent stenosis of the post-laryngectomy stoma, a tracheostomy tube must be inserted through it.

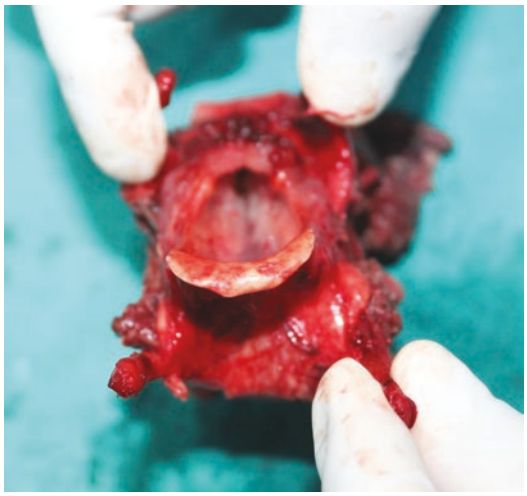

Fig. 8 Superior view of the surgical specimen

6.5 Case 2

A 43-year-old male patient, a known smoker and alcoholic, presented with a history of change in voice; on video laryngoscopic examination, an ulceroproliferative growth was present over the right true vocal cord. The patient underwent a direct laryngoscopic examination and biopsy under general anaesthesia. The final HPE report was well-differentiated squamous cell carcinoma. The patient underwent radical radiotherapy. The patient was disease-free for 8 months, following which the patient again presented with similar complaints of change in voice. The patient had recurrence, and he was planned for salvage laryngectomy.

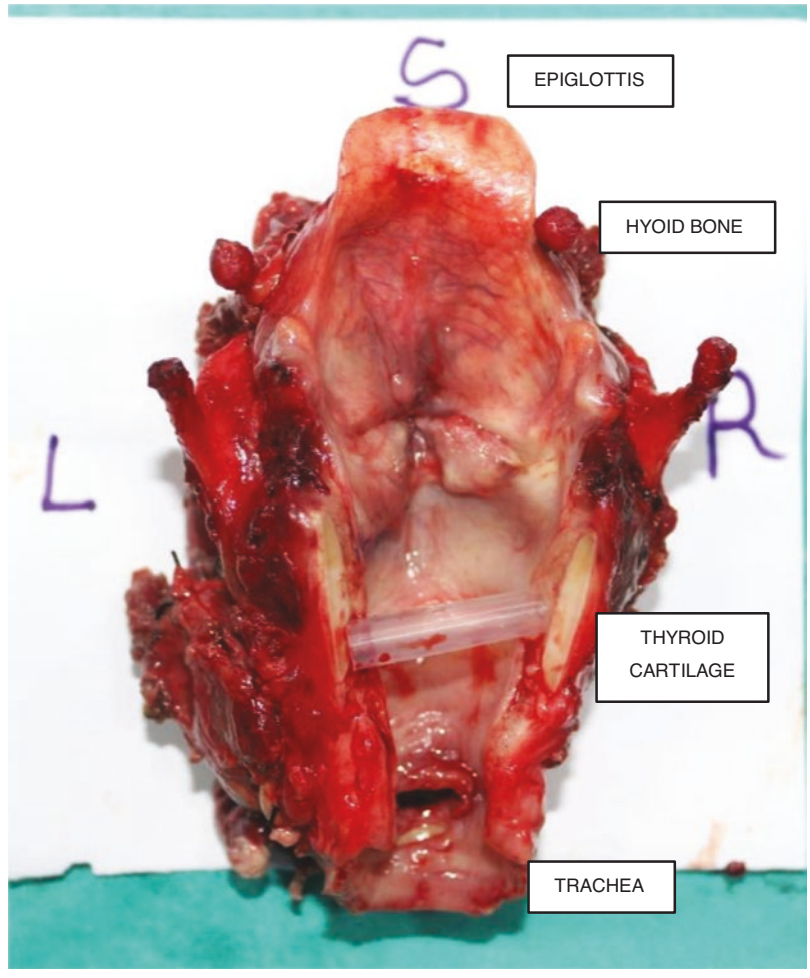

Fig. 9 Posterior view of the total laryngectomy surgical specimen after the posterior cricoid split to visualise the true vocal cords showing the lesion in the right true vocal cords

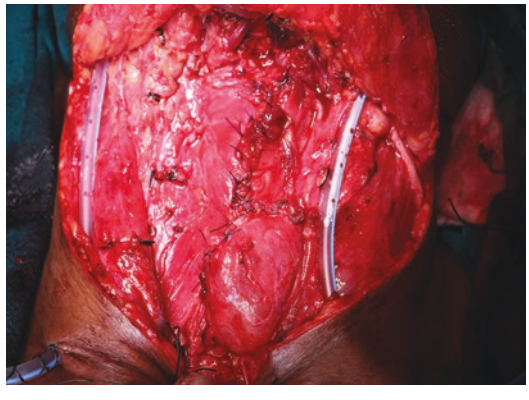

Fig. 10 Vertical closure of the pharyngeal defect in layers with the left lobe of the thyroid in the surgical bed and bilateral neck drains placed

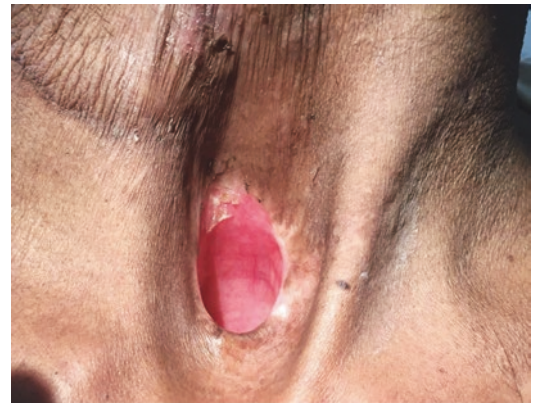

Fig. 11 Follow-up after 6 months with well-healed laryngectomy stoma

The operative surgical steps of salvage laryngectomy, surgical specimen and follow-up of the same patient are depicted in Figs. 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, and 24.

Fig. 12 Marking for skin incision with marking for stoma

Fig. 13 Lateral view of the skin incision

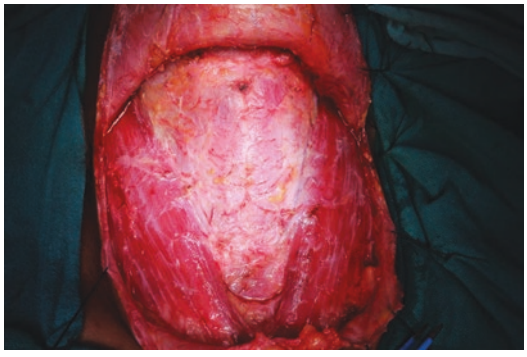

Fig. 14 Elevation of upper and lower skin flaps

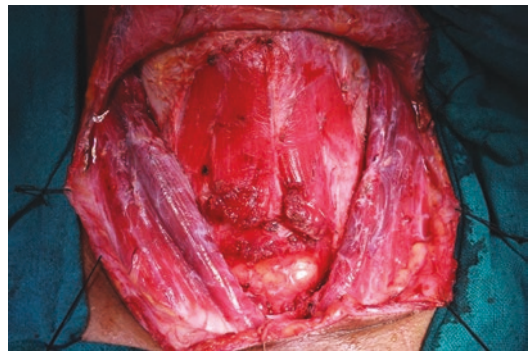

Fig. 15 Exposing the strap muscles and the sternocleidomastoid muscle with the inferior end of the strap muscles cut to expose the trachea

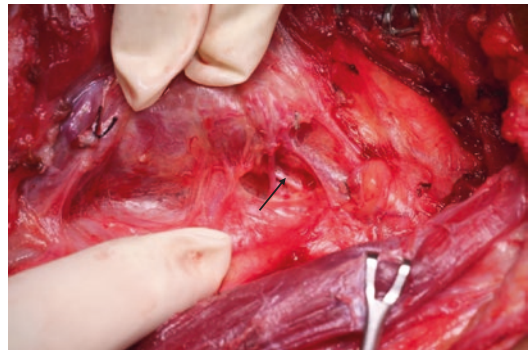

Fig. 16 The arrow shows the recurrent laryngeal nerve after retraction of the right lobe of the thyroid

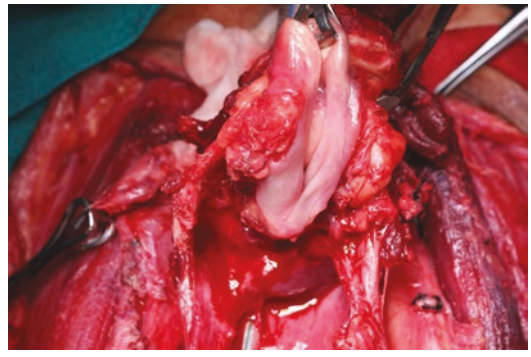

Fig. 17 Entering the pharynx superiorly through the vallecula

Fig. 18 Separating the larynx from the pharynx, Pharyngeal defect can be visualised. * is used to show the carotid artery

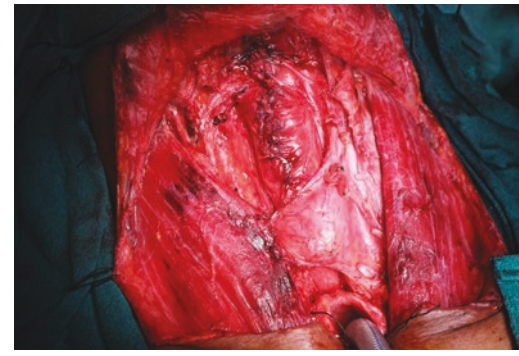

Fig. 21 Closure of the pharyngeal defect by suturing the pharyngeal mucosa, T-shaped closure done. The pharyngeal mucosa closed in two layers

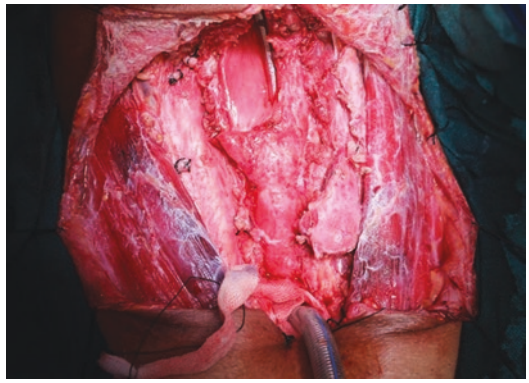

Fig. 19 Post-laryngectomy surgical bed, showing the pharyngeal defect along with the preserved left lobe of the thyroid

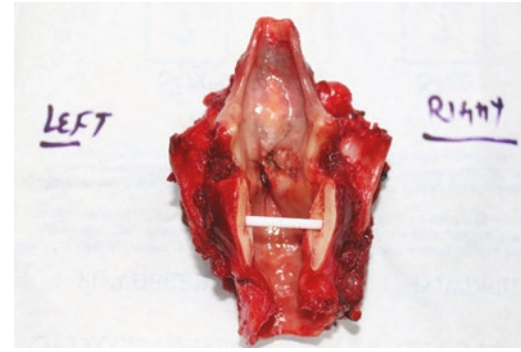

Fig. 22 Post-laryngectomy specimen showing ulceroproliferative growth in the right true vocal cords, and the lesion is seen extending to the anterior commissure

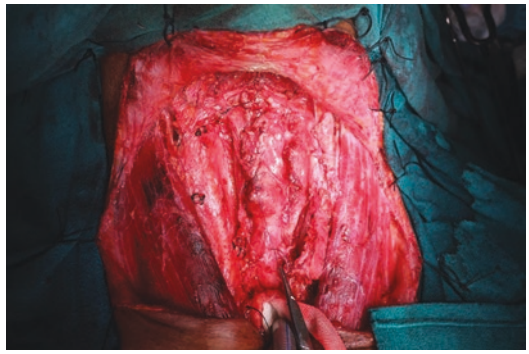

Fig. 20 Connell suturing of the pharyngeal mucosa

Fig. 23 Closure of the total laryngectomy neck wound

Fig. 24 Third-week follow-up post-salvage laryngectomy with well-healed neck wound and laryngectomy stoma

7 Airway Management

7.1 Introduction

Laryngotracheal stenosis care continues to be a difficult surgical issue, particularly in the juvenile age range. The intricacy of the numerous preoperative circumstances suggests that the issue cannot be resolved by a single therapeutic approach.

One must take this into account:

- The type of the stenosis:
  - Congenital
  - Acquired
- Location:
  - Supraglottic
  - Glottic
  - Subglottic
  - Combined
- Degree of obstruction
- Length in the craniocaudal axis
- Association with vocal cord ankylosis or neurogenic paralysis

Furthermore, significant comorbidities or congenital defects in both adults and children, as well as the existence of tracheal injury (stenosis or localised malacia) connected to the tracheostoma or to the tracheotomy cannula, might further complicate surgical care.

7.2 Aetiology

7.2.1 Subglottis

Prolonged intubation is the most frequent cause of subglottic stenosis (SGS) in children.

The most prevalent laryngeal anomalies in infants are listed below in a descending order of incidence [15]:

1. Laryngomalacia
2. Bilateral vocal fold paralysis
3. Congenital SGS

Congenital SGS is divided into cartilaginous and soft tissue stenoses, according to Holinger [16]. When the cricoid region's lumen is less than 4 millimetres in diameter in a full-term baby or 3 millimetres in a preterm baby, it is present. After the eighth week of pregnancy, there is incomplete recanalization of the laryngeal lumen, which leads to the cartilaginous type. The cricoid may have different defects such as overall thickening of the cricoid ring, a big anterior or posterior lamina or an elliptical form, or it may be normal in shape but too small for the infant's size.

A congenital SGS is connected to mediastinal abnormalities such cardiovascular, tracheobronchial or oesophageal defects in about 50% of patients [17]. This suggests that any mediastinal malformation necessitates bronchoesophagoscopy before treatment to rule out a mild asymptomatic congenital SGS for the otolaryngologist, thoracic surgeon and anaesthesiologist.

Infants and children are more likely to sustain injuries that result in acquired SGS following traumatic intubation for resuscitation, after intubation for severe cranial injuries, when laryngoscopy is challenging due to anatomical issues or when a mild congenital subglottic stenosis has been disregarded. The subglottic damage brought on by the indwelling ET tube, as well as gastroesophageal reflux, will be exacerbated by any systemic dis-

ease that reduces capillary perfusion (e.g. shock, anaemia) or increases susceptibility to infection (e.g. diabetes, immuno-suppression). Benjamin in 1993 provided a detailed description of the transformation of acute intubation lesions into cicatricial sequelae of the glottis and subglottis [18]. They are comparable in both adults and children, although children's glottis involvement is more pronounced. Although many conditions, including blunt trauma, inhalation injuries, high tracheotomies, thyroidectomies, Wegener's granulomatosis and idiopathic causes, can result in benign SGS in adults, post-intubation injury is still by far the most frequent cause of SGS that can be treated with resection and primary reconstruction. Endotracheal intubation consequences are often nonexistent or minor in the supraglottic area. They primarily appear as bands of the scar tissue tying the vocal cords posteriorly, either with or without cricoarytenoid ankylosis, at the level of the glottis. This condition is known as posterior glottic stenosis (PGS). A fusion of the voice cords is also occasionally seen. Circumferential ulcerations in the subglottis can result in the development of granulation tissue, which evolves into contracting scars and causes SGS.

Grading System

The Cotton-Myer grading scale is used in the paediatric community [19] and is frequently employed to grade SGS. SGS is graded using this approach in four categories.

Cotton-Myer Grading System for Subglottic Stenosis

Cotton came up with the traditional SGS grading method, which is used all around the world. The updated Cotton-Myer grading system [19] was first used in 1994 and is based on the estimation of the stenotic diameter obtained by putting an endotracheal tube through the stenosis to determine the percentage of blockage (Table 1).

Table 1 Cotton-Myer grading system for subglottic stenosis

Classification	From	To
Grade I	No obstruction	50% obstruction
Grade II	51% obstruction	70% obstruction
Grade III	71% obstruction	99% obstruction
Grade IV	No detectable lumen	

McCaffrey Grading System for Laryngotracheal Stenosis

McCaffrey's grading scale for laryngotracheal stenosis in adults [20] is the most often used, and it would categorize laryngotracheal stenosis according to severity. It would also serve as a foundation for evaluating the outcomes of various treatment methods for this ailment and as a predictive tool (Table 2).

7.2.2 Trachea

Tracheal stenosis in infants is typically brought on by either intrinsic compressions from cardiovascular defects or congenital deformities of the trachea itself (tracheomalacia, web, long-segment stenosis with circular "O" rings of cartilage). A localized tracheomalacia may require more precise tracheal surgery even after the vascular defect is corrected.

Adults may develop benign tracheal stenosis as a consequence of cuff lesions brought on by endotracheal or tracheostomy tubes or as a result of tracheostomy complications, such as anterior granuloma or stenosis and suprastomal granuloma or collapse at the location of the previous stoma.

7.3 History of T-Tube

William Montgomery, a physician at Massachusetts General Hospital's Department of Otorhinolaryngology and Harvard Medical School, created the T-tube for the first time in 1962.

After tracheal surgery, the T-tube was initially used to prevent tracheal stenosis [21]. The first T-tube was made of acrylic, which was too inflex-

Table 2 McCaffrey grading system for laryngotracheal stenosis

Stage	Location of stenosis
I	Subglottic or trachea <1 cm long
II	Subglottic within the cricoid ring >1 cm long
III	Subglottic + upper trachea
IV	Subglottic + glottic (vocal cord fixation or paralysis

ible to be intubated and negatively impacted the tracheal cilia's ability to expectorate.

The "safe-T-tube" was created by Boston Medical Products in 1986, and it was subsequently made of implantable silicone [22].

The T-tube was first used to temporarily prepare for tracheal repair or tracheotomy and anastomosis as well as to treat acute tracheal injuries and stenosis.

Additionally, it was used to transitionally treat benign tracheal lesions before surgery and for individuals with laryngotracheal stenosis who were not candidates for surgical therapy [23].

7.4 Features of T-Tube

These are the design characteristics of the T-tube in comparison to other tracheal stent designs:

(I) When compared to metallic-coated stents, T-tubes have smooth inner and outer walls and preserve the mucociliary expectoration function the most. Additionally, the T-highly tube's polished surface prevents scabbing and adhesion, and the connection's cone-shaped design on the edge gives comfort without encouraging granulation tissue hyperplasia.

(II) The internal branch of the T-tube is used to support and mould the trachea, and the exterior branch is utilized to secure the T-tube, which overcomes the straight silicone stent's frequent displacement propensity. According to reports, 6–18% of patients who get straight silicone DUMON stent insertions experience stent displacement [24, 25]. Additionally, the external branch's ring and groove design makes it possible to secure the spacer and utterance valve, preventing the T-tube from shifting when the patient breathes or speaks.

(III) Patients with various tracheal thicknesses have alternatives thanks to the various models and widths of the internal branch of the T-tube. The length of the stenotic tracheal portion and the tracheostomy site, which necessitates careful and exact measurement and evaluation before putting the T-tube, can be used by the operating physician to calculate the upper and lower branch lengths.

(IV) When the external branch is opened, ventilation and sputum aspiration can be safely carried out externally or through the ventilator in situations of upper T-tube stenosis or a secretion blockage.

It is straightforward and secure to insert and remove the T-tube [26], and following implantation, the patient may speak and breathe normally once more.

In summary, the Montgomery T-tube is a superior option for subglottic stenosis than the traditional self-expanding metal stents and straight silicone DUMON stents.

7.5 Laryngofissure and T-Tube Insertion

7.5.1 Operative Procedure and Caveats

- Since Montgomery first detailed the basic technique of insertion, other surgeons have developed small adjustments to make the insertion process simpler and quicker.
- Here, we describe how to introduce a laryngofissure T-tube using our method.
- The installation of the T-tube requires a tracheotomy opening, which may already exist or be made just before the surgery.
- Based on radiographic information, such as computed tomography (CT) of the airways with exterior (3-D reconstruction) or interior (virtual bronchoscopy) depiction, the diameter of the T-tube is determined.
- Alternatively, the diameter can be calculated while being directly seen via a bronchoscope.
- The optimal diameter would result in the T-tube fitting the airways tightly with the least amount of anterior-posterior displacement.
- The distance between the vocal cords and the tracheotomy stoma and the length of the tracheal lesion distal to the tracheotomy stoma are carefully measured using a bronchoscopic

procedure to determine the optimal length of the T-tube.
- The intraluminal limb's edges are polished with sandpaper after being cut with a scalpel to the required length.
- Midway between the thyroid prominence and the cricoid cartilage, a transverse skin incision is made.
- In the subplatysmal plane, superior and inferior skin flaps are elevated.
- After locating the midline raphe between the sternohyoid muscles, the soft tissue is dissected until the thyroid cartilage is reached.
- The cricothyroid membrane, cricoid cartilage and thyroid cartilage are all exposed.
- A midline incision is made over the thyroid perichondrium, which is then exposed vertically and removed of its cartilage.
- A midline incision is made over the thyroid cartilage to split it vertically.
- The lower end of the stenotic segment is reached by extending the incision inferiorly over the trachea.
- To reveal the stenotic segment, the trachea and incised thyroid cartilage are laterally pulled back.
- Either the cold steel technique or the CO_2 laser is used to carefully dissect the stenotic fibrous tissue.
- A laryngofissure is used to implant the vertical limb of the T-tube.
- The T-tube is positioned at the level of the now-removed stenotic section.
- The tube's superior end should be positioned so that it touches neither the true vocal cords nor their lower end at any point.
- Vicryl 3-0 suture is used to seal the laryngofissure.
- On either side of the surgical incision, corrugated rubber drains or glove drains are positioned.
- The horizontal limb of the tube is removed, and layers of the skin and soft tissue are closed around it.

7.5.2 Case 1

A 64-year-old man who had respiratory problems for a month, which worsened for a week, now complains that he is unable to engage in intensive activities. Due to a lower respiratory tract infection, the patient had previously had prolonged intubation and mechanical ventilator support for roughly 25 days. After being extubated, the patient had respiratory problems but was still able to do daily tasks. The patient received an emergency low tracheostomy when he arrived at our hospital, and after that, he was thoroughly examined to determine the degree and scope of the stenosis.

The patient's laryngofissure procedure was then scheduled to include the stenotic segment's removal and the implantation of a T-tube.

Preoperative radiological imaging, intraoperative surgical steps of laryngotracheal fissure and insertion of T-tube, follow-up endoscopy and the follow-up soft tissue neck are depicted in Figs. 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37 and 38.

7.5.3 Care of T-Tube

- To maintain phonation and enable appropriate humidification of air entering the respiratory tree, the T-tube should always be blocked. Dryness of respiratory secretions and luminal blockage are encouraged by open T-tubes. When the T-tube is blocked, breathing becomes difficult, which may be an early sign of laryngeal oedema or a later sign of the development of subglottic granulation tissue.
- Respiratory secretions can be kept moist if the T-tube needs to be left open for a brief period of time by using humidified air, mucolytic drugs (acetylcysteine) or expectorants (Guaifenesin).
- With the first 1 or 2 weeks following surgery, it is advised to wipe the extraluminal limb with a Q-tip soaked in hydrogen peroxide and provide two to three daily instillations of 1 cc to 2 cc of normal saline into the T-tube's lumen.
- Similar to this, regular suctioning of the T-tube is recommended during the first few days following surgery, but if the tube remains blocked, subsequent suctioning is seldom required.

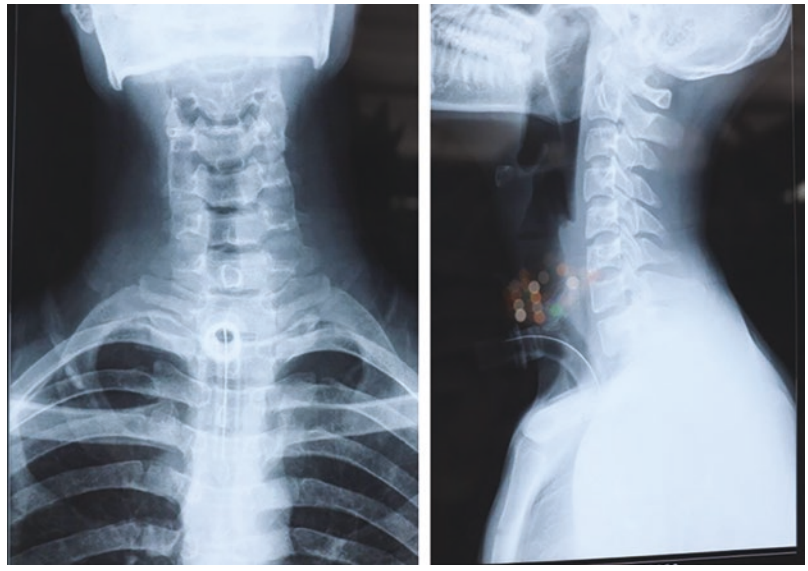

Fig. 25 X-ray anteroposterior view showing suprastomal stenosis in the upper tracheal regin

Fig. 26 CT scan sagittal cuts showing stenosis in the upper tracheal region above the level of tracheostomy site

- To help steer the suction catheter up and down, the extraluminal limb of the T-tube can be angled superiorly and inferiorly.
- To prevent irritating the mucosa, the suction catheter shouldn't be advanced past the intraluminal parts of the T-tube.
- To replace the T-tube that is critically blocked, the patient should always have access to a replacement tracheostomy tube in a size chosen to accommodate the tracheotomy orifice.

7.5.4 Removal of the T-Tube

- The extraluminal limb can be pulled anteriorly with a strong pull to remove the T-tube. Except in cases of malpositioning in the initial postoperative period, failure to remove residual secretions leading to chronic partial blockage or acute total obstruction, T-tube removal is seldom indicated [27].
- T-tubes have been observed to plug in and remain there for extended periods of time without frequently needing to be changed.

Fig. 27 Marking of the skin incision

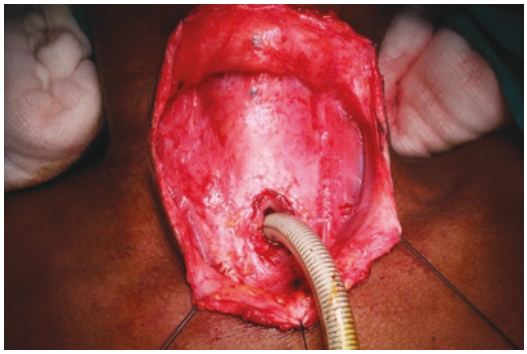

Fig. 28 Elevation of the upper and the lower skin flaps in the subplatysmal plane and stomal freshening

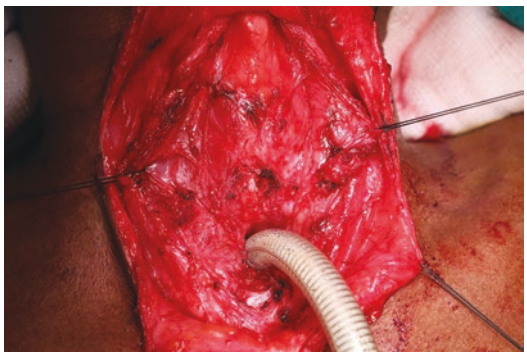

Fig. 29 Exposure of strap muscles and strap muscles being retracted laterally

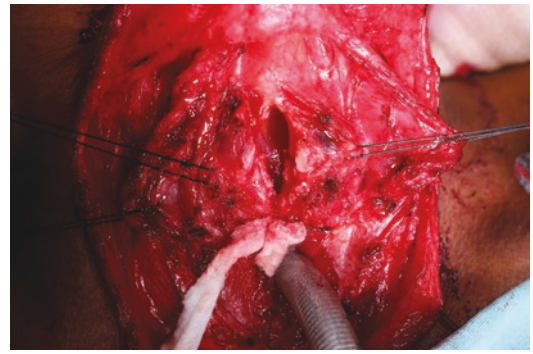

Fig. 30 Laryngofissure: Midline incision over the thyroid cartilage and thyroid cartilage being retracted laterally

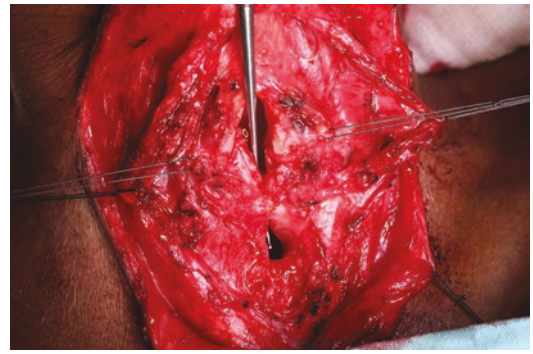

Fig. 31 Intact anterior tracheal wall which is below the laryngofissure incision and above the tracheostoma

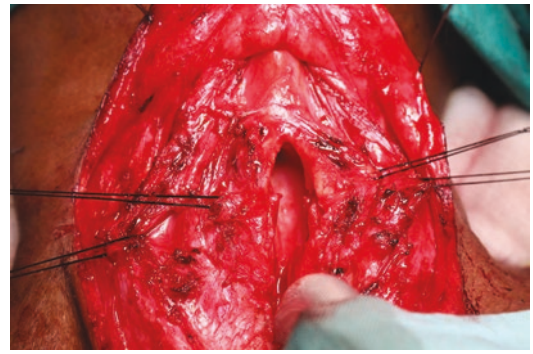

Fig. 32 Complete exposure of the upper trachea and subglottis after removal of the stenotic segment using CO_2 laser and coblator with T-tube placement

Fig. 33 Vertical limb of the T-tube is placed in the upper trachea, and the anterior wall of the trachea is closed around the horizontal limb of the T-tube

Fig. 36 Surgical wound in the immediate post-operative period

Fig. 34 Skin and soft tissue closed in layers around the horizontal limb of the T-tube with glove drains on either side

Fig. 37 Follow-up after 9 months with a well-healed surgical scar, and horizontal limb of the T-tube is plugged

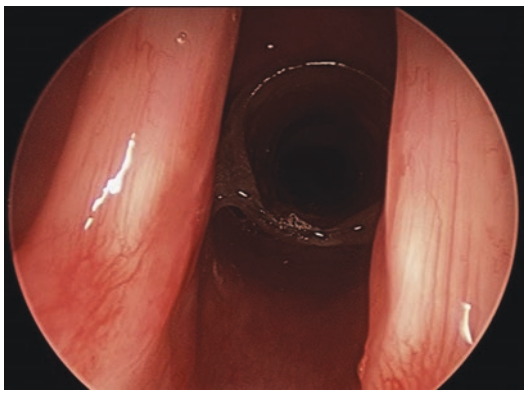

Fig. 35 Endoscopy in the immediate post-operative period showing the upper end of the T-tube just below the level of the true vocal cords but not touching them

- In benign tracheal disorders, it is advised to periodically remove the T-tube and evaluate the stenotic tracheal segment beneath it to determine if the T-tube still needs to be used as a stenting device.
- Unknown is the appropriate time frame for such an evaluation. The observations made in the T-tube series are consistent with a 6- to 12-month time frame.

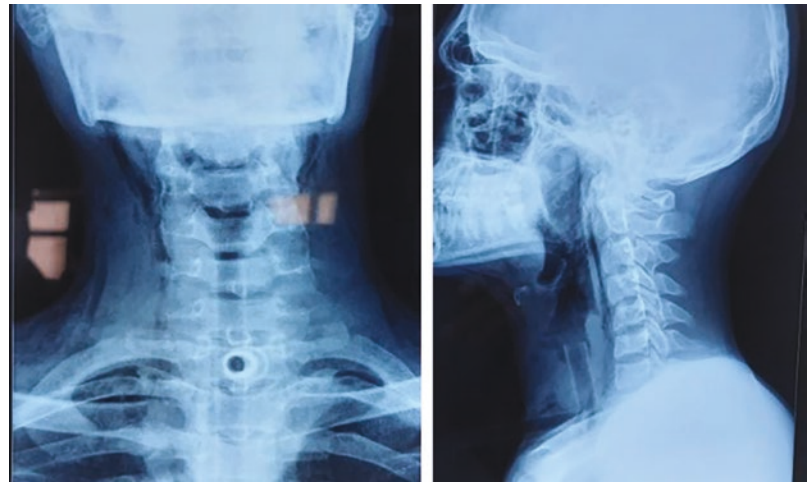

Fig. 38 X-ray anteroposterior and lateral view in post-operative period showing the T-tube in place and an adequate airway

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Neurogenic and Benign Neck Tumours

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1 Introduction

Neurogenic tumours and paragangliomas are rare head and neck neoplasms near the cranial nerves, carotid artery, internal jugular vein and sympathetic chain. They are often found in the parapharyngeal space (PPS) (base of the skull and hyoid bone apex). The lateral pharyngeal wall and tonsillar fossa form its medial wall. In contrast, the pterygoid muscles, parotid gland and prevertebral muscles form their lateral boundary.

For clinical purposes, the PPS is divided into two components separated by the styloid process and its attached muscles. The masticator space is anterolateral to the styloid process and consists of vessels, fat, deep parotid lobe, areolar tissue and retropharyngeal nodes. Salivary neoplasms are most frequent in the deep parotid lobe or smaller salivary glands.

The carotid space, located posteromedial to the styloid process, contains the carotid artery, internal jugular vein, lower cranial nerves (IX–XII) and sympathetic chain. Common tumours in this space are paragangliomas and neurogenic tumours from lower cranial nerves.

2 Imaging Studies

Soft tissue tumours of the head and neck can be evaluated using various imaging techniques. Depending on the type of tumour, the location and the closeness to important structures, different techniques have different benefits. Imaging neck masses are used to identify:

(1) Characteristics that might narrow the differential diagnosis.
(2) The severity of the illness or the prevalence of perivascular invasion.
(3) Connection of the mass with neurovascular structures.
(4) Disease on nodes is present.

Though radiographic features may suggest the diagnosis within the differential, a precise diagnosis requires histologic analysis.

2.1 Computed Tomography

For the diagnosis of the majority of neck masses, contrasted CT imaging is regarded as the gold standard for imaging the neck. The inclusion of contrast enhances the diagnostic value of the soft tissue examination, and CT imaging is particu-

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_11

larly helpful in examining bone detail and calcification inside the tumour. When determining the integrity, level of tumour involvement and/or carotid artery compression in neck neoplasms, CT angiography can be a helpful adjunct.

In situations when carotid impairment may be anticipated as a result of tumour development or surgical excision, balloon test occlusion investigations can be used in conjunction with angiography to assess intracranial circulation. The parapharyngeal space and other difficult-to-access tumours may be candidates for CT-directed biopsy to obtain diagnostic tissue.

2.2 Magnetic Resonance Imaging

When comparing soft tissue extent and relation in the head and neck area, MRI offers benefits over CT; nevertheless, patient movement can impair picture quality. Nasopharyngeal, sinonasal and parotid lesions are the most common tumours in this area that are close to important tissues. MRI is preferred over CT for determining whether perineural invasion is present. Gadolinium compounds for contrast enhancement during MRI should be conducted similarly to CT. However, non-enhanced T1-weighted images without fat suppression should always be obtained since they typically give the greatest delineation of the normal anatomic structures and the degree of pathologic processes. Fat suppression is important for post-contrast and T2-weighted sequences [1].

2.3 Positron Emission Tomography

In the treatment of patients with head and neck cancer, PET with 18-fluorodeoxyglucose and fused CT images (PET/CT) has developed into a well-established imaging technique. During the initial examination, PET/CT is frequently utilized to evaluate:

(1) The size of the primary tumour
(2) The presence of regional and distant metastatic disease
(3) The presence of second primary tumours such as lung or oesophageal cancers

Also, PET/CT has an expanding role in:

(1) To look for an unknown primary [1].
(2) With decisive chemoradiation, the choice of neck treatment [2].
(3) When evaluating a neck tumour that is known or suspicious to be malignant, PET/CT is helpful.

PET has a high sensitivity for malignancy (it detected 93.9% of a series of 212 sarcomas, with a sensitivity of 93.7% for soft tissue sarcomas and 94.6% for osseous sarcomas); however, a significant overlap between low- and high-grade sarcomas remains in standardized uptake values (SUV), and differentiation of the grade based on PET avidity remains imprecise [3].

However, it can occasionally be helpful in the treatment of patients with head and neck paragangliomas (PGLs) to test for the existence of metastases or other sites of illness. PET/CT with 18-fluorodeoxyglucose is of minimal benefit for the majority of benign neck tumours. Some radionuclides, such as 18F-fluorodopa, may be more sensitive than others [4]. These modalities help with correct staging and therapy because they offer information on localized nodal metastatic disease and probable distant metastasis in addition to identifying malignant disease inside the neck. Before beginning aggressive therapy for high-grade malignancies, it is sometimes crucial to employ PET or PET/CT to obtain general staging information.

2.4 Ultrasound (US)

The use of ultrasound imaging as a diagnostic tool has grown in importance for head and neck surgeons. In addition to the physical examination, it can allow for the streamlining of services in the hands of qualified personnel and the inclusion of imaging to the in-office neck examination. Using ultrasonography, a neck mass may be examined to characterize its boundaries, vascularity and accompanying tissues. It can also be determined whether the mass is a lymph node and, if so, whether it has any worrisome signs of cancer (loss of fatty hilum, necrosis, microcalci-

fications, hypoechoic, abnormal vasculature). It enables image-guided FNA biopsy of masses or nodes of interest in addition to the characterization of lymph nodes and neoplasms in the neck.

3 Carotid Paraganglioma

The sympathetic or parasympathetic nervous system contains neuronal cell clusters called paraganglia, often present in the adventitia of blood arteries and nerves. Parasympathetic paragangliomas normally do not secrete catecholamines and mostly affect the head and neck area, in contrast to sympathetic paragangliomas, which do so and most frequently develop in the adrenal medulla. According to the location of parasympathetic ganglia, the majority of paragangliomas in the head and neck area develop in the jugulotympanic region, vagal body, carotid body, superior and inferior laryngeal paraganglionic tissue, nasal cavity or orbit.

3.1 History

- The carotid body was initially described by anatomist von Haller in 1743:
  - At that time, its purpose was not known. The carotid body was termed the carotid gland after histologic examination of the structure showed glandular acini.
- In 1862, von Luschka published the first description of a carotid body tumour.
- Reigner conducted the first carotid body tumour (CBT) excision in 1880. However, the patient did not live.
- Six years after Maydl removed a carotid body tumour, the patient was still alive but suffered postoperative hemiplegia and aphasia.
- The first surgeon to effectively remove a carotid body without ligating the carotid vessels was Albert in 1889.
- In 1903, Scudder reported the first successful resection of a carotid body tumour in the United States [5].
- Histologist Kohn originally coined the term "paraganglion" to characterize the carotid body in that same year [6].
- The name "paraganglionic" was chosen since the carotid body's cells migrate in close proximity to autonomic ganglion cells and are derived from the neural crest.
- Because the carotid body serves as a chemoreceptor, Mulligan coined the term "chemodectoma" to characterize a neoplastic degeneration of the carotid body in a dog in 1950 [7].

3.2 Anatomy and Physiology

In the adventitia of the posteromedial portion of the common carotid artery, bifurcation is where the carotid body is situated. The carotid body typically has a diameter of 3–5 mm; however those who reside in higher altitudes sometimes have bigger carotid bodies. The typical adult gland weighs 12 mg on average, with a previously reported range of 1–47 mg [8].

A little reddish-brown tan ovoid structure, joined to the carotid arteries at the bifurcation by the Mayer ligament, is often discovered during surgical excision. Through this structure, the feeding vessels run predominantly from the external carotid artery, and therefore, meticulous dissection has to be performed to prevent bleeding.

Gram for gram, the carotid body's blood flow and oxygen consumption are greater than those of the thyroid or brain [9].

The Hering nerve, a branch of the glossopharyngeal nerve that starts about 1.5 cm distal to the jugular foramen, is responsible for sensory innervation. By altering respiratory and cardiovascular function in response to changes in arterial pH, oxygen and carbon dioxide tension, the carotid body serves as a chemoreceptor. Acidemia, hypoxia and hypercapnia cause the carotid body to start an autonomic response, which raises the heart rate, blood pressure and the activity of the cerebral cortex.

3.3 Etiology

PGLs have a number of different causes, although most of them (60%) are solitary [10]. In family disorders, particularly multiple endocrine neoplasia types 2A and 2B, numerous pheochromocytomas and PGLs are common.

Neurofibromatosis type 1 and von Hippel-Lindau disease, which is characterized by retinal angiomas and cerebellar hemangioblastomas, are two more disorders connected to PGLs. The relationship between PGL, pulmonary chondroma and stomach leiomyosarcoma is shown by a Carney triad [11, 12]. In addition to these connections, a syndrome of familial PGLs has been reported that up to 20–25% of cases is characterized by many PGLs, particularly in the head and neck area. This disease is linked to germline changes in the succinate dehydrogenase gene. In his 1933 account of two sisters who had carotid body tumours, Chase originally hypothesized that carotid PGLs are hereditary [12].

Genes that code for a subunit of succinate-ubiquinone oxidoreductase have been shown to contain mutations that cause the hereditary type of PGL:

1. Succinate dehydrogenase subunit D (SDHD), which maps to chromosome 11
2. Succinate dehydrogenase subunit B (SDHB), which maps to chromosome 1
3. Succinate dehydrogenase subunit C (SDHC), which maps to chromosomes 1

The inner mitochondrial matrix contains an enzyme complex called succinate dehydrogenase that is in charge of both the reduction of ubiquinone to ubiquinol in the respiratory chain and the oxidation of succinate to fumarate in the Krebs cycle.

Genetically, there are four subtypes of hereditary PGL syndrome:

1. PGL1
2. PGL2
3. PGL3
4. PGL4

PGL1, PGL4 and PGL3 have been shown to have SDHD, SDHB and SDHC germline mutations, respectively. More recently, a mutation in SDHAF2 has been linked to PGL2 [13]. Compared to people with sporadic illness, those with inherited PGL syndrome had earlier tumour development and a greater probability of bilateral and/or numerous tumours. According to earlier findings, familial PGL is uncommon. However, recent research refutes this claim and contends that PGLs are likely associated with germline genetic abnormalities in a considerably greater percentage of cases—typically between 25% and 30%—with SDH mutations being the majority, followed by VHL, RET and NF1 mutations [10, 14].

The most prevalent inherited genetic defect in families with a history of PGLs is PGL1, which is brought on by mutations in the succinate dehydrogenase gene's D-subunit. All three diseases have an autosomal-dominant inheritance pattern, whereas PGL1 has a genomic imprinting-modified autosomal-dominant inheritance pattern. After analysing data from 15 significant Dutch pedigrees, van der Mey and colleagues identified genomic imprinting in PGLs [15]. The imprintable gene is transferred in a Mendelian way, but the sex of the transmitting parent determines how the gene is expressed. When a PGL is inherited from the father, the gene causes a tumour to grow. Children of female carriers never had tumour development. However, offspring of male carriers showed a 50% frequency of tumours.

Although pheochromocytoma is also at risk due to PGL1, head and neck tumours are the most frequently related. By the age of 40, it is predicted that 75% of people with gene mutations would acquire a tumour, with multifocal tumours developing in 56% of instances [14].

The prevalence of SDHB (PGL4) germline mutations is lower than that of their D-subunit counterparts. They also have a greater risk of chest or abdominal PGLs and pheochromocytomas, as well as a 33% probability of malignancy. However, they are linked to a later disease start (40% penetrance by age 40) and a higher likelihood of unifocal illness [14]. In order to manage at-risk family members and ensure that thorough screening for pheochromocytoma and other PGLs occurs, it is imperative to identify these people. It is impossible to overstate the importance of genetic counselling and testing for this patient population.

About 4% of head and neck PGLs are caused by uncommon germline mutations in SDHC

(PGL3). There aren't many families known to possess this mutation. Malignancy and multicentricity are less common than in SDHB mutations and SDHD mutations, respectively. Every patient with a carotid body tumour or other PGLs should receive genetic counselling and testing due to the high likelihood of mutations in patients with a history consistent with sporadic PGLs.

The presence of persistent hypoxemia at a high altitude is another risk factor for head and neck PGLs. It has been noted that altitude and the prevalence of paragangliomas are dose-dependently correlated [16].

3.4 Clinical Presentation and Diagnosis

The usual time between the onset of symptoms and diagnosis for carotid PGL is between the second and fifth decades. Because it clings to the carotid arteries, a carotid body tumour often manifests as a lateral cervical mass that is movable laterally but less mobile in the craniocaudal direction. A positive Fontaine sign has been assigned to this physical finding [17]. An alternative symptom of a carotid PGL is a parapharyngeal mass. Due to their great intrinsic vascularity, many carotid PGLs pulsate by transmission from the carotid arteries or, less frequently, grow on their own. Auscultation may occasionally reveal the presence of a bruit, although carotid compression can make it go away. These tumours can range in consistency from soft and stretchy to hard, and they are often not sensitive. Progressive dysphagia, odynophagia, hoarseness and other cranial nerve deficits (IX through XII) symptoms develop as they expand. Carotid PGLs have been linked to carotid sinus syndrome syncope in some cases [18]. The condition includes reflex bradycardia, hypertension and a loss of consciousness. The head moving on its own or applying digital pressure on the tumour is a triggering cue.

Rarely, PGL of the head and neck may manifest as a tumour that secretes functional neuropeptides. However, clinical results are not necessarily consistent with a head and neck PGL's ability to synthesize catecholamines. All PGLs feature neurosecretory granules, although about 1–3% of them are thought to be functional. Glenner and colleagues originally identified a norepinephrine-secreting functioning carotid body tumour in 1962 [19]. Patients should be questioned about any symptoms or indicators that point to high catecholamine levels. It is important to investigate headache, palpitation, flushing and sweat complaints. A 24-h urine collection from these individuals is tested for norepinephrine and its metabolites, such as normetanephrine and vanillylmandelic acid. An alternative measurement method is plasma metanephrine. Head and neck PGLs lack the enzyme to convert norepinephrine to epinephrine; therefore excessive epinephrine should raise suspicion of an adrenal pheochromocytoma (phenylethanolamine N-methyltransferase).

Preoperative—and—adrenergic-blocking drugs are used to optimize the patient if a tumour is confirmed to be functioning; doing so reduces the chance of abrupt catecholamine release, which can happen while manipulating tumours during surgery. Only in cases with numerous or familial PGLs or in the presence of catecholamine-related symptoms is routine testing for urine metanephrines, vanillylmandelic acid and serum catecholamines recommended.

Multicentricity—The carotid PGL is the most prevalent PGL in the head and neck, and bilateral carotid body tumours are the most common combination of multiple tumours. The so-called "rule of 10" states that the total prevalence of numerous tumours in sporadic instances is around 10%; some of these tumours might include undetected family genetic alterations. Depending on the individual germline mutation, the incidence of multiple tumours is reported to range between 30 and 50% when a family pattern is identified.

Malignancy—Harrington and Dockerty made an effort to categorize malignant carotid body tumours using criteria for malignancy that included:

- Mitoses with giant cells
- Nuclear pleomorphism
- Capsular invasion [20]

These standards would classify half of the 20 tumours under study as malignant. Since then, it has been discovered that higher mitotic rate and capsule invasion should not be regarded as indicators of malignancy, especially given that virtually all carotid body tumours exhibit some level of capsular invasion [21, 22].

Therefore, the only factor that determines whether a tumour is malignant is the existence of metastasis, which must be confirmed by biopsy because PGLs might have multicentricity. Evidence of the disease's spread to nearby lymph nodes or distant locations, most often the bones and the lungs, should be used to make the diagnosis of malignancy [23, 24]. According to Batsakis, 6% of PGLs in the carotid body are malignant [22]. The risk of malignancy for orbital and laryngeal paragangliomas is greatest at 25%, followed by vagal PGLs (10%) and jugulotympanic paragangliomas (5%).

The low malignancy rate of this unusual tumour makes it challenging to determine accurate 5-year survival statistics. According to the data from the National Cancer Database, the overall 5-year survival rate is 60% [25]. Regional disease spread fared far better, with a 5-year survival rate of 78%, but distant metastases had a poorer prognosis with a 5-year survival rate of 11.8%.

3.5 Imaging Studies

There are several diagnostic imaging methods for the investigation of carotid body tumours.

Noninvasive duplex US shows the carotid artery's connection to the tumour and a hypervascular mass. Additionally, the US may identify any intrinsic carotid artery disease.

Typically, a CT scan with intravenous contrast reveals a hypervascular mass at the carotid bifurcation, which splays the internal and external carotid arteries, with ardent contrast enhancement, comparable to normal vessels. Flow gaps could be seen. To show how the carotid vessels relate to the growing neck tumour, CT angiography may be used.

Since MRI with gadolinium provides higher soft tissue contrast compared to CT scanning without the use of ionizing radiation, it may be the most helpful imaging examination for assessing carotid body tumours. It should be acquired for almost all PGLs to assess vascular and soft tissue involvement. MRI is capable of detecting tumours as tiny as 0.8 cm [26]. PGLs bigger than 2 cm in diameter frequently show internal flow gaps, dark lines and dots (salt and pepper look) corresponding to vascular structures on T2-weighted images. However, carotid PGLs don't always have this. PGLs often take up and wash out gadolinium more quickly than other carotid sheath tumours like schwannomas, which take up and wash out the dye more steadily [1].

The internal and external carotid arteries bend and move as part of the lyre sign, which is a hallmark of carotid body tumours. The diagnosis of carotid body PGL should be made only on the basis of radiographic examination. MRI, especially magnetic resonance angiography (MRA), sometimes known as formal angiography, has taken the position of carotid angiography.

The abundance of somatostatin receptors in PGLs makes it possible to use metaiodobenzylguanidine and octreotide scanning, two more recent nuclear medicine functional imaging approaches. These are still not regarded as standards of treatment but are becoming more effective for screening for metastases or pheochromocytoma. Indium-111-labelled somatostatin analogue octreotide is used in octreotide scanning to detect primary amine precursor and uptake the decarboxylase system tumours as well as their metastases [1, 27, 28]. A more recent radioisotope with great sensitivity and specificity is 18F-FDOPA (fluorodopa) PET/CT [4]. These functional imaging techniques have been suggested as potential familial PGL screening tests for people at risk [29]. Additionally, they allow for the investigation of individuals who are at a high risk for multifocality or malignancy, such as those with SDHB mutations, or the discovery of additional tumours when a malignant PGL is suspected [29].

3.6 Classification

A categorization scheme for carotid body tumours has previously been developed, albeit it hasn't been widely used in the literature on carotid body PGLs. The categorization system developed by Shamblin and his colleagues in 1971 for grading the difficulty of excision in carotid body tumours and the possibility of resulting long-term functional impairments in the cranial nerves is still appropriate and still in use today [30].

Group I tumours—small, localized and only loosely connected to the carotid vessels. In this group, surgical excision is typically reported as being performed easily.

Group II tumours—moderate arterial attachment and adhering to or partly encircling the arteries. According to reports, these tumours may be removed surgically with caution.

Group III tumours—encase the carotids completely.

Shamblin and colleagues advised treating these tumours with extreme caution and taking vascular replacement into account.

3.7 Management

Treatment options for carotid body tumours include surgical excision, radiation therapy, preoperative embolization and combinations of these. The patient's comorbidities, surgical risk, tumour size, tumour extension, location and multicentricity are a few factors that influence the therapy option.

3.7.1 Conservative

Due to sluggish growth rates, some studies recommend that non-surgical therapy with periodic imaging follow-up be taken into consideration [31–33]. A patient's characteristics should inform the appropriate intervals.

Li et al. [34] demonstrated that 60 CBT patients who received either conservative or surgical therapy were retrospectively examined. 13.3% of the patients received the latter. The surgical group did not have a higher survival rate than the group receiving conservative care during follow-up.

Rodriguez-Cuevas et al. [35] presented a series of 120 CBT instances, of which 41 (34.1%) patients were followed up on without receiving surgical care. These patients' median follow-up lasted 47 months. None of these individuals provided any further information on rapid tumour growth or metastasis.

When lymphadenopathy, fast growth and discomfort are present during the observation phase, the authors advise taking surgical intervention into account.

3.7.2 Radiotherapy

Fractionated external beam radiation therapy (EBRT), a kind of conventional radiotherapy, has been used as a main treatment, in conjunction with surgery, or as a salvage procedure for individuals for whom surgery is absolutely contraindicated. Valdagni et al. [32] reported 13 CBTs. A median follow-up of 2.5 years (range: 1–19 years) and local control were achieved in all ten patients who had EBRT as their main therapy.

A meta-analysis including the biggest cohort of CBT patients receiving radiation was released:

- Seventeen series yielded a total of 127, with a mean follow-up time of 99.9 months.
- For the patients receiving fractionated radiation, the total doses ranged from 40 to 65 Gy.
- The majority of the patients (44.1%) ($n = 56$) got a total dosage of 40–50 Gy in 20–25 fractions, whereas the remainder either received a total dose of more than 50 Gy or were not recorded.
- Information was collected from 120 (94.5%) patients about the control of the disease; of these, 32 (25.2%) patients had a decrease in the size of the tumour, 88 (69.3%) patients had no change in the size of the tumour and 7 (5.5%) patients had an increase in the size of the tumour.
- Only one mild consequence was noted, and four (3.5%) individuals passed away as the condition advanced.

- No appreciable differences in tumour control or mortality were found between patients who received radiotherapy and those who underwent surgical resection; however, the surgical group had higher rates of major complications (OR: 1.4; 95% CI: 0.66–2.25 vs. OR: 0; 95% CI: 0 to 0; $p = 0.047$) and cranial nerve palsies (OR: 9.65; 95% CI: 6.79–12.5 vs. OR: 0.37).

Instead of eradicating tumours, radiation aims to regulate illness and inhibit tumour development. The avoidance of early severe surgical consequences, such morbidity to cranial nerves and vascular structures, and the high rates of local control are its principal advantages [36]. On the other hand, a significant drawback of radiation is the elevated chance of developing cancer, and certain minor side effects include hearing loss, disorientation and xerostomia [37].

Intensity-modulated radiation therapy (IMRT) is the recommended treatment for CBT, with doses in the range of 46–50, 2 Gy per fraction and a risk of major problems that is negligible below 45 Gy [36].

For individuals who cannot have surgery owing to age, concomitant disorders, or reduction of the risk of baroreflex failure syndrome and prevent its long-term treatment, radiotherapy is a well-known option [38, 39].

3.7.3 Preoperative Angioembolization

Preoperative angioembolization has been extensively suggested as an adjuvant treatment since CBT is highly vascularized and the surgical complications this offers [40].

Therefore, the main justification for preoperative embolization has been to reduce surgical blood loss, operating time and vascular and brain damage [41].

Schick et al. [42], Were the first to perform preoperative embolization prior to surgical excision of a CBT was documented in 1980.

Three meta-analyses comparing the results of individuals who received preoperative embolization prior to surgical resection vs. those who did not undergo preoperative embolization have been reported [40, 43, 44]:

- In two of these studies, patients who underwent preoperative embolization experienced statistically significantly less blood loss and a shorter operating time than patients who did not [43, 44].
- However, another research that examined patients who were slated for CBT surgery found neither operational nor postoperative benefits of preoperative embolization [40].

When dealing with tumours that are larger than 4 cm, encase the internal carotid artery or expand over the angle of the jaw or the C2 vertebral body, some writers advise employing preoperative embolization [45, 46].

Super-selective catheterization of the supplying branches combined with a transarterial technique is frequently used to achieve embolization.

Embolization agents include:

- Non-spherical polyvinyl alcohol (PVA) particles ranging from 150 to 1000 μm
- Gel foam
- Alcohol conjugates
- Tris-acryl gelatin microspheres
- Liquid embolic (glue)
- Coils, among others [40, 47–50]
- N-butyl cyanoacrylate or onyx (Medtronic, Minneapolis, MN)—onyx due to its viscous, "lava-like" flow, allows for progressive dispersion across the tumour vasculature and allows the operator to halt and resume the embolization. It gives the treatment more control [50].

Overall, technical success has been defined when there is an achievement of >50% mean estimated devascularization [51]. The ideal time for surgical resection after embolization is within 24–48 h from the embolization; it has been suggested to perform the procedure before 48 h to minimize the risk of revascularization and recruitment of collateral tumour blood supply [46, 49, 52, 53].

3.7.4 Vascular Resection and Reconstruction

The only effective treatment for CBT is surgical resection [54]. Periadventitial dissection of the tumour from the carotid vessels is typically all that is needed to treat Shamblin I tumours; in contrast, more involved procedures are usually needed to treat Shamblin II or III tumours.

Section and ligation of the common, external or internal carotid arteries may be necessary for treating Shamblin III tumours. Resection or ligation of the external carotid artery can be done when the external carotid vascular surrounds the tumour in order to improve medial exposure and access to the carotid bifurcation for better view of the anatomy. Additionally, the external carotid artery does not need to be rebuilt and can be strangled [52]. The internal or common carotid artery can also be cut or ligated, but the vessel must be repaired or rebuilt thereafter.

For individuals who need to have the carotid vessels resected or tied, a balloon occlusion test can be used to determine how well the patient will tolerate the carotid occlusion. In this procedure, intracranial blood vessels are balloon-occluded, while vasodilators are used to reduce systemic blood pressure. The blood pressure threshold at which the patient experiences unilateral neurologic symptoms is then identified [52, 55–60].

The repair can be:

- Primary
- Autologous vein interposition graft (i.e. saphenous vein, femoral vein)
- Prosthetic vascular interposition graft
- Patch angioplasty with the bovine pericardium
- Prosthetic graft [54, 61, 62]

Additionally, the common carotid artery can be reinserted into the internal carotid artery if necessary [63].

It is best to avoid internal carotid artery ligation if at all feasible because it has been linked to a higher risk of stroke and a higher fatality rate [64].

3.7.5 Operative Technique and Caveat

Preoperative Preparation

- All patients undergoing surgical resection for neurogenic tumours and paragangliomas must be aware of the procedure's nature, the risks involved during the procedure and the necessity of receiving adequate preoperative counselling regarding any potential nerve deficits that may develop as a result of the procedure.
- Preoperative angiographic investigations should be carried out to show the feeding vessels if the lesion is big and highly vascular. Preoperative embolization should also be taken into consideration and, ideally, carried out within 24 h of the anticipated surgical procedure.
- Adequate blood and blood products should be arranged.
- Appropriate occlusion investigations should be carried out to establish acceptable intracranial crossover circulation for bigger lesions when the integrity of the internal carotid artery is at danger. Therefore, it is necessary to establish cross-perfusion from the contralateral carotid artery to the ipsilateral cerebral hemisphere and a balloon occlusion test of the ipsilateral carotid artery.
- If carotid artery ligation is required during the course of the surgery, the preoperative risk of a neurological impairment must be evaluated, and the patient must be made aware of this risk.
- A vascular surgeon should be a part of the operating team.
- Patients who have functional paragangliomas, sporadic facial flushing and hypertension may need to have their hypertension treated with an alpha blocker before surgery. Vigilant intraoperative blood pressure monitoring is required.

Excision of Carotid Body Tumour

- Position: Supine on the operative table, neck extended and rotated to opposite side. Incision: Upper neck skin crease, providing generous exposure of upper neck structures.

- Elevate upper and lower skin flaps as usual. Preserve greater auricular nerve. Use electrocautery for meticulous dissection to reduce blood loss. Handle tumour carefully to avoid significant blood loss.
- After elevation of the skin flap and soft tissue dissection, the underlying carotid body tumour comes into view.
- The internal jugular and common facial veins block the anterior view of the tumour. Dissecting the proximal common carotid artery and the control of the common carotid artery is the initial step in the operative procedure.
- Incise fascia on the sternomastoid, and retract laterally to expose carotid sheath. Secure proximal control of common carotid, and then begin dissection of tumour.
- Incise fascia over sternomastoid, and retract muscle laterally to isolate tumour. Identify/dissect vagus and hypoglossal nerves for preservation.
- Carefully dissect the common carotid artery in the subadventitial plane until bifurcation. Maintain haemostasis with bipolar cautery. Divide the coagulated tissue with tenotomy scissors. Electrodesiccate adventitia with bipolar cautery to minimize haemorrhage.
- Occasionally, a Shamblin type III tumour may encase the carotid artery and bifurcation, necessitating incision and bisection of the tumour. Absolute haemostasis must be secured with electrocautery for a safe procedure.
- Dissection of the common carotid artery provides access to the carotid bifurcation. Retract the tumour cephalad, and use sharp and blunt dissectors for subadventitial dissection. Nearly all blood supply to this tumour comes from the external carotid artery.
- Isolation of the internal carotid artery precedes tumour mobilization to protect it. Bradycardia/hypotension may occur near bifurcation; 1% lidocaine infiltration locally reverses it promptly.
- As dissection of the tumour proceeds, its blood supply is cut off, causing shrinkage and making dissection easier. If the external carotid artery is inseparable from the tumour, it can be cross-clamped, divided and ligated distal to the carotid bulb.
- Dissect and ligate/coagulate each vessel feeding the tumour from the branches of the external carotid artery. Pass a vessel loop around the distal parts of the internal and external carotid artery for retraction and immediate bleeding control.

Case 1

A 15-year-old female patient presented with complaints of swelling in the right side of the neck for 1 year. A pulsating, firm, painless mass measuring approximately 2 × 2.5 cm in size was noticed during a physical examination. This mass had no pressure symptoms and was more mobile transversely than vertically. Deep palpation revealed pulses, and auscultation revealed a mild bruit.

A well-circumscribed, lobulated, heterogeneously enhancing soft tissue density mass measuring 2 cm × 2.5 cm × 2 cm was seen on contrast-enhanced computed tomography (CT), which shows splaying of the external and internal carotid arteries. It is abutting the right internal jugular vein's anterior wall on the side. There was no evidence of intrusion into the nearby structures.

In the right carotid region, an MRI revealed a 2.5 2 cm lobulated oval well-defined lesion mass. On T1-weighted images, the mass signal intensity was almost identical to that of muscle, and on T2-weighted images, the signal intensity increased; there were clearly visible flow gaps. Restricted diffusion was seen in diffusion-weighted imaging. On improved MR angiography, the external and internal carotid arteries were shown to be splayed.

Preoperative radiological imaging, intraoperative photos depicting the steps of surgery and surgical specimen are shown in Figs. 1, 2, 3, 4, 5, 6, 7, 8, 9, and 10.

3.8 Surgical Complications

The most important consequences following surgery in CBT are CNI and stroke. Infection of the

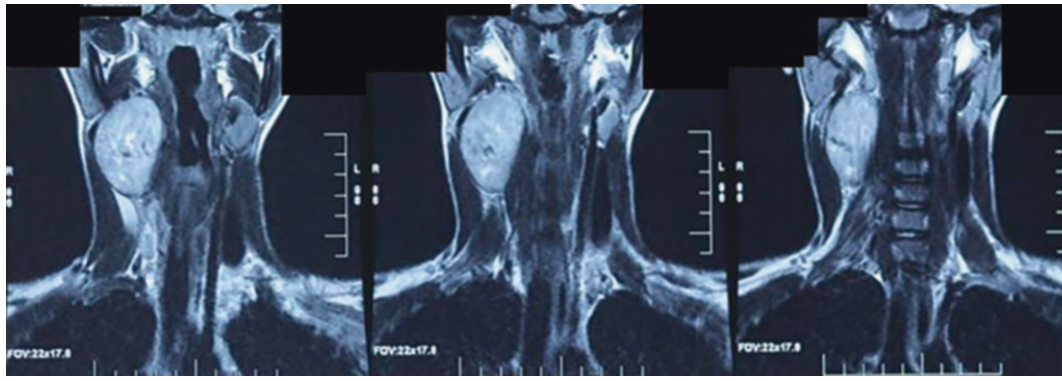

Fig. 1 MRI scan: coronal cuts showing the heterogeneous hyperintense lesion in the right sides

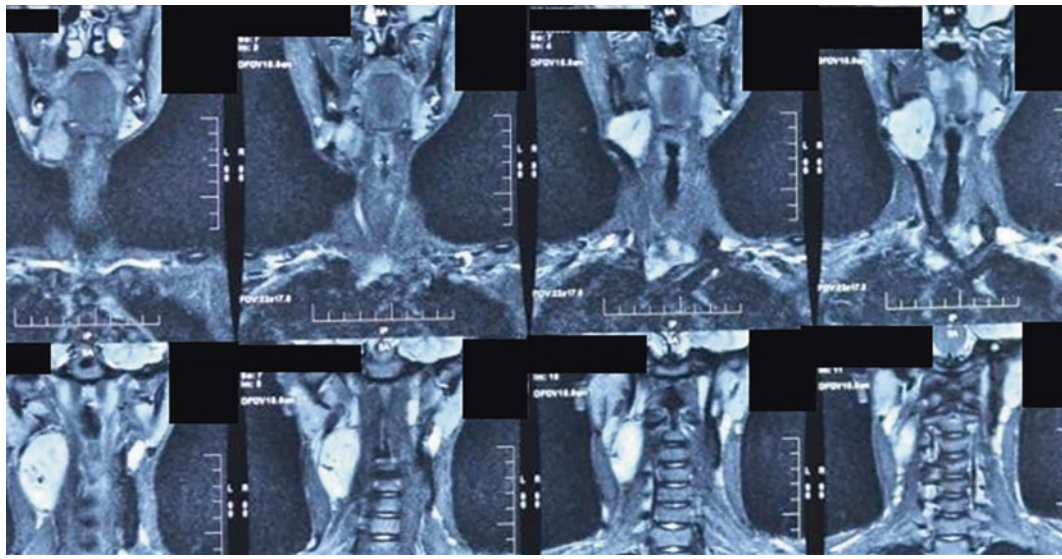

Fig. 2 Contrast-enhanced CT scan from the skull base to the clavicle showing contrast-enhanced hyperdense heterogeneous lesion present at the level of the right-sided common carotid bifurcation

wound, bypass occlusion, carotid artery damage, first bite syndrome, jaw claudication, dysphagia, dysphonia, cardiac issues and postoperative respiratory failure are among the numerous risks.

According to reports, postoperative complications occur anywhere from 20 to 27% of the time [54, 65, 66].

3.8.1 Cranial Nerve Injury (CNI)

The most common significant complication is CNI [67, 68]. The risk of cerebrovascular problems has decreased as a result of surgical advancements; however CNI has not shown any documented benefits [69, 70]. Furthermore, CNI has been reported to occur at rates of up to 50% [70, 71]. The hypoglossal nerve (9.59%) and vagus nerve (25.6%) were the most often damaged in the biggest meta-analysis to date, which included 4327 patients (8.07%) [45].

Other less common damaged nerves such as sympathetic nerves, recurrent laryngeal nerves, lingual nerves and superior laryngeal nerves were found to occur at a rate of 2.01% overall [72].

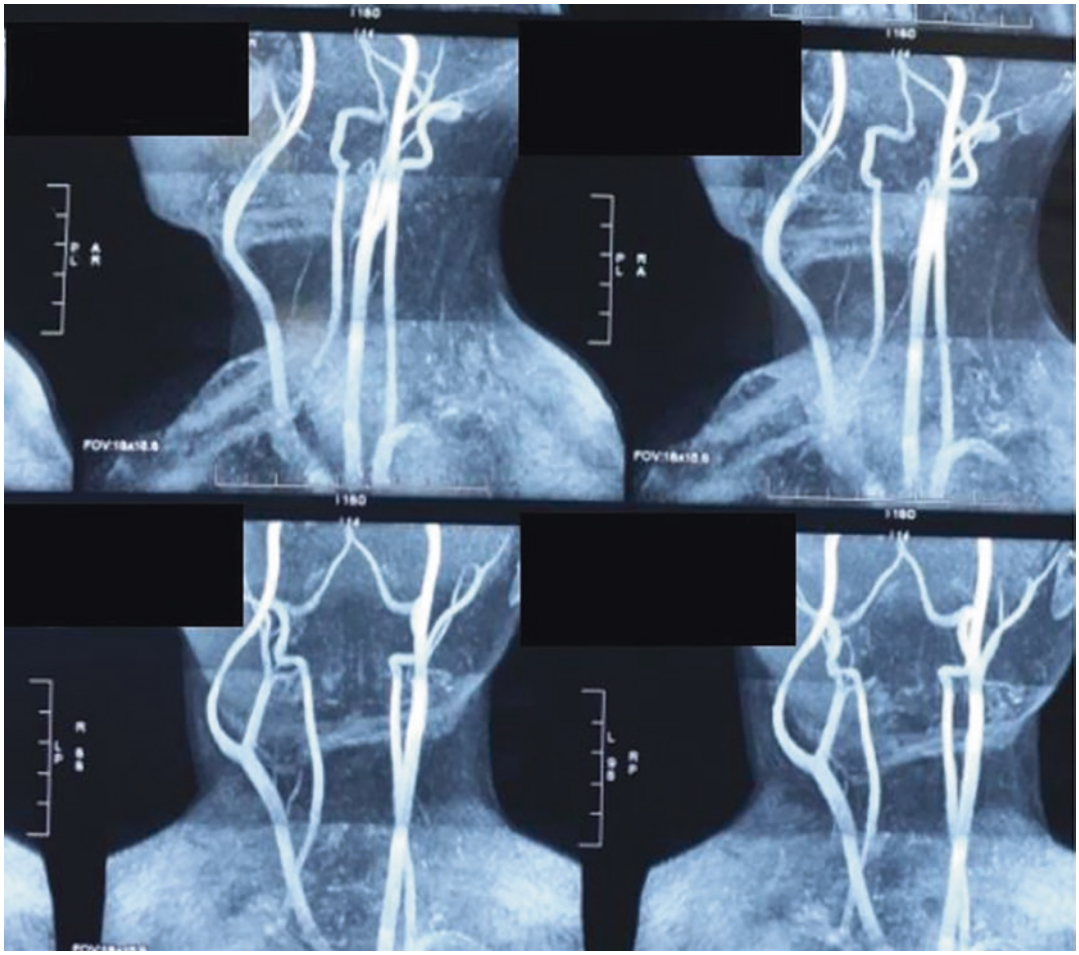

Fig. 3 MR angiography showing internal and external carotid arteries without any involvement, Lyre sign: splaying of the internal and external carotid artery can be seen

Fig. 4 Marking of the location of the tumour and skin incision

Fig. 5 Upper and lower subplatysmal skin flaps elevated

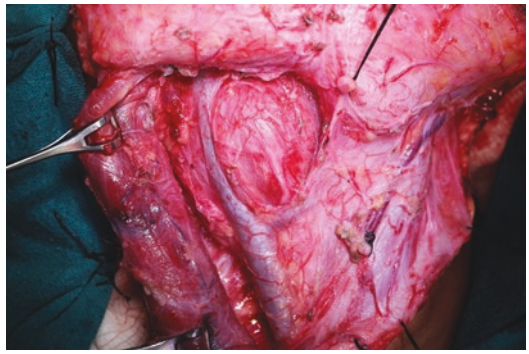

Fig. 6 Exposure of the anterior surface of the tumour with the compressed superior end of the internal jugular vein

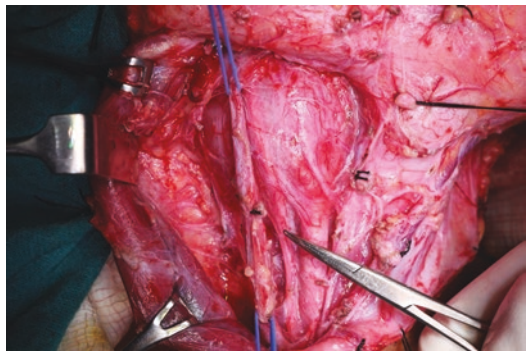

Fig. 7 Meticulous dissection with delineation of the vessels around the tumour

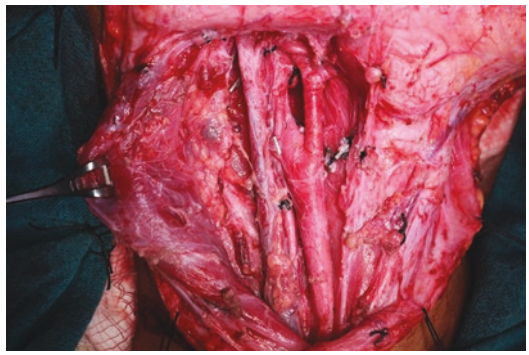

Fig. 8 Surgical bed showing the internal jugular vein and carotid

Carotid artery damage and CNI risk reduction, van der Bogt et al. [73] introduced the craniocaudal dissection of the tumours, drastically lowering the risk of persistent CNI from 26% to 7%.

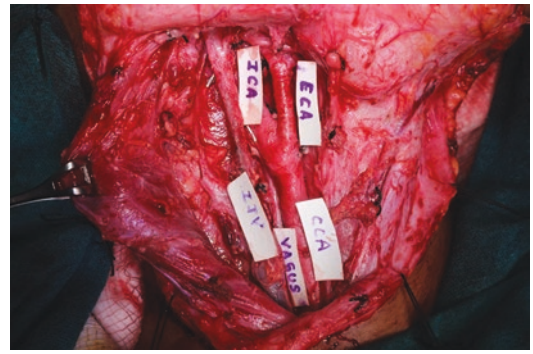

Fig. 9 Surgical bed with the marking of structures

Fig. 10 Specimen in toto

Higher Shamblin grades may be linked to vascular problems and the requirement for vascular resection and reconstruction, but studies have shown no connection between them and the likelihood of CNI [59, 74]. However, it is known that there is an association between bigger tumours, particularly those that are >4 cm and CNI [74–76].

3.8.2 Stroke

However, it is known that there is an association between bigger tumours, particularly those that are >4 cm, and CNI [66, 77, 78]. Robertson et al. [45] found a mean 30-day stroke rate of 3.53% [95% confidence interval (CI) 2.91–4.29], and Shamblin III patients had a higher stroke rate than Shamblin I patients [3.99% (CI) 2.34–6.74 versus 1.89% (CI) 0.92–0.82], respectively).

4 Neurogenic Tumours

The majority of benign peripheral nerve tumours are seen in the head and neck area. The medial and lateral neck compartments are where neurogenic tumours of the cervical area might develop. Lower cranial nerves or the sympathetic chain are the source of neurogenic tumours in the medial compartment. The cutaneous or muscular branches of the cervical plexus or the brachial plexus are the origin of neurogenic tumours of the lateral compartment of the neck. These tumours can occasionally develop at the spinal foramina and manifest as "dumbbell tumours" because they have both an intraspinal and extraspinal component.

Numerous benign and malignant tumours of neurological origin exist; a few of them are listed here.

Benign:
- Reactive
- Traumatic neuroma
- Hamartoma: mucosal neuromas (MEN IIB, Gorlin's syndrome)
- Schwannoma (neurilemoma)
- Neurofibroma: solitary, multiple, diffuse, plexiform (NF-1)
- Perineurioma (storiform perineurial fibroma)
- Dermal nerve sheath myxoma
- Granular cell tumour

Malignant:
- The malignant peripheral nerve sheath tumours (malignant schwannoma, neurofibrosarcoma):
  - Epithelioid MPNST
  - Pigmented (melanotic) MPNST
  - Peripheral primitive neuroectodermal tumour (Askin tumour)
- Autonomic nerve tumour (plexosarcoma)
- Malignant melanoma of soft tissues (clear cell sarcoma)

4.1 Vagal Schwannoma

Schwannomas develop in the neuroectodermal sheath that covers the nearby nerves. They may develop in the face, scalp, cranial cavity, orbit, middle ear, nasal cavity, oral cavity, larynx and neck, among other areas of the head and neck. The sympathetic chain and the cranial nerves V, VII, VIII and X are other sources of schwannomas. The glossopharyngeal and hypoglossal nerves can occasionally have schwannomas, and other cranial nerves even less frequently. Schwannomas are often single-node tumours. Neurofibromatosis type 2 (NF2), which is brought on by hereditary mutations in the NF2 suppressor gene, is suspected when several lesions are detected. Somatic NF2 gene changes have also been linked to the development of non-familial schwannomas.

4.1.1 Management

Different factors need to be taken into account while treating neurogenic neck tumours in the central and lateral compartments. Because the resulting impairment is typically not considerable, lateral compartment schwannomas (i.e. those affecting the cervical plexus and brachial plexus) can be addressed with early surgical surgery. Surgery should be used to treat brachial plexus schwannoma in order to prevent the invasion of more nearby nerves, which would result in yet another neurological disability.

However, before determining whether to do surgery, schwannomas of the medial compartment—specifically, those affecting the sympathetic chain and cranial nerves VII, IX, X and XII—need to be carefully evaluated. Even if the tumour is removed surgically while leaving the surrounding nerve fibres intact, the affected nerve often loses function following the procedure. Therefore, the basic clinical observation with periodic imaging examinations to track the tumour's progress is advised if the damaged cra-

nial nerve is functioning and the tumour does not appear to be expanding in size. Surgical intervention is necessary if the lesion is seen to be growing significantly. A neurological deficiency often only affects the nerve that the tumour originated in.

Operative Technique and Caveat

- An anterior upper neck skin crease starting at the tip of the mastoid process and curving up to the midline of the neck in the submental area is where the incision is made to surgically expose this lesion.
- To expose the tumour, the upper and lower cervical flaps are lifted, and the skin incision across the platysma is deepened.
- Overlying the palpable tumour mass and caudal to the digastric muscle, there are many hyperplastic deep jugular lymph nodes and fibrofatty tissue that obscure the view and the location of the tumour's origin.
- To first show the carotid sheath's structure and the tumour's location, these hyperplastic lymph nodes and fibrofatty tissue are removed.
- The common carotid artery is located after these nodes are removed, and a vascular loop is placed around it for positioning and proximal control of the carotid artery.
- It is possible to see the internal jugular vein next to the common carotid artery, which has been posteriorly displaced by the tumour.
- The internal jugular vein, which is retracted posteriorly, and the common, external and internal carotid arteries, which are retracted anteriorly, are now carefully released by meticulous dissection in the carotid sheath.
- The hypoglossal nerve is precisely located and properly retracted cephalad away from danger.
- The upper region of the neck medial to the digastric muscle at the jugular foramen and the carotid canal must be exposed in order to further dissect the internal carotid artery.
- The tumour's origin from the vagus nerve is now readily apparent.
- The carotid sheath's other anatomical components are located and removed from the tumour for dissection.
- To enable mobility of the distal portion of the vagus nerve, the internal jugular vein is passed behind the tumour medially, while the carotid veins are retracted anteriorly.
- In order to ensure complete haemostasis, the tumour is now carefully gripped, and its dissection is carried out cephalad at the jugular foramen while being seen directly.
- The tumour is delivered in a monobloc method by alternate blunt and sharp dissection, leaving a sizable dead space between the carotid vessels anteriorly, the internal jugular vein posteriorly and the sympathetic chain anteriorly.
- After ensuring complete haemostasis, the wound is irrigated, a drain is placed and the incision is then stitched up twice.
- The postoperative complications of this surgical treatment are caused by vagus nerve paralysis, which causes hoarseness of voice and aspiration of liquids and saliva due to a loss of feeling in the right side's supraglottic larynx.
- Most patients are able to make up for this deficiency without experiencing severe functional impairment.
- A vocal cord medialization treatment combined with laryngoplasty procedures can enhance voice quality.
- These medialization techniques help improve larynx competence and lessen aspiration pneumonia.

Case 1

A 37-year-old female patient presented with complaints of difficulty in swallowing for 2 years, change in voice and swelling in the left side of the neck for 6 months. On physical examination, a swelling of size 2 × 2 cm was present in the left infra-auricular region, and the anterior and superior border of the lesion could not be palpated as the ramus of the mandible was overlying the lesion. Lower cranial nerve palsy of the 10th, 11th and 12th cranial nerves was found on examination.

On CT imaging with contrast, a septate hypodense cystic lesion was detected in the left carotid space measuring 2 × 3.3 × 4.1 cm. Widening of the jugular foramen was seen, with

intracranial extension along the left jugular foramen.

On MR imaging with contrast, there was evidence of a large altered signal intensity space-occupying lesion of size of approximately 4.7 × 3.8 × 2.6 cm seen in the neck region on the left side in the carotid space and anterior to the prevertebral muscle. It shows likely intracranial extension through the jugular foramen up to the ninth and tenth nerve complex.

Preoperative radiological imaging, intraoperative steps of surgery and postoperative follow-up images are shown in Figs. 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, and 26.

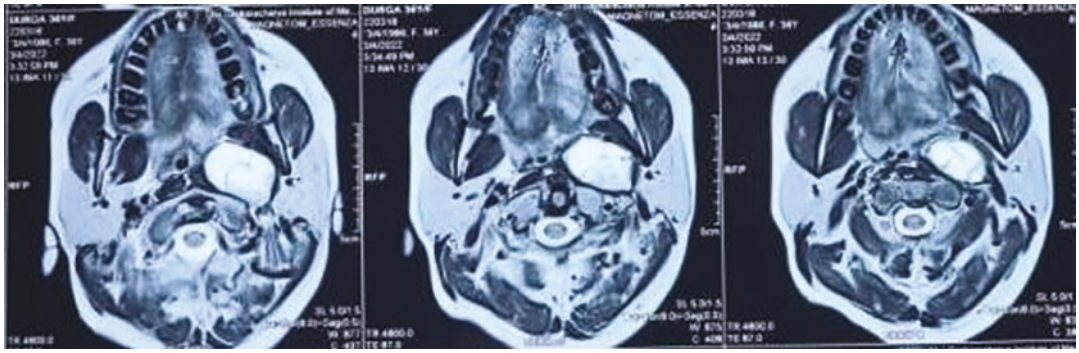

Fig. 11 MRI imaging: axial cuts showing a lesion present in the left parapharyngeal space

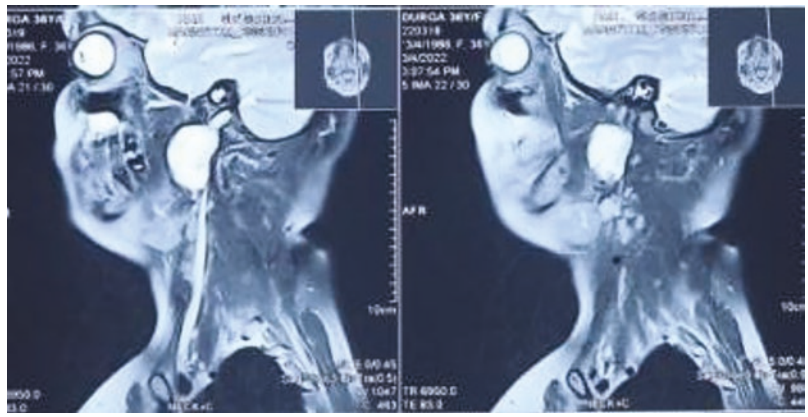

Fig. 12 MRI images: sagittal cuts showing a lesion present in the left parapharyngeal space with lesion extending intracranially through the jugular foramen, and a dumbbell shape of the lesion can be appreciated

Fig. 13 MRI scan: coronal cuts showing a lesion in the left parapharyngeal space

Fig. 14 Diffuse swelling appreciated in the left infra-auricular region; the ramus of the mandible is overlying the anterior border of the swelling

Fig. 15 Surface marking of skin incision, mastoid tip and angle of the mandible

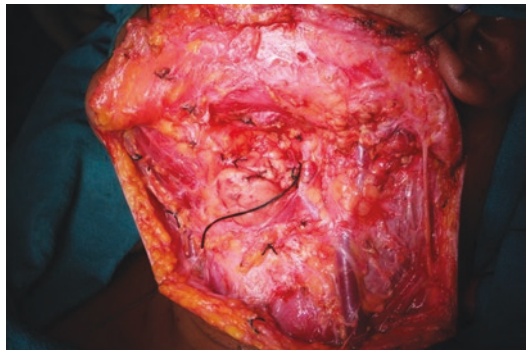

Fig. 16 Elevation of the upper and lower skin flap

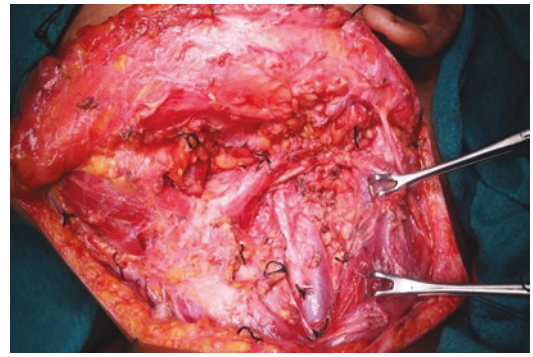

Fig. 17 Surgical field after removal of the submandibular gland

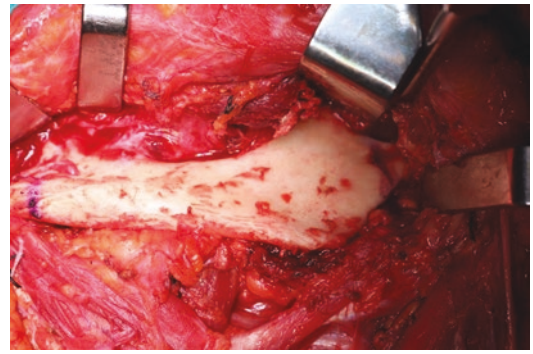

Fig. 18 Baring of the left hemi-mandible and marking for parasymphyseal mandibulotomy and horizontal mandibulotomy over the body of the mandible and ramus of mandible, respectively

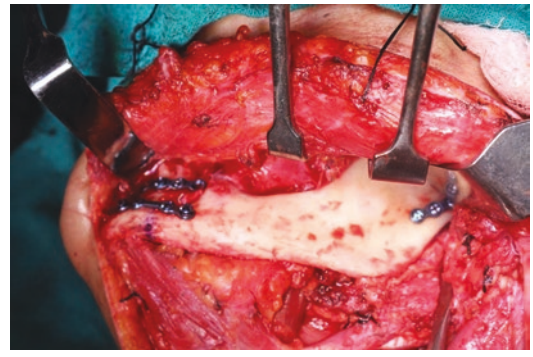

Fig. 19 Plates and screws are placed over the planned mandibulotomy site

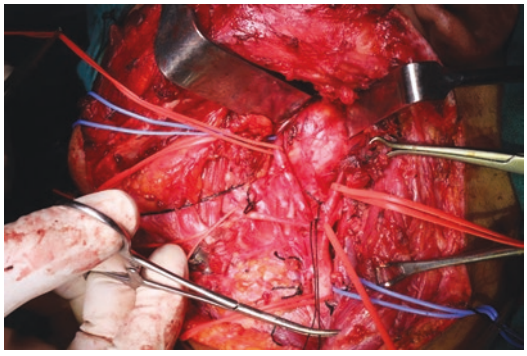

Fig. 20 Lifting mandibulotomy done to improve the access of the tumour; the tumour now can be accessed anteriorly and superiorly till the jugular foramen. Major vessels and nerves secured and isolated using vessel loops

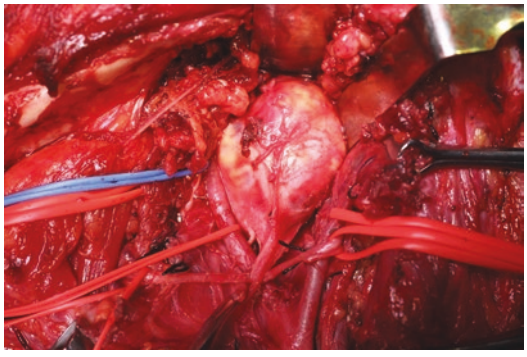

Fig. 21 The internal jugular vein is displaced laterally to facilitate dissection of the distal aspect of the vagus nerve

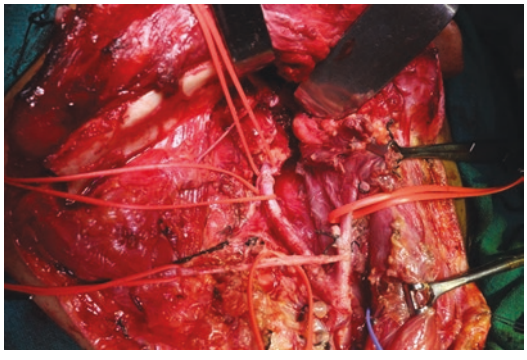

Fig. 22 After the removal of the tumour, the surgical field shows intact carotid bifurcation and adjacent cranial nerves

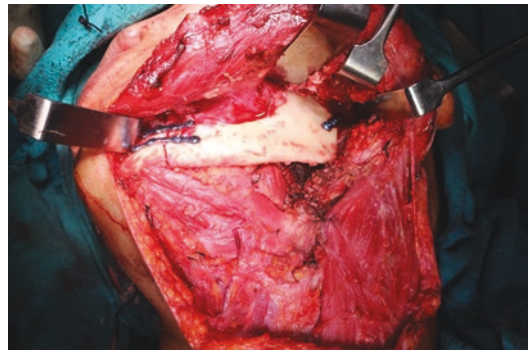

Fig. 23 Surgical wound after excision of the tumour and repositioning the mandible in place with plates and screws

Fig. 24 Gross specimen of the tumour after surgical excision of the lesion

Fig. 25 Surgical wound after closure

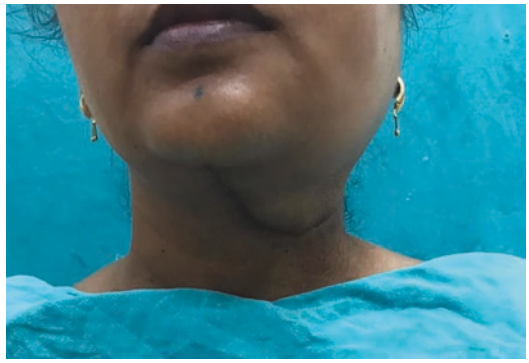

Fig. 26 Follow-up after 6 months with a well-healed scar on the left side of the neck

5 Thymoma

5.1 Introduction

Thymomas are uncommon tumours with a slow growth rate, making it simple to confuse them with benign growth [79]. Myasthenia gravis is associated with one-third of them, and in certain cases, removing them significantly improves the symptoms of the disease. A thymoma should be removed as soon as it is discovered due to its likelihood of being cancerous. Complete resection, the cornerstone of therapy, has a great cure rate [79].

Of the tumours in the anterior mediastinum, they make up 47% [80, 81]. There is a surge between the ages of 35 and 70, yet they affect persons of all ages. Thymoma affects both sexes roughly equally, while older women are somewhat more likely to get it [82]. Patients with myasthenia gravis are often a bit younger, peaking between the ages of 30 and 60 [79, 82].

Nearly 40% of patients experience local symptoms. They often include chest discomfort, a cough and shortness of breath brought on by myasthenia gravis' neuromuscular effects or airway constriction. Less frequently, rapidly expanding tumours can cause superior vena cava syndrome and weight loss [83]. Local symptoms are seen in around 40% of individuals. They often include coughing, shortness of breath and chest discomfort due to airway constriction or the neuromuscular consequences of myasthenia gravis. Rapidly growing tumours less frequently experience superior vena cava syndrome and weight loss [81, 84].

Thymomas first often present as well-defined round or oval lumps. Areas of low attenuation, many calcifications and uneven margins are indicators of an invasion [81, 85]. They can wrap around these structures but are often anterior to the major vessels [86]. The use of CT is crucial in determining whether a tumour is easily resectable or not, as well as if nearby tissues might also need to be removed. In addition, CT is helpful for identifying recurrence following the prior resection.

5.2 Surgical Resection

Given that thymoma has the potential to become cancerous, they should be removed in otherwise healthy people. It should be attempted to completely remove all thymic cells. In the surrounding tissue, thymic cells are located in little nests. Today, the majority of centres conduct an en-bloc excision of the whole thymus gland and the surrounding areolar tissue [79]. Every effort should be taken to perform a full resection, which has repeatedly been proven to be of substantial prognostic significance in the treatment of thymomas at every stage [79, 87]. It could involve the excision and repair of crucial organs like the aorta or superior vena cava. Even in individuals with myasthenia gravis, if one phrenic nerve is affected, it should be sacrificed unless the patient has a considerably diminished pulmonary reserve. For a successful resection, careful surgical planning is essential.

5.3 Operative Technique

The common method is a median sternotomy [79]. The whole thymus is removed, including all four lobes. Except for a little quantity surrounding the phrenic nerves, all pericardial fat should be eliminated. Any attached structure should be removed in its entirety rather than being just adjacent.

If excision of nearby structures such as the superior vena cava or phrenic nerve is necessary, an open approach is advised [88]. The sternotomy method of oncologically sound resection is substantially simpler.

5.4 Case 1

A 43-year-old woman with no prior history of breathing, swallowing or voice changes arrived with swelling in front of her neck on the left side for 3 months. A contrast-enhanced CT scan was performed on her, and the results revealed a well-defined hyperdense lesion in the lower region of her neck that had spread into her anterior mediastinum. A transcervical and sternotomy technique was then planned for the patient's excision.

Preoperative radiological imaging, intraoperative steps of surgery and wound closure images are shown in Figs. 27, 28, 29, 30, 31, 32, 33, 34, 35, and 36.

Fig. 28 Surface marking of the hyoid bone, skin incision and sternal notch with the superior part of the lesion on the left side of the lower part of the neck

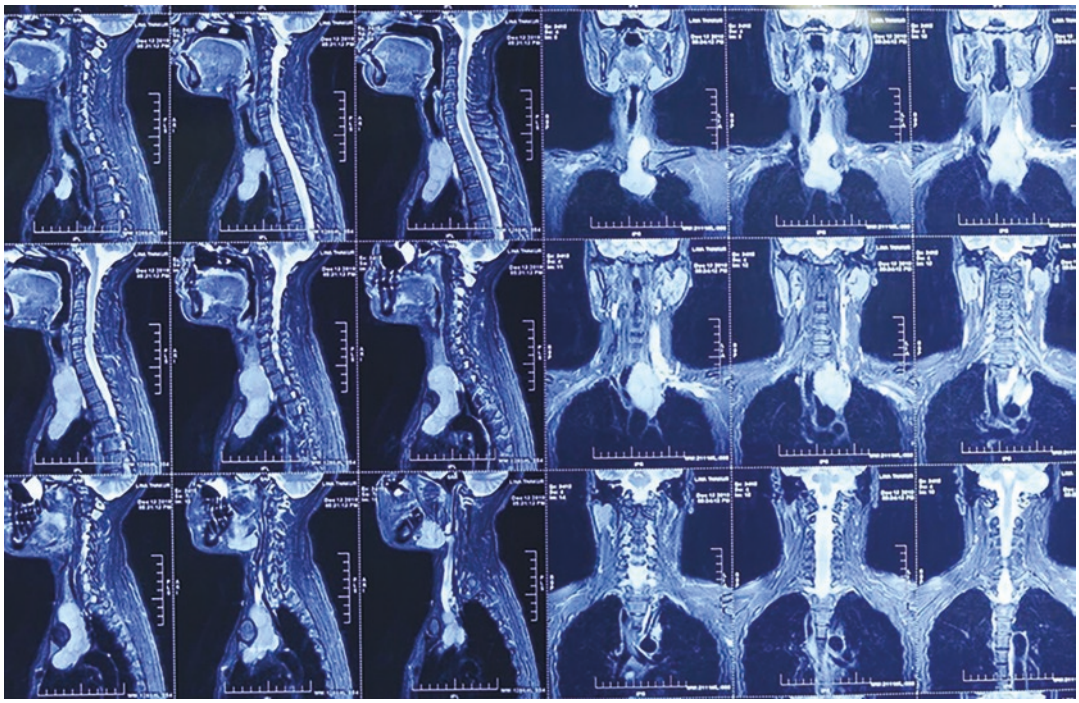

Fig. 27 Contrast-enhanced CT scan: coronal and sagittal section showing a well-circumscribed hyperdense lesion in the lower part of the neck with the lesion extending into the anterior mediastinum

Fig. 29 Surface marking of the sternotomy incision

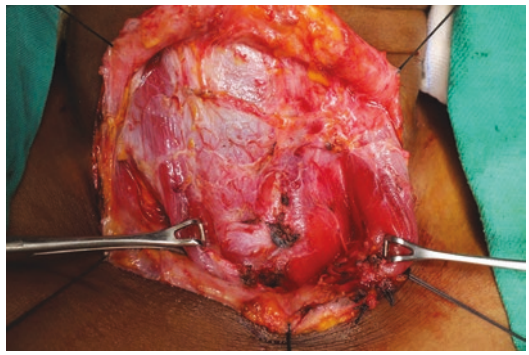

Fig. 30 Elevation of the upper and lower skin flap

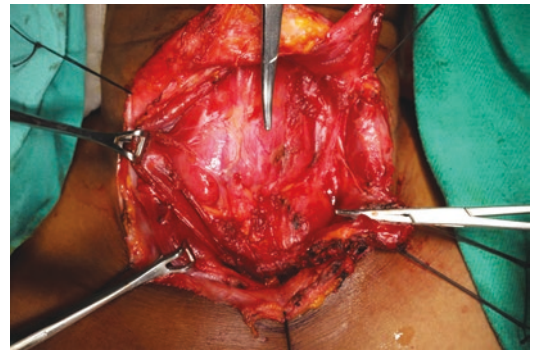

Fig. 31 Retraction of sternocleidomastoid muscles laterally to expose the anterior part of the neck

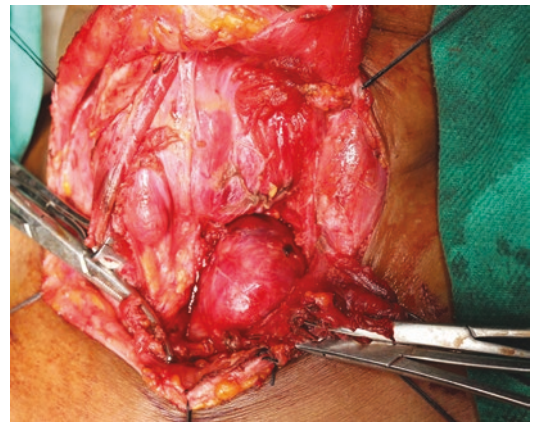

Fig. 32 Soft tissue dissection done to expose the superior part of the lesion which is present in the neck

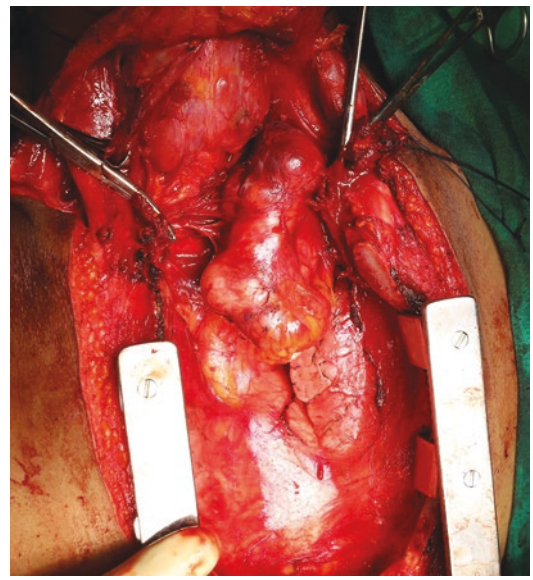

Fig. 33 Sternotomy done to expose the lesion which extends into the anterior mediastinum

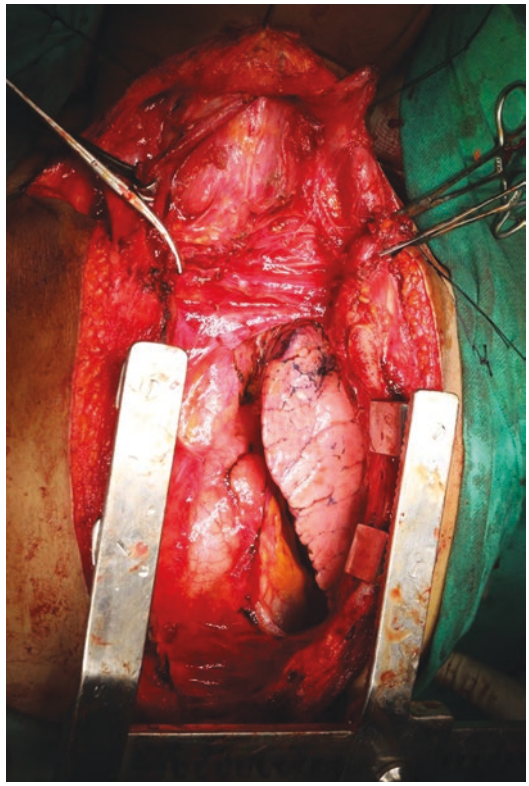

Fig. 34 Surgical field after excision of the lesion

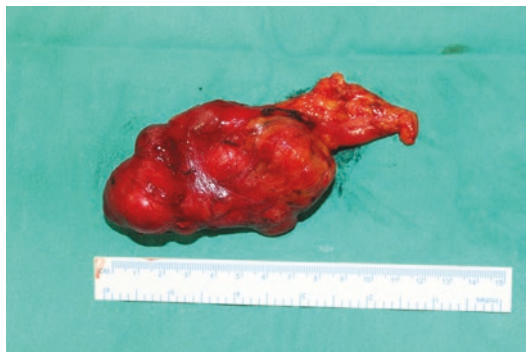

Fig. 35 Entire surgical specimen after excision

Fig. 36 Surgical wound after the closure of the neck and sternotomy wound

6 Branchial Cyst

6.1 Embryology

The branchial arches, which are divided apart externally by grooves and inside by pharyngeal pouches, are where the neck's tissues originate. The branchial arches' internal pouches, which are covered with the ectoderm on the exterior, are made of foregut endoderm. The interlayer region contains the tissue that is developed from the mesoderm [89]. When two nearby arches are poorly or inadequately fused, a branchial cleft anomaly results [90]. Fistulas (an open tract between the skin and throat), sinuses (connecting the skin or pharynx to a blind pouch in the neck) and cysts are a few examples of these abnormali-

ties (isolated epithelial-lined structures lacking connection to the skin or pharynx). Their positions vary depending on the arch from which they emerge and how close they are to muscles, arteries and nerves [91].

6.2 First Arch Anomaly

The first branchial arch accounts for 8% of the cervical sinus tracts and cysts [92]. Despite the fact that some people with these first arch anomalies show no symptoms, others complain of neck, parotid or ear pain. Around the mandibular angle, there might be a pit-like depression that, if infected, could lead to drainage.

6.2.1 Type 1 First Arch Cyst

Type I first arch cysts typically open in the pre- or postauricular areas. The sinus tract may adhere to the tympanic membrane or the skin of the external auditory canal. The external auditory canal and the sinus tract normally run side by side [89, 90]. Type I lesions are lateral to the facial nerve.

6.2.2 Type II First Arch Cyst

Type II first arch cysts, on the other hand, appear medial to the facial nerve [89]. They are located in the anterior neck, above the hyoid. The type II first arch sinus typically goes via the parotid gland and encircles the facial nerve as it moves anterior to the hyoid [90].

6.3 Second Arch Anomaly

The most common type of cervical cyst, second branchial arch cysts, accounts for 90 to 95% of all cases [89, 90]. If these anomalies come from the second branchial pouch, they may lead to fistulous tracts that connect the palatine tonsil to the lateral neck skin at the anterior edge of the sternocleidomastoid muscle. Examination reveals that there is a skin pit in this location. As they approach the supratonsillar fossa, they can alternatively pass near to the glossopharyngeal and hypoglossal nerves if they originate from the second branchial cleft. If these pits are bilateral, it is important to check the newborn for branchio-oto-renal syndrome since it may potentially indicate kidney and hearing issues. These cysts can occasionally be detected near to the carotid sheath, between the internal and external carotid arteries [89]. If a skin pit is present, it is at the anterior edge of the sternocleidomastoid muscle. Patients, usually suffering from an upper respiratory tract infection, may develop acute cyst enlargement or superinfection. Depending on its location and size, this enlargement may result in dysphagia, torticollis or a compromised respiratory system [89].

6.4 Third and Fourth Arch Anomaly

Cysts, which are incredibly uncommon, make for less than 2% of anomalies in the third and fourth branchial arches [90]. Before terminating in the pyriform fossa, the sinus tracts of these cysts frequently penetrate far into the thyroid gland and anterior cervical structures. If children develop secondary infections, these problems may present in the form of suppurative thyroiditis in young children. If they spread fast due to infection, they can potentially result in tracheal constriction or airway compression in young infants [89].

The mainstay of treatment for all of these cysts is surgical removal. Preoperative imaging is often used to plan the surgical approach and anticipate potential problems. Sinuses and cysts that are left untreated present a high risk of infection. Greater recurrence rates might also result from incomplete resection [89]. Certain surgical safety precautions can be necessary due to their anatomical positioning. For instance, in order to thoroughly remove the cyst and sinus and avoid damaging the facial nerve, a superficial parotidectomy may be required for type II first branchial arch cysts. Finding the internal sinus entry, which is often on the left side, for the third and fourth branchial arch cysts requires endoscopic assessment of the pyriform fossa [90]. Ipsilateral hemithyroidectomy is required for the fourth arch anomalies in order to remove the whole tract [89].

6.5 Case 1

A 30-year-old female presented with swelling on the left side of her neck since childhood; the swelling is non-progressive in size with no compressive symptoms. On examination, a swelling of 4 × 3 cm is present on the left side of the neck along the anterior border of the neck in the middle one-third. On FNAC, aspirates yield abundant viscous fluid, which contains mature squamous cells and keratinous debris. Further characterization of the swelling was done by contrast-enhanced CT scan. The patient was posted for excision of the lesion.

Preoperative radiological imaging, intraoperative steps of surgery and surgical specimen images are shown in Figs. 37, 38, 39, 40, 41, 42, and 43.

Fig. 37 Surface marking of the swelling along with marking the external jugular vein

6.6 Case 2

A 22-year-old female presented with swelling on the right side of her neck since childhood; the swelling is non-progressive in size with no compressive symptoms. On examination, a swelling of 7 × 6 cm is present on the right side of the neck along the anterior border of the neck in the middle one-third. On FNAC, aspirates yield abundant viscous fluid, which contains mature squamous cells and keratinous debris. Further characterization of the swelling was done by contrast-enhanced CT scan. The patient was posted for excision of the lesion.

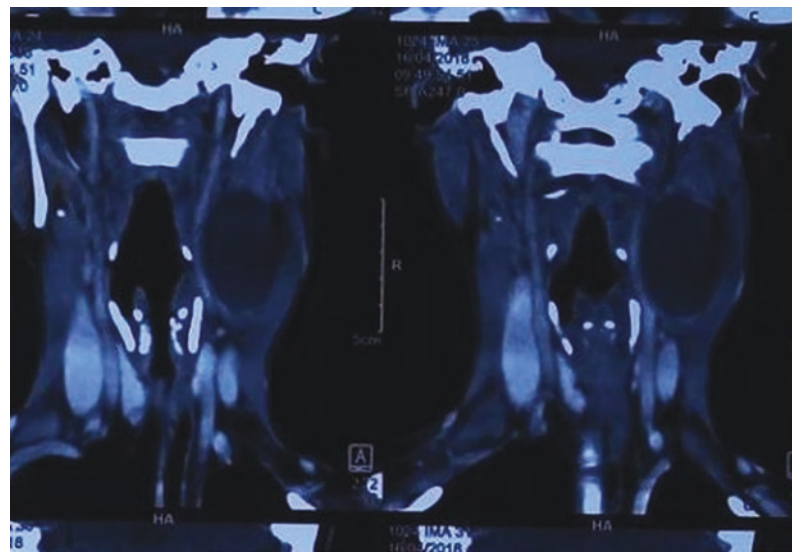

Fig. 38 Contrast-enhanced CT scan coronal section showing a homogenous hypodense lesion

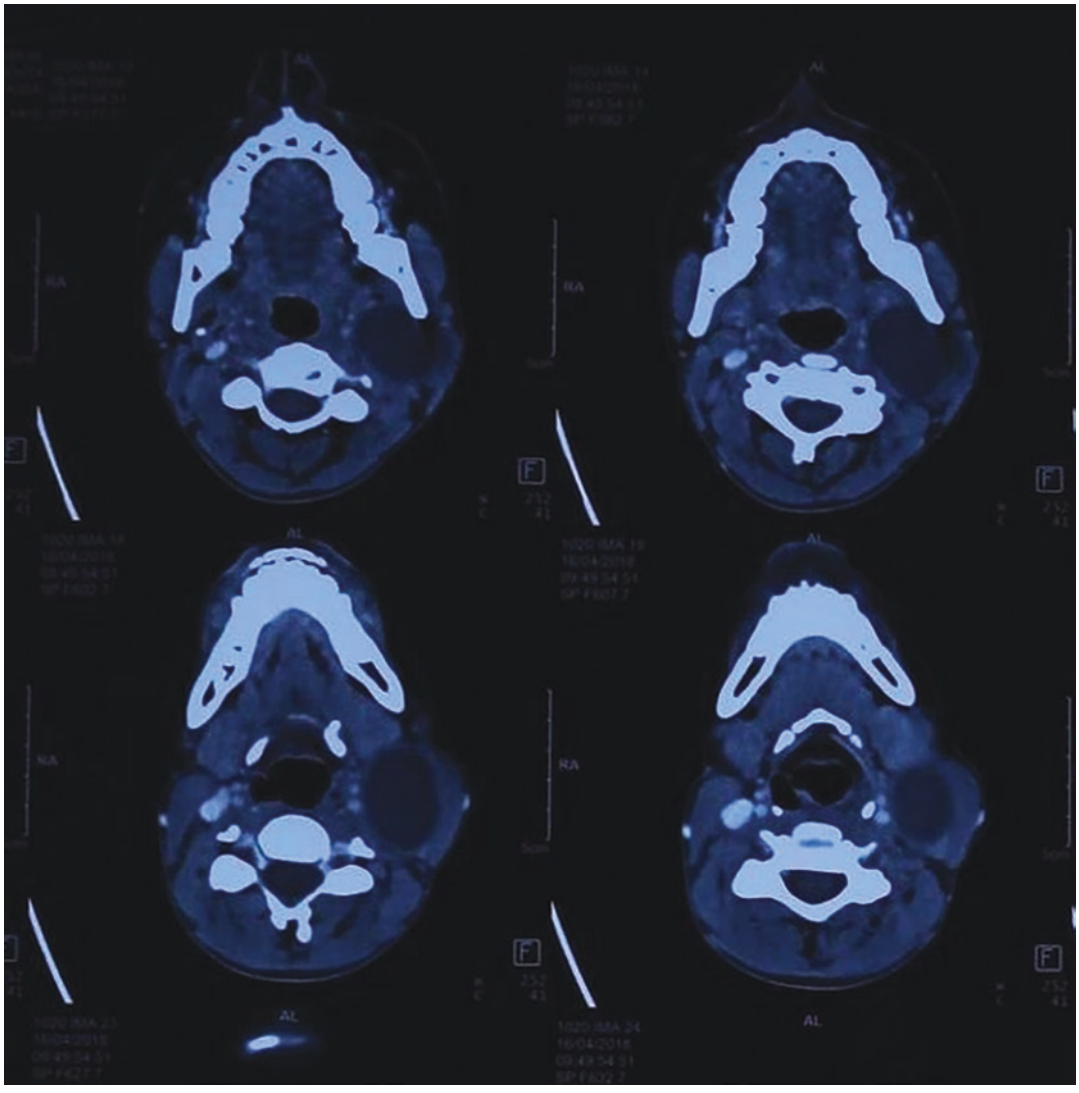

Fig. 39 Contrast-enhanced CT scan axial images showing a well-defined hypodense lesion in the left side of the neck

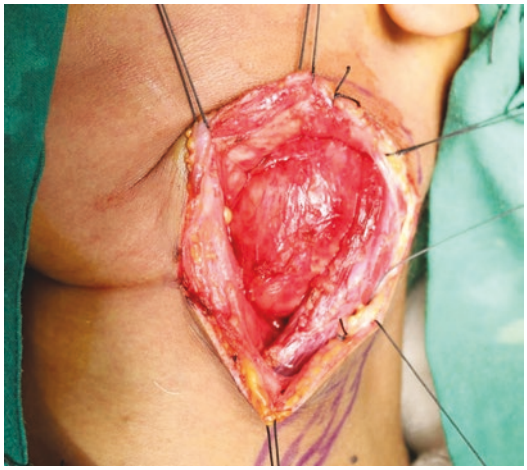

Fig. 40 Elevation of the upper and lower skin flaps with exposure of the anterior border of the sternocleidomastoid muscle

Fig. 41 Delivery of the smooth-walled cystic swelling

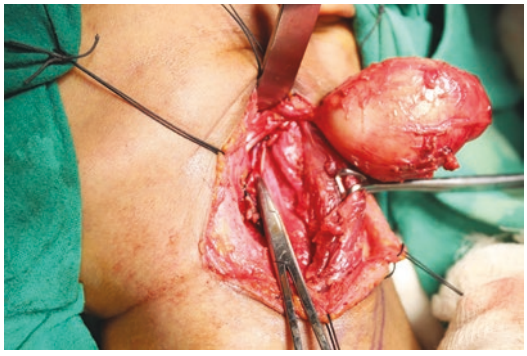

Fig. 42 Surgical bed after excision of the lesion, which shows the preserved underlying structures

Fig. 43 Excised branchial cyst

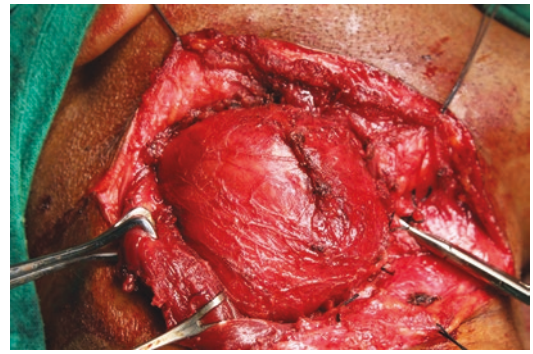

Fig. 44 A smooth-walled cystic swelling along the anterior border of the right sternocleidomastoid muscle

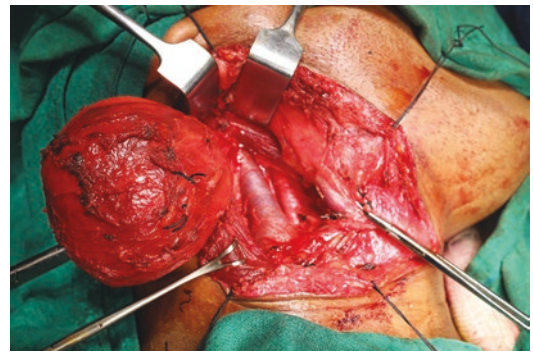

Fig. 45 Surgical bed showing the underlying preserved IJV and carotid

Intraoperative steps of surgery and surgical specimen images are shown in Figs. 44, 45, and 46.

Fig. 46 Excised branchial cyst

7 Lymphangioma

Congenital lymphatic spongy multiloculated cystic lesions are known as lymphangiomas. When a significant lesion exhibits a clear deformity or when a lesion compromises function due to encroachment into the airway or swallowing passageways, excision of these lesions is suggested. Despite the fact that the cause is yet unclear, these lesions could be congenitally present and not show up clinically until much later in life, when they might start to enlarge.

For full excision of the lesion without leaving any loculi or parts of the cyst wall behind, radiological imaging using an MRI scan is essential in these lesions to determine the extent of numerous loculations interdigitating inside muscle planes.

Operative Procedure

- A transverse incision which is placed over the upper part of the neck.
- Upper and lower skin flaps are lifted, and the skin incision is deepened till the level of the platysma.
- It is essential to be careful and cautious while lifting the skin flaps to prevent rupturing the thin walled cystic lesion.
- The multiloculated cystic process is difficult to dissect when the lesion ruptures, and this frequently results in partial excision and recurrence.
- Therefore, great care should be used to maintain the cyst wall's integrity in order to enable a full excision.
- The loculated lesion is seen as the dissection moves further. The cystic lesion is then meticulously dissected in tissue planes around it, progressively mobilizing each of its loculi and bringing them into the surgical area.
- The lesion is removed in one piece once all of its loculi have been removed.
- The skin incision is closed in stages, and suction drains are installed.
- The surgical specimen reveals a multiloculated cystic tumour that was completely excised, resulting in its permanent management.
- For long-term management, it is crucial to pay close attention to every lymphangioma loculation that has to be removed. If the cyst lining is not removed completely, the lymphangioma may return locally.

7.1 Case 1

The patient in the image has a sizable cystic lesion that extends to the parapharyngeal borders and takes up much of the upper section of the neck. The anterior triangle of the top part of the right neck is affected by a well-defined multiloculated cystic lesion in the T2-weighted MRI scan's axial view. The cystic lesion's bright white colour denotes the presence of fluid.

Preoperative radiological imaging, intraoperative steps of surgery and surgical specimen images are shown in Figs. 47, 48, 49, 50, 51, 52, 53, and 54.

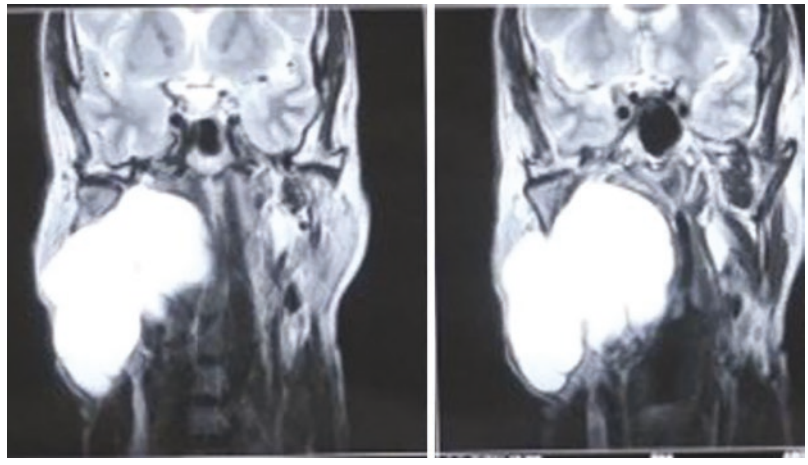

Fig. 47 MRI scan: coronal cuts showing large hyperintense lesion in the right parapharyngeal space

Fig. 48 Planned modified Blair/Bailey incision in the right side of the neck for adequate exposure

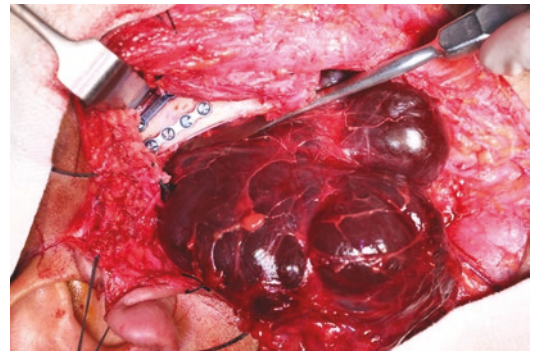

Fig. 50 Exposure of the part of lymphangioma which was present in the neck and plating done at the planned mandibulotomy site

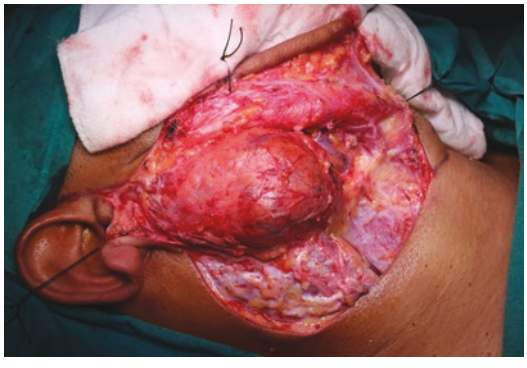

Fig. 49 Elevation of the skin flap for exposure of the lesion

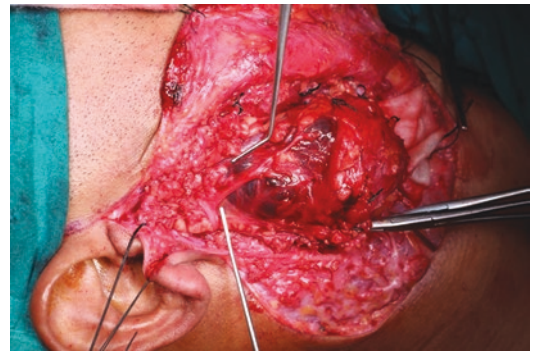

Fig. 51 Complete exposure of the lesion in the neck and in the paramandibular region, medial to the mandible

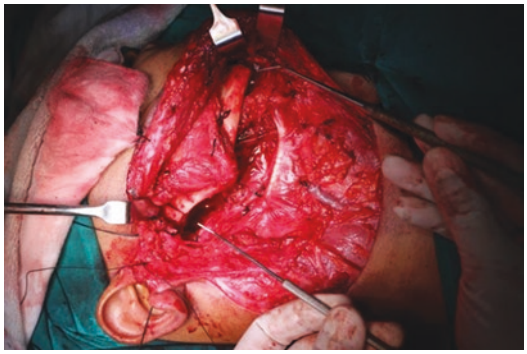

Fig. 52 Mandibulotomy cuts: horizontal over the ramus of the mandible and vertical cut over the body of the mandible in the parasymphyseal region

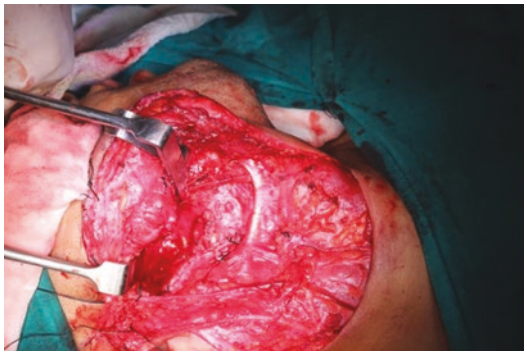

Fig. 53 Surgical site after complete excision of the lesion

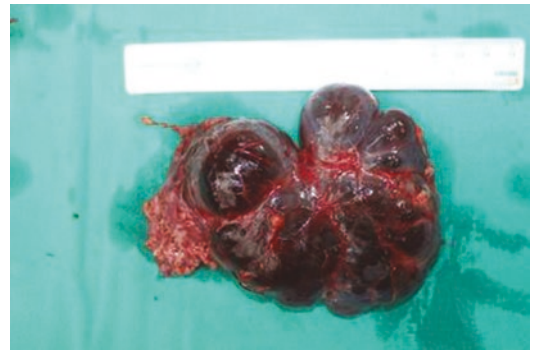

Fig. 54 Surgical specimen in toto after complete surgical excision showing a multiloculate specimen

8 Neurofibromatosis

Preoperative radiological imaging, intraoperative steps of excision of neurofibromatosis and surgical specimen images are shown in Figs. 55, 56, 57, 58, 59, 60, 61, 62, 63, and 64.

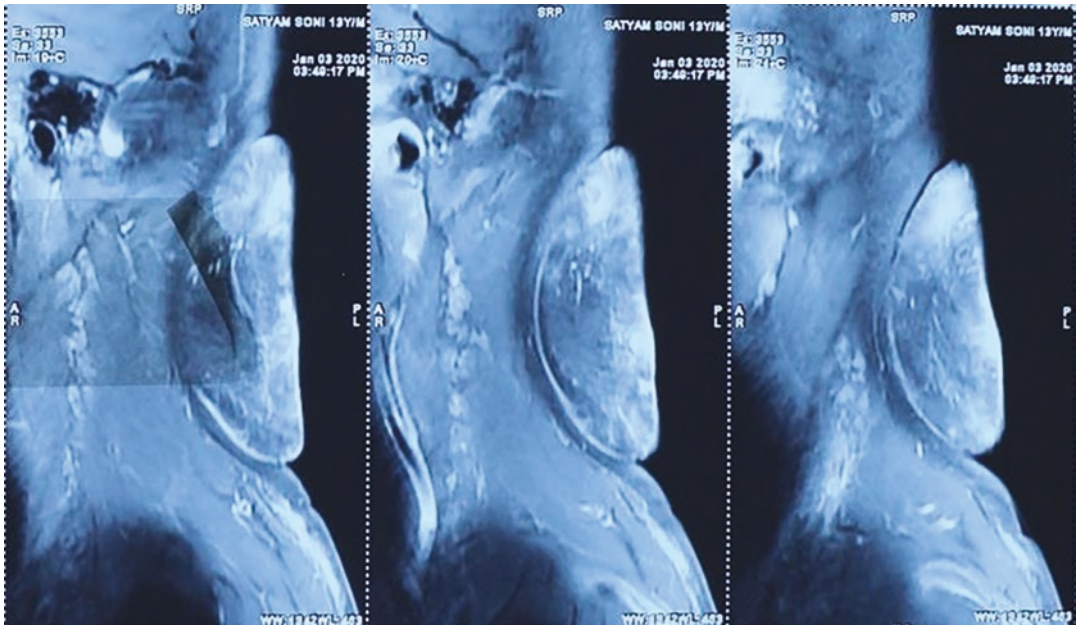

Fig. 55 MRI scan sagittal cuts showing lesion in the nape of the neck

Fig. 56 MRI scan coronal cuts

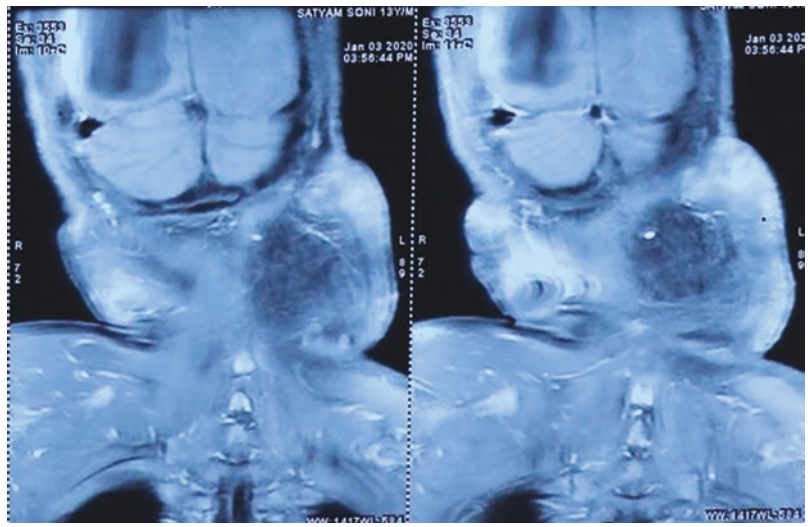

Fig. 57 Multiple cutaneous neurofibromatosis lesions present in the bilateral malar region

Fig. 58 Large neurofibroma lesion present in the nape of the neck

Fig. 59 Soft tissue dissection and complete excision of the lesion from its bed

Fig. 60 Surgical site after complete excision of the lesion

Fig. 61 Surgical specimen after excision

Fig. 62 Upper surface of the surgical specimen

Fig. 63 Inferior surface of the surgical specimen

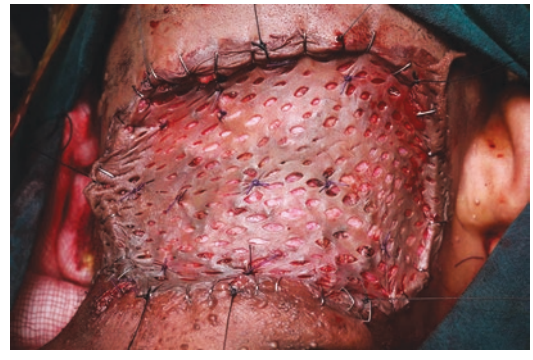

Fig. 64 Split-thickness skin graft over the surgical sit

9 Nasal Dermoid

The most typical location for dermoid cysts, which are benign tumours of neuroectodermal origin, is the anterolateral frontozygomatic suture [93]. The anterolateral frontozygomatic suture is the most typical site for dermoid cysts, benign tumours of neuroectodermal origin [94, 95]. Gliomas, encephaloceles, epidermoid cysts and haemangiomas are among the possible differential diagnosis for midline nasal tumours [96, 97]. The dura is in touch with the skin in the early embryo and separates when the frontal bone grows in between them, according to the most commonly accepted cranial hypothesis. Dermoid cyst development results from this process' inability to achieve separation [98, 99]. Since intracranial extension may be present in certain nasal dermoid cysts; it has been highlighted how crucial a proper preoperative diagnosis is [100]. In this respect, preoperative computed tomography or magnetic resonance imaging studies may be necessary for dermoid cysts with intracranial extension [101].

Intraoperative transillumination, steps of surgery, surgical specimen and follow-up images are shown in Figs. 65, 66, 67, 68, 69, and 70.

Fig. 65 Skin marking for mid-columellar inverted V incision

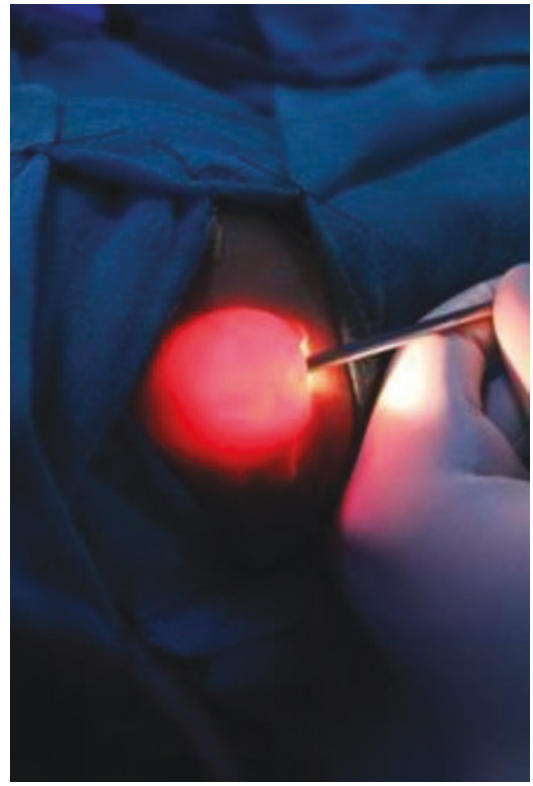

Fig. 66 A brilliant transilluminating dermoid lesion

Fig. 67 The soft tissue envelope is further reflected superiorly in a relatively avascular supra-perichondrial plane to expose the dermoid till the upper lateral cartilage

Fig. 68 Surgical bed after excision of the lesion

Fig. 69 Excised dermoid cyst

Fig. 70 Post-op follow-up after 2 months

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Plastic and Reconstructive Surgery

Karthik N Rao, Rupa Mehta, and Ambesh Singh

1 Principles of Head and Neck Reconstruction

The problem of reconstructing abnormalities in the head and neck is distinct. In contrast to other portions of the body that can occasionally be temporalised by dressing changes or even let to heal by secondary purpose without incurring major repercussions, the immediate or early closure of head and neck deformities is essential for a variety of reasons. It is crucial to preserve the patient's ability to eat and the health of their digestive system. The face must be reconstructed in order for a person to express themselves through facial expression. Finally, by covering the neck and preserving an unbroken intraoral seal, life-threatening issues like blowout of the major vessels in the neck are prevented. Here, we provide a decision-supporting algorithm for head and neck reconstruction.

When reconstructing a head and neck deformity, it's crucial to think about the reconstruction's objectives and if the alimentary canal, the face and the neck should be repaired first or if it's possible to also restore the function to the head and neck's organs in addition to form. Especially if they have had cancer surgery, many patients who need reconstruction may not be in the best of physical shape. All patients should have their alimentary tract, face and neck integrity restored as a priority since problems like orocutaneous fistulas can impair their ability to eat and can result in life-threatening scenarios like a carotid artery blowout. Although free tissue transfer is the method of reconstruction that is preferred, in some circumstances, using a straightforward pectoralis major pedicled flap to fill a hole or restore continuity in the pharynx or oesophagus may be sufficient to achieve the goal of restoring the integrity of the alimentary tract for the majority of patients. In skull base restoration, it is vital to restore the integrity of the separation between the cerebral contents and the sinuses and alimentary system. Additionally, in order to avoid meningoencephalitis and cerebrospinal fluid leak, it's crucial to employ the vascularised tissue to cover any empty spaces.

It is crucial to think about regaining function in the head and neck region if the patient can have significant surgery. This area is in charge of several important processes, including speaking, chewing, swallowing and expressing facial expressions. For instance, since the defect can be directly repaired, reconstructing a partial glossectomy defect is not necessary for the patient's survival. The neotongue can contact

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_12

the palate when the bulk of the tongue is restored through free tissue transfer, though, which can make swallowing easier. Another illustration is the rehabilitation of speech using methods like free ileocolon flaps or tracheoesophageal punctures. While a person can survive following total laryngectomy without this operation, recovering speech can improve communication and quality of life. Bony deficiencies in the mandible must be repaired with vascularised bone in order to preserve function and make room for the installation of dental implants. Restoring both function and form should be the aim of head and neck restoration. There are techniques for repair that only involve soft tissue, but by restoring the facial buttresses and enabling oral rehabilitation, utilising a segmented free fibular flap can offer both functional and aesthetic benefits. In the restoration of the soft tissue defect following total parotidectomy, a de-epithelialised parascapular free flap is used yet another time. Facial nerve repair after a complete parotidectomy promotes smile restoration and prevents the stigma associated with nerve palsy. On a more limited scale, optimising the cosmetic outcome after surgery is made possible by designing the reconstruction while taking into account the aesthetic subunits of the face and maintaining these boundaries. The best aesthetic outcome is achieved when a defect is replaced with the tissue that is equivalent in delicate areas like the lip and nose. Reconstructive surgery's objectives can include restoring integrity, function and shape, but they can also change based on the patient's health, coexisting conditions and preferences. To assess the risks of anaesthesia and surgery and to decide the best course of action, a complete preoperative evaluation is required. A pedicled locoregional flap may be utilised to restore integrity alone in patients with significant coronary artery disease or other medical conditions that prevent them from receiving a free tissue transfer. A young, healthy patient with a benign tumour, on the other hand, might be an excellent candidate for a reconstruction that takes into account the three objectives of integrity, function and shape. The unique needs of the patient and the nature of the defect, including the types and volume of the tissue involved, will determine the selection of the reconstruction strategy. Multiple skin paddles could be required in some situations to cover both intraoral and extraoral flaws.

The various algorithms for reconstruction of various head and neck defects are given in Figs. 1, 2, 3, 4, 5, 6, 7, 8 and 9.

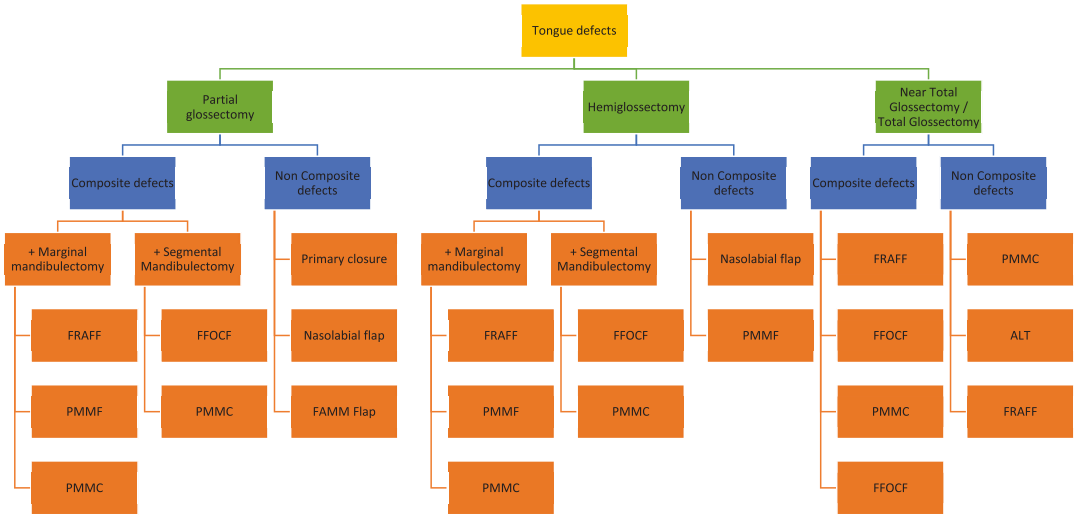

Fig. 1 Reconstructive algorithm for the tongue defects

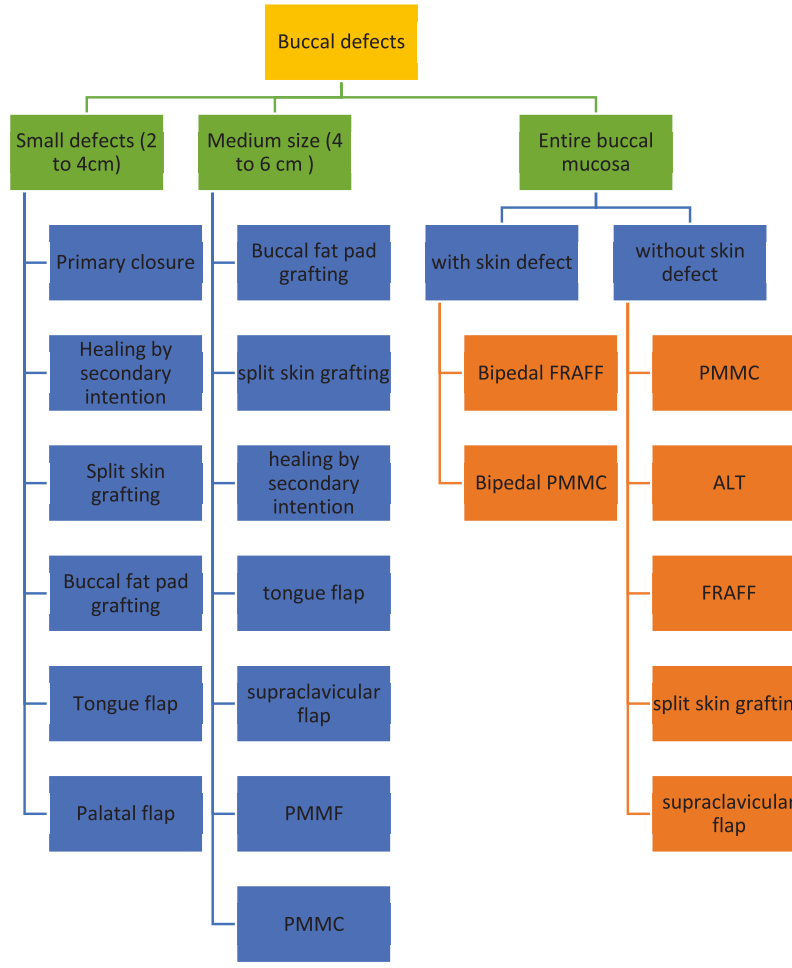

Fig. 2 Reconstructive algorithm for the buccal mucosa-only defects

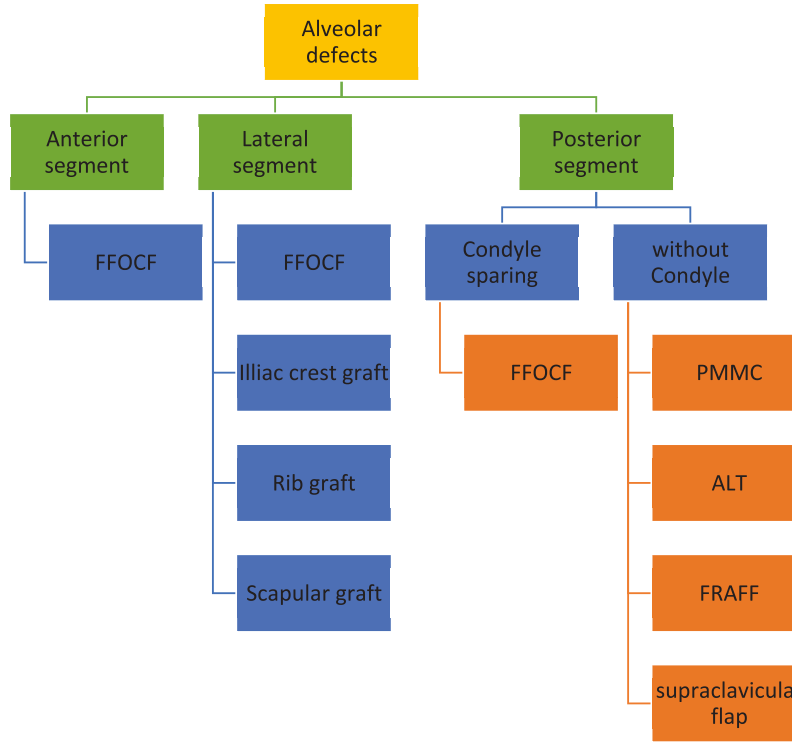

Fig. 3 Reconstructive algorithm for the alveolus-only defects*With cutaneous defect - soft tissue flap for cover – PMMC/DP/ Scalp flap/forehead flap/ second free flap – FRAFF/FALT

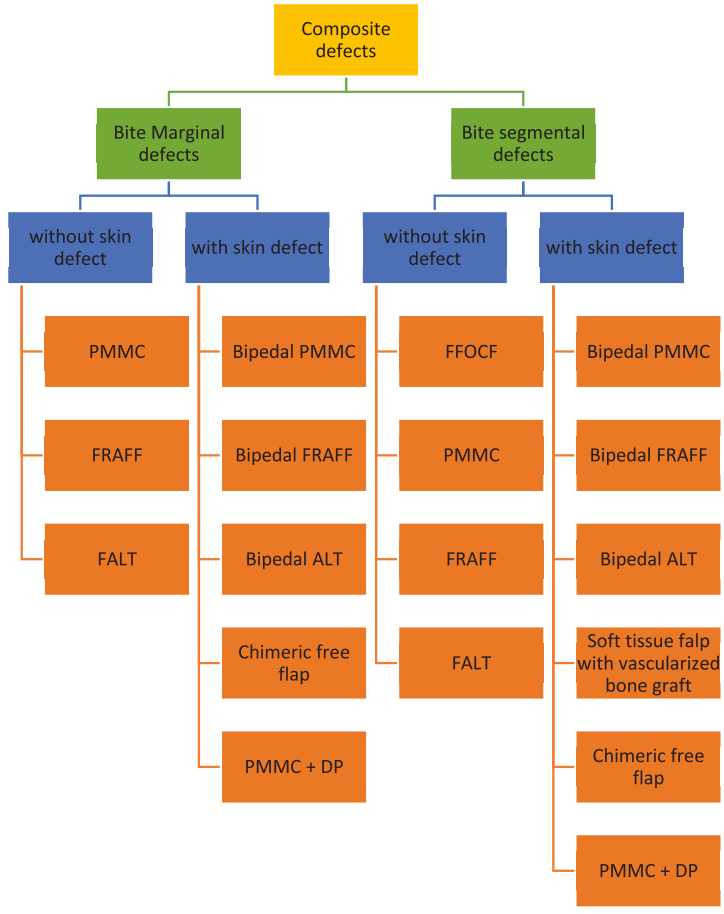

Fig. 4 Reconstructive algorithm for the composite buccal and alveolar defects

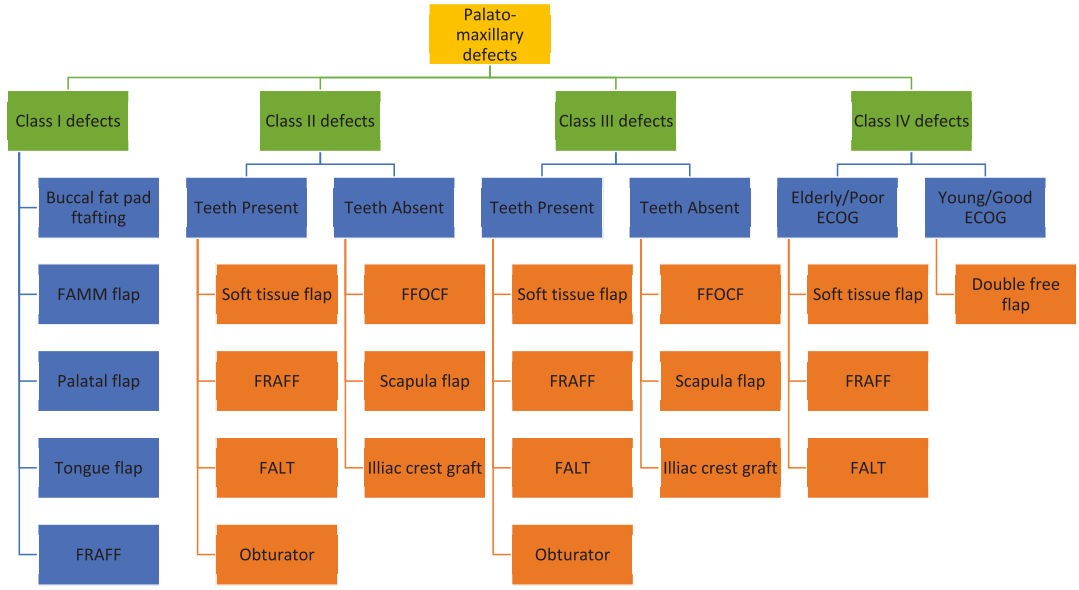

Fig. 5 Reconstructive algorithm for the palate and maxillary defects

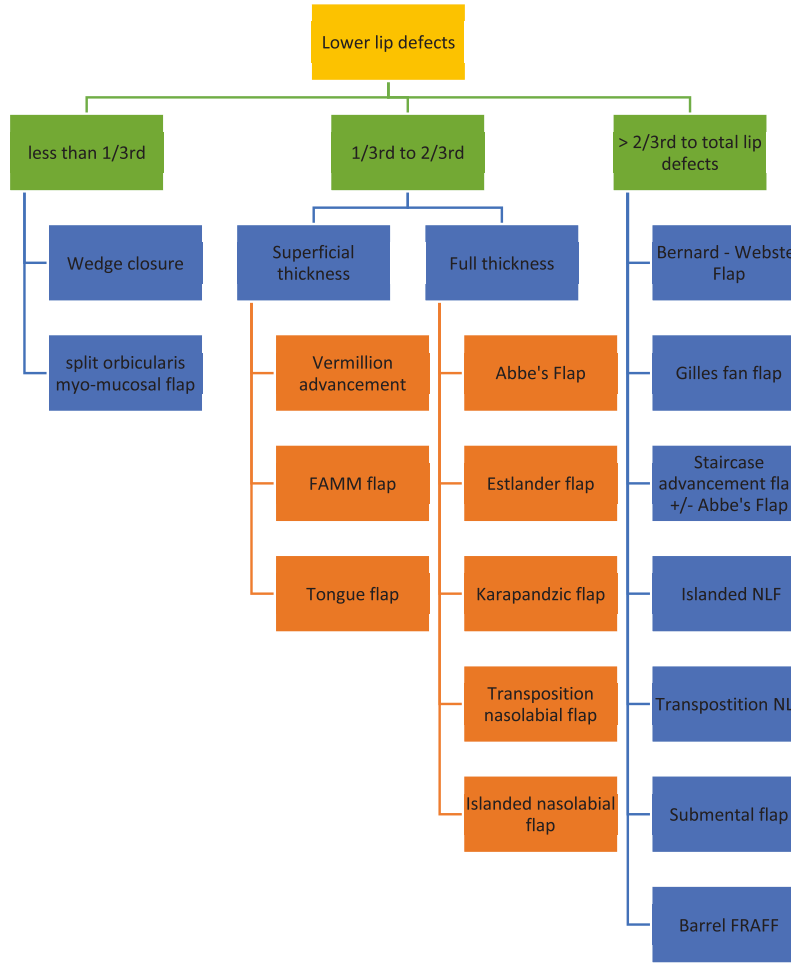

Fig. 6 Reconstructive algorithm for the lower lip defects

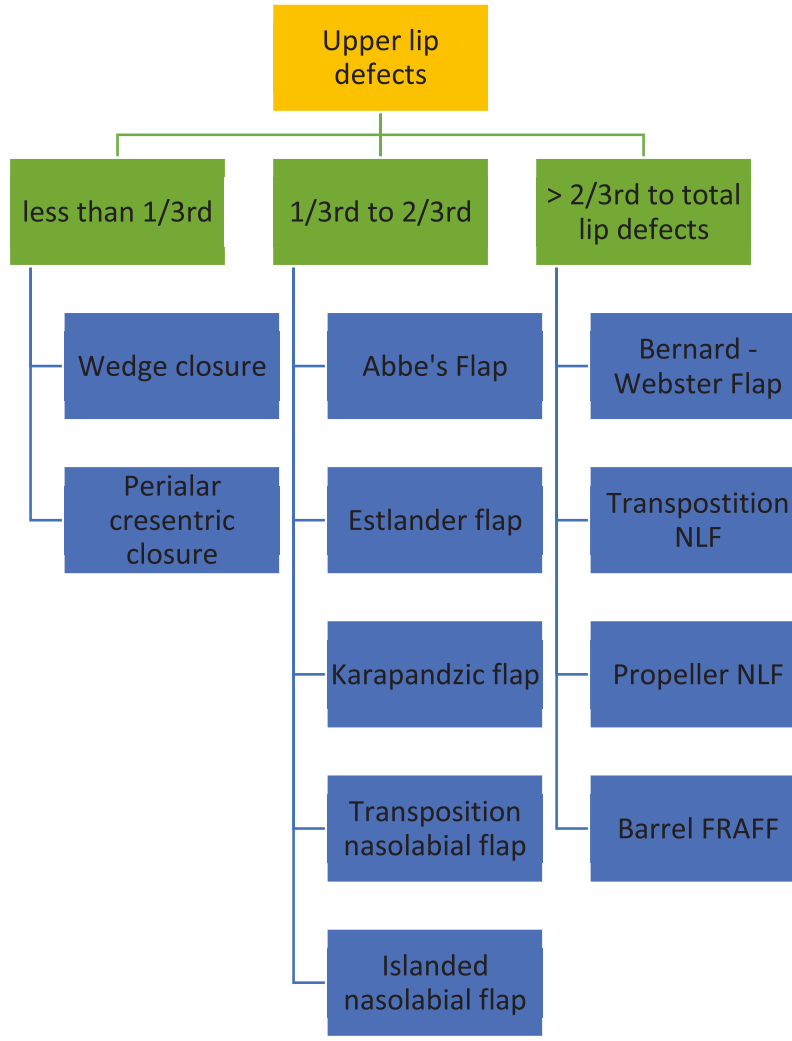

Fig. 7 Reconstructive algorithm for the upper lip defects

Fig. 8 Reconstructive algorithm for partial pharyngeal defects

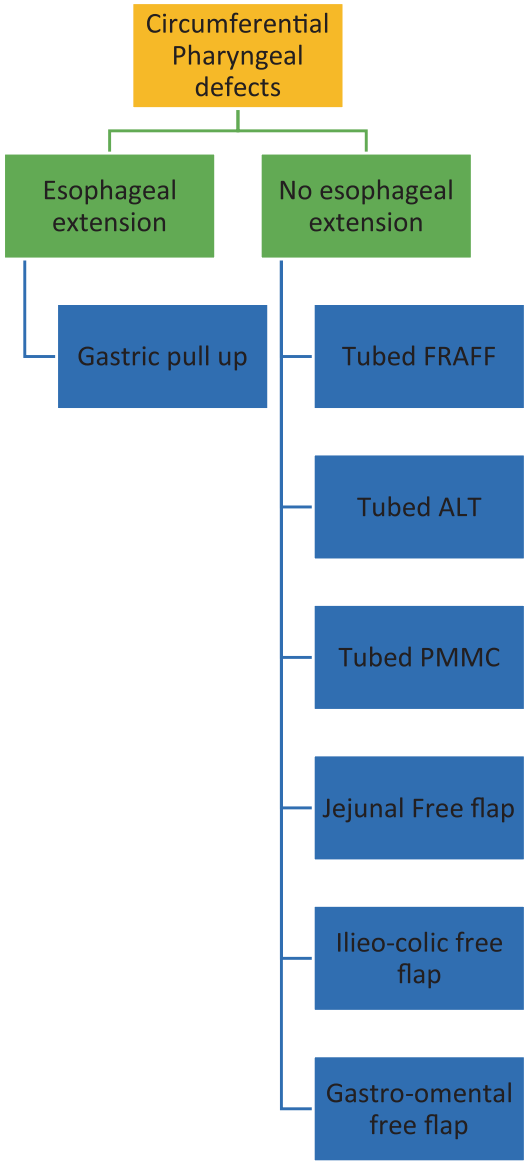

Fig. 9 Reconstructive algorithm for circumferential pharyngeal defect

2 Reconstructive Algorithm for the Tongue Defects

3 Reconstructive Algorithm for the Buccal Mucosa-Only Defects

4 Reconstructive Algorithm for the Alveolus-Only Defects

5 Reconstructive Algorithm for the Composite Buccal and Alveolar Defects

6 Reconstructive Algorithm for the Palate and Maxillary Defects

7 Reconstructive Algorithm for the Lower Lip Defects

8 Reconstructive Algorithm for the Upper Lip Defects

9 Reconstructive Algorithm for Partial Pharyngeal Defects

10 Reconstructive Algorithm for Circumferential Pharyngeal Defects

The reconstruction of complex head and neck defects is challenging as it provides both aesthetic and functional outcomes following the reconstruction.

The defects of maxilla apart from affecting the functions of the speech, swallowing and mastication also cause cosmetic disfigurement. The various maxillary reconstruction methods have been dealt with in detail by Iyer et al. [1].

Pharyngeal reconstruction is a challenging aspect of reconstruction after resections for head and neck cancer. The goals of reconstruction are to restore the continuity of the pharyngeal passage to enable oral alimentation and rehabilitation of speech wherever possible. Pharyngeal reconstruction is dealt with in detail by Balasubramanian et al. [2].

For reconstruction of the complex head and neck defects, the free flaps are used, and it has been dealt with in detail in the literature [3–8].

Barclay et al. [9] summarise commonly encountered functional and aesthetic issues that the restorative dental team is facing and highlight widespread preventive challenges.

Yadav et al. [10] summarised in detail the various reconstructions of the head and neck defects.

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Adjuvant Therapy

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Stage III/IV resectable locally advanced head and neck squamous cell carcinoma (HNSCC) has a dismal prognosis in most cases. Positive microscopic resection margin, multiple cervical lymph node metastases (2), lymph node metastases with a diameter of 3 cm or more, perineural invasion, Level 4 (inferior internal jugular lymph node) or Level 5 (accessory nerve lymph node) lymph node metastasis in oropharyngeal or oral cavity cancer and signs of vascular tumour embolism are risk factors for recurrence. For patients without these risk factors, the 5-year local relapse rate is only 10%, and post-operative adjuvant treatment is typically not recommended. Patients with extracapsular nodal extension or two or more recurrence risk factors, however, had a dismal prognosis even with post-operative radiation, with a 5-year local relapse rate of 32% and a 5-year survival rate of 42%. As a result, more effective treatment options are being sought because surgery with post-operative radiotherapy is thought to be insufficient.

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1 Adjuvant Chemoradiotherapy

1.1 RTOG 85-03 Study and RTOG 88-24 Study

- According to the findings, patients with certain recurrence risk factors, such as a microscopically positive resection margin, extracapsular nodal extension and multiple lymph node metastases (2), had a higher 5-year local relapse rate (61% for a positive resection margin, 27% for extracapsular nodal extension/multiple cervical lymph node metastases and 17% for no relapse risk factors), as well as a lower 5-year. It is therefore important to improve the prognosis in patients with any of these three risk factors for recurrence [1].

1.2 EORTC 22931 Study

Results showed that certain factors can increase the risk of recurrence in patients with head and neck cancer, including a positive microscopic resection margin, extracapsular nodal extension, stage III/IV disease, perineural infiltration, Level 4/5 lymph node metastasis in oropharyngeal or oral cavity cancer and signs of vascular tumour embolism.

Despite some differences in the definition of post-operative risk factors for recurrence between

N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_13

the EORTC [2] and RTOG [1], the two key trials (Table 1). To account for these differing definitions, data from the two studies were consolidated in a combined analysis [3].

Therefore, major high-risk factors for recurrence are presently defined as (a) microscopic resection margin positivity and (b) extracapsular nodal extension positivity, and patients with either of these major risk factors should receive post-operative CRT.

Table 1 Differences in risk factors for recurrence between RTOG and EORTC

Risk factor only in RTOG	Risk factors only in EORTC	Common risk factors in RTOG and EORTC
1. Multiple lymph node metastases (≥2)	1. Stage III/IV disease 2. Perineural infiltration 3. Level 4/5 lymph node metastasis in oropharyngeal cancer/oral cavity cancer 4. Vascular tumour embolism	1. Microscopic resection margin positivity 2. Extracapsular nodal extension positivity

2 Adjuvant Radiotherapy Alone

For patients with resectable locally advanced head and neck malignancies, the major treatment objectives are to find a cure and to maintain functional status through organ preservation. For the majority of patients with stage III or stage IV head and neck cancer that may be resectable in the past, surgery combined with post-operative radiotherapy (PORT) has been the recommended course of treatment. Patients who have advanced main (T3 or T4) or nodal (N2 or N3) or perineural illness, affected surgical margins, perineural involvement, bone or cartilage invasion or both may benefit from PORT. However, there have been no randomised controlled trials to test the efficacy of PORT with surgery alone. The linear accelerator used in our institute is shown in Figs. 1 and 2.

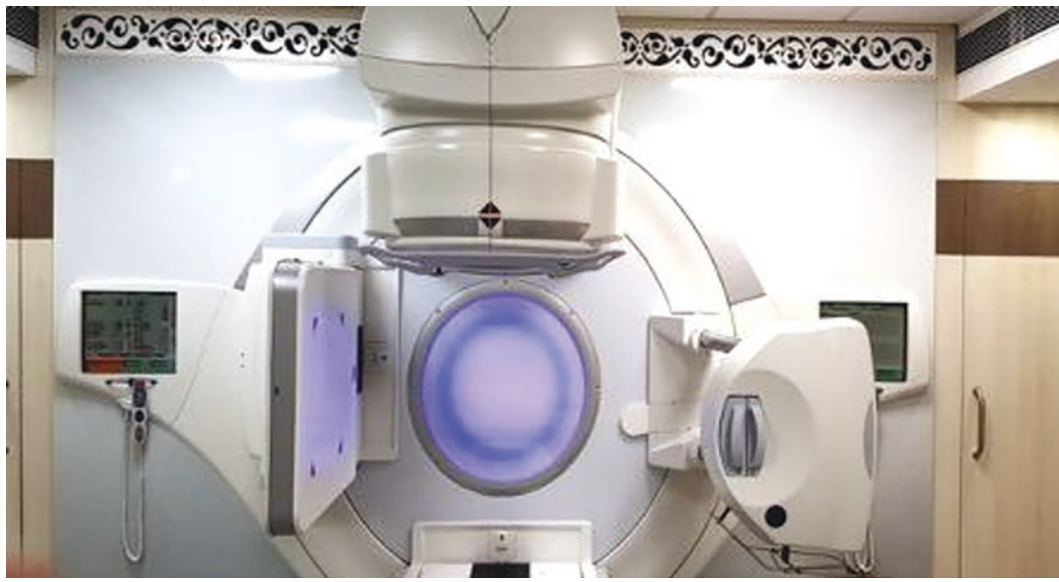

Fig. 1 Linear accelerator with a 160-leaf multi-leaf collimating system allowing for precision shaping of the beam to the target area

Fig. 2 Patient undergoing radiotherapy for head and neck cancer, radiation thermoplastic mask used for immobilisation

Lundahl et al. [4] conducted a retrospective, matched-pair analysis to compare the outcomes of surgery alone versus surgery plus post-operative radiotherapy (PORT). The study found that the PORT group had a significantly higher rate of locoregional control, disease-specific survival and overall survival compared to the surgery alone group.

Although post-operative radiation (PORT) can increase patients' chances of surviving head and neck cancer, it can also negatively impair patients' quality of life (QoL) in both acute and chronic ways. For further information, please visit the website. Due to its decreased toxicity and comparable survival results, intensity-modulated proton therapy (IMRT) has replaced PORT as the gold standard of therapy for oral cavity cancer. Intensity-modulated proton therapy (IMPT) has also been investigated as a treatment for head and neck cancer. Compared to IMRT, IMPT potentially reduces radiation-associated toxicities since it delivers a lower dosage of radiation outside the targeted area. Studies comparing the long-term oncologic, functional and QoL results of IMPT versus IMRT are currently being conducted.

3 Neoadjuvant Chemotherapy

Neoadjuvant chemotherapy (NACT) has been explored as a potential treatment option for oral cavity cancers with the aim of preserving organs. Various studies have shown that NACT can potentially enable the preservation of the mandible during surgery, leading to improved cosmetic and functional outcomes for patients. However, there is a lack of agreement regarding the definition of technically unresectable oral squamous cell carcinoma (OSCC), which can make it challenging to determine whether NACT is appropriate. Despite this, one study indicated that NACT can be effective for patients with technically unresectable OSCC and should be pursued with the goal of achieving resectability.

When it comes to unresectable oral squamous cell carcinoma, there is a debate regarding the usefulness of induction chemotherapy compared to standard treatment with concurrent radiotherapy. While some studies have shown that NACT followed by concurrent radiotherapy (CRT) can improve overall survival and progression-free survival in patients with locally advanced head and neck cancer, others have found no significant

difference in overall survival between NACT and CRT alone. Therefore, the benefit of NACT compared to standard treatment for inoperable/unresectable disease is not entirely clear.

4 Immunotherapy

The update presented at the American Society of Clinical Oncology (ASCO) 2019 revealed that the pembrolizumab group receiving chemotherapy also experienced an improvement in overall survival [5]. The algorithm for immunotherapy in recurrent/metastatic HNSCC is shown in Fig. 3.

The revolutionary effects of immunotherapy have not been spared squamous cell carcinoma of the head and neck. In fact, checkpoint inhibitors are already a crucial component of the therapeutic arsenal, and novel indications for either monotherapy or combination therapy are constantly developing. Additionally, numerous additional kinds of molecules are constantly being developed. An essential difficulty in the application of these novel medicines is still the selection of patients who will benefit from immunotherapy with new biomarkers. It is still unclear how long these immunotherapy treatments should last and what combinations are the most effective.

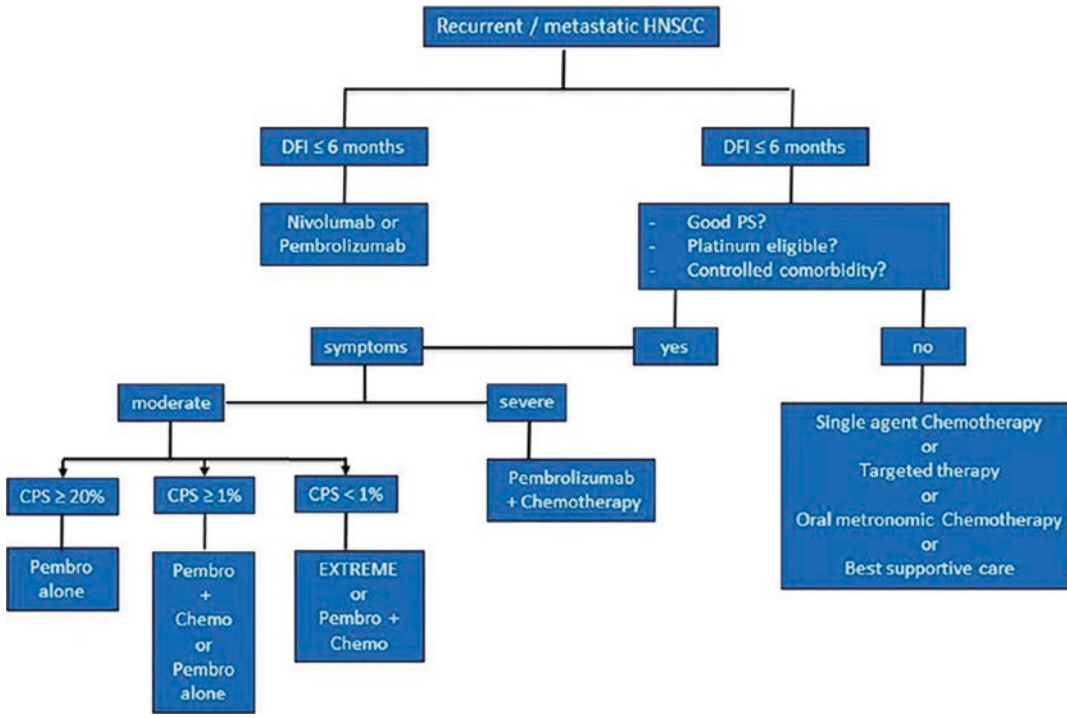

Fig. 3 Role of immunotherapy in recurrent/metastatic HNSCC

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Transoral Robotic Surgery

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Traditional open procedures have been the standard way of resection for oropharyngeal and laryngeal tumours. Due to their location, these tumours cannot be removed via transoral surgery. Open procedures enable a wide surgical field exposure, which facilitates the resection of tumours with adequate oncologic surgical margins. The quality of life for those who use these open procedures is, however, typically lowered as a result of long-term sequelae of poor functional outcomes, such as swallowing and speech issues. For the purpose of creating surgical techniques and treating patients with head and neck problems, transoral surgery is growing in popularity. Traditional open procedures have been the standard way of resection for oropharyngeal and laryngeal tumours. Due to their location, these tumours cannot be removed via transoral surgery. Open procedures enable a wide surgical field exposure, which facilitates the resection of tumours with adequate oncologic surgical margins. The quality of life for those who use these open procedures is, however, typically lowered as a result of long-term sequelae of poor functional outcomes, such as swallowing and speech issues. For the purpose of creating surgical techniques and treating patients with head and neck problems, transoral surgery is growing in popularity. Transoral laser microsurgery (TLM), transoral robotic surgery (TORS) and other cutting-edge techniques in head and neck surgery were developed by Strong and Jako in the late 1970s [1]. Robotic surgery is the practice of performing surgical procedures with greater accuracy and efficiency using an electromechanical instrument.

Neil Hockstein from the University of Pennsylvania first applied the da Vinci technology to the head and neck in 2005 [2]. The most popular robotic system in head and neck surgery, the da Vinci Si HD (Intuitive Surgical, Sunnyvale, CA), has been utilised for transoral resections of head and neck tumours since 2005. The FDA authorised robotic surgery for head and neck operations in 2009. The FLEX Robotic System, the next iteration of the robotic system, was developed and manufactured by Medrobotics Inc. (Raynham, MA). The endoscope on this highly articulated platform can acquire a semi-rigid or flexible condition and rotate freely with the aid of altering the tension on the wires that run through the segments. The gradual adoption of robots in clinical practice has led to further developments by Intuitive Surgical and other

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N. M. Nagarkar et al. (eds.), *Atlas of Head Neck and Skull-base Surgery*,
https://doi.org/10.1007/978-981-99-6132-0_14

businesses to produce revolutionary surgical systems. TORS was able to overcome a number of TLM-related restrictions by allowing surgeons the freedom to perform precise surgical manoeuvres with exceptional (3D) endoscopic visualisation, easier en bloc resection, better optics, more flexible instruments, better haemostasis and hand tremor filtration with increased range of motion.

1 Oropharynx

Oropharyngeal squamous cell carcinoma (OPSCC) has typically been treated with open surgery due to their challenging access and complex anatomical locations. Speech and swallowing were substantially compromised, with a morbidity rate of almost 30%. A paradigm breakthrough in medical treatment, concurrent chemoradiotherapy (CTRT), produced comparable survival rates, acceptable locoregional control and minimal morbidity. Even if the early results of radiation therapy appear to be favourable, the long-term effects were a serious issue since they had a negative impact on the patients' quality of life. Even though CTRT is effective in treating oropharyngeal cancer caused by HPV, the dreadful long-term repercussions have a negative impact on survivorship, especially in young people.

TORS is a fascinating new technology which enables the management of some OPSCC cases. TORS is prepared to take on a larger role in the management of OPSCC as more surgeons get access to the technology and training.

The concept behind TORS is an en bloc resection with a clear margin. What constitutes an inadequate resection margin is not currently agreed upon. A narrow margin is defined by the National Comprehensive Cancer Network (NCCN), European Institute of Oncology (IEO) and American Society of Clinical Oncology (ASCO) as 5 mm or less without distinction for subsite. Alicandri-Ciufelli et al. observed in their detailed analysis of surgical margins in the head and neck that most research use a margin distance of 5 mm or more to determine margin clearance [3]. Weinstein et al. determined that a margin of 2 mm or less was close and a margin of more than 2 mm was regarded as a free margin [4].

Performing a radical tonsillectomy is one of the first procedures doctors do when beginning a TORS practice. In 2007, Weinstein et al. presented the initial study on TORS radical tonsillectomy.

1.1 Oncological Outcomes

In comparison to primary radiation therapy (RT) or chemoradiation series, there is evidence that transoral robotic surgery (TORS) may generate equivalent oncologic outcomes and superior functional outcomes; however the selection of TORS patients may be biased. TORS has been demonstrated to have survival rates that range from 81 to 100% for overall survival and from 85.7 to 96% for disease-free survival, respectively. It also has a lower toxicity profile. TORS has been linked to longer life times in patients with oropharyngeal squamous cell carcinoma (OPSCC) who tested negative for the HPV virus in certain trials, but not in others where there was no discernible difference in survival between TORS and radiation therapy. There was also no significant difference in survival rates between TORS and traditional open surgery; however TORS was related with a higher rate of margin negative in one research. Transoral laser microsurgery (TLM) has been linked to 3-year overall and disease-free survival rates of 86% and 82%, respectively, and has been proven to have improved functional outcomes compared to traditional open surgery in cases of initial or recurring oropharyngeal cancer [5].

1.2 Functional Outcomes

Data on functional outcomes following treatment for oropharyngeal squamous cell carcinoma (OPSCC) are scarce and heterogeneous, and it is challenging to compare outcomes across treatment modalities due to the use of various quality of life (QOL) questionnaires; small, diverse patient populations; short follow-up times; and high dropout rates. However, some research suggests that, when compared to chemotherapy and radiation therapy, transoral robotic surgery

(TORS) may result in improved swallowing outcomes and reduced rates of tracheotomies (CRT). At specific time points, TORS may possibly be linked to superior speech results when compared to adjuvant CRT or radiation (RT).

According to numerous studies, TORS may also be helpful for locating and treating unknown primary tumours in the head and neck, with a primary detection rate of 53–78% and a positive surgical margin rate of 19.4% [6]. When patients have negative results from a physical examination, diagnostic imaging and positron emission tomography (PET), TORS or transoral laser microsurgery (TLM) may be especially helpful for finding occult primary tumours.

TORS has been utilised for supraglottic laryngectomy with satisfactory functional results, but there is limited data on its usage for total laryngectomy or glottic malignancy. With the proper neck dissection, TORS might be an effective primary therapy option for early hypopharyngeal carcinoma. The use of TORS for thyroid surgery is still in its infancy and needs more development, standardisation and oncological result and safety testing. The use of remote access procedures for thyroid surgery has reduced due to concerns about patient candidacy, safety and resource use. In specialised clinics, robotic-assisted thyroid surgery is still in its infancy, but there is a market for it among patients who value its aesthetic advantages.

2 Contraindications for TORS

TORS may not be used if a patient has certain contraindications, such as trismus (restricted jaw movement), specific tumour sites or sizes or other medical disorders that may render a patient unfit for general anaesthesia. Preoperative imaging can help pinpoint the tumour's position, size and extent as well as any anatomical traits that might have an impact on the operation's outcome. The distance from the posterior pharyngeal wall to the hyoid bone, the angle between the epiglottis and the vertical plane of the larynx and the distance from the posterior pharyngeal wall to the soft palate are additional markers that can suggest restricted access to the area being treated. For some types of advanced cancer, neck conditions that cannot be surgically treated and numerous distant metastases, TORS may not be advised. Medical comorbidities that preclude a patient from receiving general anaesthesia, trismus that restricts access to the oral cavity and cervical spine illness that prevents the patient from assuming the proper position are all non-oncological contraindications to TORS.

There are various limitations to adopting robotic surgery, including cost-effectiveness, loss of tactile sense and the necessity for haptic feedback between the operator and the machine. Many towns find it financially unviable due to the expensive cost of the equipment and the uncommon usage of otolaryngologic equipment in the neck. The camera's better visual information can somewhat make up for the loss of touch sensibility, but haptic feedback is still necessary for precise surgery [7].

For the treatment of oropharyngeal and supraglottic laryngeal malignancies, transoral robotic surgery (TORS) has been proven to have high functional results and low tracheotomy rates. Additionally, it might be helpful for the detection and management of unidentified primary tumours as well as thyroid disorders. For the best results, cases should be carefully chosen because TORS may not be appropriate for all patients. The price of TORS and the surgeon's lack of tactile perception place restrictions on its use. TORS has a wide range of potential applications, and as technology advances, so may its pricing.

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