Squamous Cell Carcinoma Extending to the Temporal Bone

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Introduction

Squamous cell carcinoma (SCC) of the temporal bone is an aggressive malignancy. It presents as a primary tumour of the temporal bone arising in the middle ear or external auditory canal (EAC). Secondary invasion of the temporal bone occurs because of direct spread from a primary lesion of the pinna and its surrounds, or from cutaneous SCC (cSCC) metastasis to the first echelon lymph node bed. In Australia, SCC of the temporal bone typically presents from direct invasion from a cutaneous primary, or from metastatic spread from a cutaneous primary to the parotid lymph nodes abutting the temporal bone [1].

The rarity of disease, variety of histological subtypes, location of the primary, lack of a universally accepted staging, and surgical nomenclature make evidence-based management problematic. Most reports include primary and secondary invasive malignancy in the same cohort [1]. Hence, treatment is driven by consensus and observational studies.

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The aggressive nature of this disease mandates aggressive surgery with a focus on achieving clear margins. Management requires a motivated patient who accepts the significant morbidity associated with achieving this result.

This chapter discusses the pathology and epidemiology of temporal bone SCC. An overview is given of the special circumstances and controversies in staging; and strategies to achieve clear surgical margins are also outlined.

Pathology and Epidemiology

Primary SCC of the temporal bone is rare (6:1,000,000) [2]. Most primary tumours occur in the EAC with only 5 % occurring in the middle ear [3]. The majority of SCCs involving the temporal bone is secondary, with direct invasion from primary lesions of adjacent structures (Fig. 10.1) or invasion from cutaneous metastatic deposits of the parotid or occipital lymph node beds. In our institution, 90 % of lateral temporal bone resections (LTBR) performed are for the treatment of metastatic cutaneous malignancy [1].

Whereas the risk factors for primary skin cancers are well documented, metastatic spread is relatively uncommon and occurs more frequently in the immunocompromised patient [4]. Primary carcinoma of the temporal bone arises in the setting of chronic inflammation [5]. Histologically, the majority of neoplasms encountered on the pinna are basal cell carcinomas (BCCs). SCC, on the other hand, is found chiefly in the EAC and middle ear. Carcinoma in the middle ear can also have an origin in the salivary gland, but this is rare [6].

Most cSCCs do not metastasize. Therefore, when they do spread to the adjacent lymph node beds of the temporal bone and become invasive, they are an aggressive phenotype [4]. SCC involving the temporal bone spreads rapidly, as the embryological fusion planes that form the temporal bone facilitate spread by the path of least resistance. Primary cutaneous malignancy of the pinna has easy access to the EAC by direct invasion. Typically, metastatic cutaneous carcinoma to the first echelon lymph nodes of the parotid bed will grow and abut the

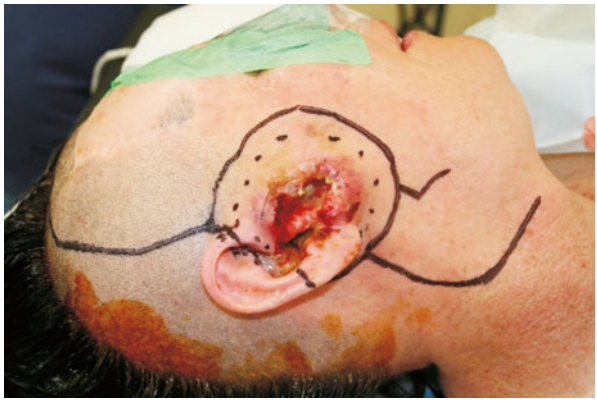

Fig. 10.1 Cutaneous SCC invading the parotid and anterior temporal bone

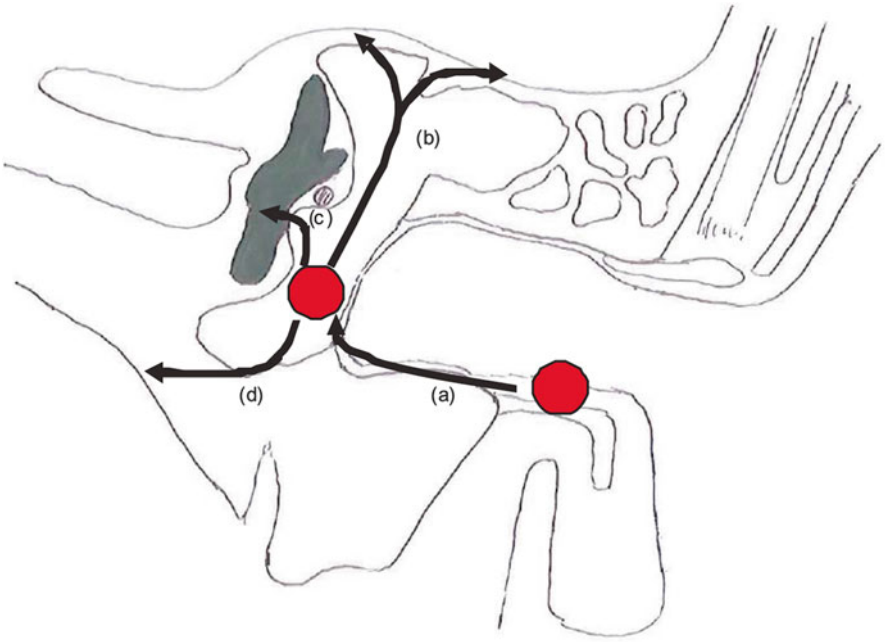

Fig. 10.2 Coronal view of the temporal bone showing tumour spread from (*a*) the cartilaginous EAC to the annulus. Once the annulus is breached, tumour in the middle ear can spread by the path of least resistance, (*b*) superiorly to the tegmen, (*c*) medially through the otic capsule, and (*d*) inferiorly into the hypotympanum

EAC. From here tumour can spread into the ear canal by the fissure of santorini in the tragal cartilage. This same pathway allows anterior spread of primary EAC malignancy to the first echelon lymph node bed in the parotid gland. Within the cartilaginous EAC a tumour can spread posteriorly through the conchae into the post-auricular sulcus. Once the bony EAC is involved, the tumour spreads rapidly under the thin skin of the EAC medially through to the tympanic membrane before entering the middle ear [7]. The skin within the EAC is thin and its proximity to underlying cartilage and bone make complete excision challenging [8]. Regardless of the origin of temporal bone malignancy, once the EAC is involved, the tumours tend to spread in a similar fashion [9].

Within the middle ear, the tumour can spread in multiple directions, depending on aeration of the middle ear and path of least resistance. Anteriorly, the tumour spreads through the mesotympanum into the carotid canal and eustachian tube. Once the carotid canal is involved, spread can proceed superiorly into Meckel's cave and the cavernous sinus. Medially, weak points in the otic capsule at the round and oval windows allow spread into the vestibule, cochlea, internal acoustic meatus and posterior cranial fossa. Inferior spread allows invasion to the jugular foramen and lower cranial nerves. Superior spread can cause tegmen erosion and dural/temporal lobe involvement (Figs. 10.2 and 10.3).

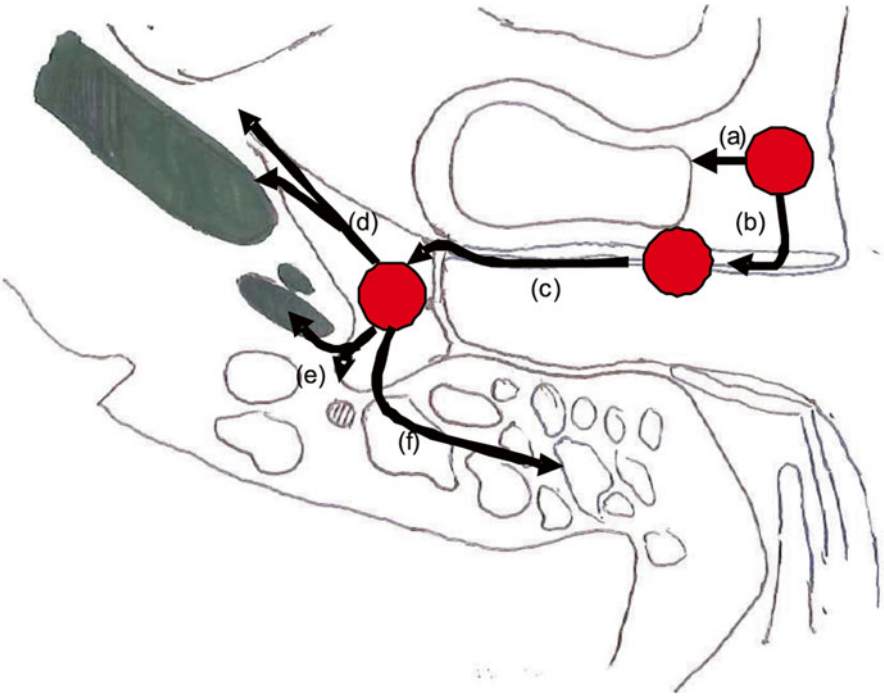

Fig. 10.3 Axial view of the temporal bone showing tumour spread (*a*) directly from the parotid bed into the temporo-mandibular joint, (*b*) from the parotid bed through the fissure of santorini into the external auditory canal (EAC), (*c*) the cartilaginous EAC to the annulus. Once the annulus is breached, tumour in the middle ear can spread by the path of least resistance, (*d*) anteriorly to the eustachian tube and internal carotid artery, (*e*) medially through the otic capsule and facial nerve, and (*f*) posteriorly to the mastoid cavity

The facial nerve can be involved by several mechanisms. Direct invasion in the mesotympanic portion of the facial nerve or, in advanced disease, direct invasion of the mastoid segment can occur. More commonly, the facial nerve is involved by metastatic deposits in the extra-tympanic portion of the facial nerve. This occurs because of large nerve perineural spread or direct invasion at the stylomastoid foramen [7].

Clinical Presentation

Primary pinna lesions often present after attempts at local excision result in a positive margin. Typically, in this scenario the degree of spread is underestimated and extends medially along tissue planes underneath the approximated skin. Metastatic cSCC spread to the temporal bone is usually obvious, with a history of progressive swelling in the adjacent lymph node beds prior to otological symptoms. The rarity of primary temporal bone carcinoma results in complacency; in the early stages it

Table 10.1 Signs and symptoms that often predict the path of invasion in temporal bone malignancy

Path of invasion	Signs and symptoms
Middle ear and eustachian tube	Conductive hearing loss, otalgia, taste disturbance
Otic capsule	Sensorineural hearing loss, facial nerve palsy, vertigo
Carotid canal	Horner syndrome, syncopal episodes
Temporomandibular joint	Trismus, malocclusion
Jugular foramen	Lower cranial nerve palsies
Intracranial	Seizure, cognitive deficits, meningitis/encephalitis
Infratemporal fossa	Mandibular nerve (V3) paresis

tends to be misdiagnosed as symptoms of otalgia and otorrhoea are non-specific [2]. Deep unrelenting pain that does not improve rapidly with standard treatment should prompt a biopsy [7]. As the tumour advances, the presentation becomes more obvious, with a facial nerve palsy or noticeable tumour extension.

A thorough clinical examination is paramount, and includes skin survey, cranial nerve examination and comprehensive head and neck examination of the nodal basins. Often, signs and symptoms can predict the path of invasion (Table 10.1). Facial nerve palsy indicates advanced disease. It is important to differentiate facial nerve weakness as a consequence of previous surgical excisions and progressive weakness indicative of perineural invasion. Often the medial EAC and tympanic membrane are not accessible because of the tumour or pain—hence imaging is vital.

Imaging

Arriaga et al. studied the correlation between computed tomography (CT) and surgical findings and concluded that CT helps to define the pathological extent of tumour and bone erosion but is limited in that mucosal inflammation cannot be distinguished from tumour without bone erosion [10]. MRI (magnetic resonance imaging) can be useful to evaluate soft tissue extension and to differentiate the contents of the middle ear [11]. The 3-Tesla (3 T) platform improves resolution and signal to noise ratio when defining cranial nerves and the skull base [12]. In our institution, we routinely perform a high-resolution CT with contrast using both bone and soft tissue windows in conjunction with a 3 T MRI neurogram. This differentiates pathological soft tissue growth from retained mucosal secretions, temporomandibular joint (TMJ) capsule invasion, and perineural spread (PNS) within the facial nerve and auriculotemporal nerve.

Staging

The complexity of anatomical relations within the temporal bone mandates accurate preoperative staging [13]. This imaging is essential for prognostication and staging when developing a surgical plan with other specialties, such as neurosurgery.

Table 10.2 Comparison of the evolution of staging of external auditory canal (EAC) squamous cell carcinoma from the standard American Joint Committee on Cancer (AJCC) cutaneous malignancy staging system with the Pittsburgh staging system

	AJCC	Arriaga et al. [13]	Moody et al. [11]
T1	Tumour <2 cm with <2 high-risk features	Tumour limited to the EAM without bony erosion or evidence of soft-tissue extension	Tumour limited to the EAM without bony erosion or evidence of soft-tissue involvement
T2	Tumour >2 cm in greatest dimension or tumour of any size, with ≥2 high-risk features	Limited EAM bone erosion (not full thickness) or radiographic finding consistent with limited (<5 mm) soft-tissue involvement	Tumour limited to the EAC with bone erosion (not full thickness) or limited soft-tissue involvement (<5 mm)
T3	Tumour with invasion of maxilla, orbit or temporal bone	Full thickness erosion of the EAM bone, tumour involving the middle ear or mastoid—facial nerve palsy	Tumour eroding the osseous EAC with limited soft-tissue involvement (<5 mm) or tumour in the middle ear or mastoid
T4	Tumour invasion of skeleton or perineural invasion of the skull base	Tumour eroding cochlea, petrous apex, medial wall of middle ear, carotid canal, jugular foramen or dura, with 5 mm soft-tissue involvement	Tumour eroding cochlea, petrous apex, medial wall of middle ear, carotid canal, jugular foramen, dura, with >5 mm soft-tissue involvement, or evidence of facial paresis

EAM external auditory meatus, *EAC* external auditory canal

Staging of invasive cSCC of the temporal bone with the American Joint Committee on Cancer (AJCC) guideline for cutaneous malignancy is problematic, as even early SCC of the EAC which has easy access to bone, is given a T4 status [8]. These tumours are allocated the same status as deep parotid tumours with facial nerve palsy that is clearly not representative of the extent of tumour spread.

Arriaga et al. developed a comprehensive system to stage tumours of the EAC on the basis of CT and pathological findings [13]. Moody et al. argue that by definition, facial nerve paralysis, when the middle ear is involved, reflects extension through the annulus and into bone of the medial wall of the middle ear [11]. If paralysis occurs on account of extra-temporal invasion, >5 mm of soft tissue is likely to be involved; thus they recommended upstaging facial paralysis to T4 (Table 10.2) [11]. The Pittsburgh tumour staging system for EAC SCC is gaining support in the literature and has been validated by other studies [8].

The N and M status of the Pittsburgh staging system is based on the original AJCC classification [14]. The staging system places considerable importance on metastasis as a T2 lesion, with any cervical metastasis considered to be stage IV disease.

Prognosis

Poor prognostic indicators are debated in the literature and reflect the variability in presentations and management. Although still debated, the following features are suggestive of a poor outcome: (i) bone invasion [15, 16], (ii) extension to middle ear [15, 17],

(iii) facial paralysis [9], (iv) dural involvement [5], (v) PNS [8], (vi) early (T1–T2) versus late (T3–T4) presentation [1], and (vii) regional lymphadenopathy [18].

Whereas these features might be debatable, the fact that recurrence after initial management offers a dismal outcome is undisputed [18], as is the fact that achieving clear margins significantly improves survival [1, 10, 19]. Often, recurrence occurs quickly and aggressively, prompting many investigators to believe that a disease-free survival of 2 years can be considered a cure [18]. Arriaga et al. quote a 75 % disease-specific survival (DSS) in patients with negative margins compared with a 25 % DSS in patients with positive margins [13]. This is corroborated by Prasad and Janecka who report that in locations where clear margins cannot be achieved, such as the petrous apex, ICA, dura and brain, survival is poor [5]. In the senior author's experience (BP), DSS was 79 % and 62 % for 2 and 5 years, respectively, with T1–2 tumours having a 100 % 5-year survival [1].

Management (History and Surgical Rationale)

Management requires close collaboration in the setting of a head and neck multidisciplinary team (HNMDT) meeting. Importantly, the meeting must have a skill mix that allows for accurate radiological staging so that an appropriate surgical plan can be established. Resection should aim to achieve clear margins, if possible. The ablative surgeon should plan to achieve this either with primary surgery to the temporal bone or in association with ancillary manoeuvres, such as parotidectomy, TMJ resection, infratemporal fossa resection, access to the middle cranial fossa, and neck dissection to gain clearance. Like others in the field, our team considers involvement of the internal carotid artery, cavernous sinus and jugular bulb incurable, and hence would offer debulking surgery or palliative radiotherapy [19]. Reconstruction is required after ablation. In our institution, this is usually achieved with a vascularized free flap. Finally, detailed specimen labelling and description is required so that a specialized head and neck pathologist can give an accurate report of the relevant margins and be aware of the air–tumour interface in the mastoid cavity for postoperative treatment by radiation and medical oncologists.

Oncologically sound surgery mandates resection with clear surgical margins [20]. Incomplete excision, in an effort to preserve cosmesis and function, places the patient at risk of local recurrence, metastasis [21], and a poor prognosis [18, 22]. The aggressive nature of this disease mandates a surgical plan that can result in morbidity. Before undertaking surgery, patient comorbidities must be taken into account by the HNMDT, who should make a decision with a motivated patient only after a frank discussion on the risks and benefits of surgery. Surgical management to address the temporal bone component of disease does not vary for primary or secondary disease [9]. Our group does not advocate the use of sleeve resection in SCC of the EAC. Less aggressive procedures, such as local canal resection, make clear margins difficult to achieve, with 54 % of tumours treated this way showing positive margins in one cohort [23] In this study, recurrence rates of 46 % for T1 and T2 tumours were reported [23].

Confusion has occurred because descriptions for temporal bone resection vary [11]. Traditionally, these malignancies were addressed with a radical mastoidectomy and ablation of the EAC. Parsons and Lewis proposed an *en-bloc* subtotal temporal bone resection (STBR) as an alternative to a radical mastoidectomy, with resection of the medial surface of the mesotympanum, leaving only the air cells of the petrous apex and internal carotid artery [24]. The total temporal bone resection (TTBR) is an extension of this with incorporation of the petrous apex and sacrifice of the internal carotid artery. To preserve facial nerve function, Conley and Novack described the lateral temporal bone resection (LTBR) [25]. In 1997, Hirsch and Chang rationalized the nomenclature for temporal bone resection with LTBR, STBR, TTBR [7].

Surgical Philosophy

To achieve a reasonable outcome, the intent of surgery must be to resect with clear margins. The temporal bone resection allows for clear medial margins in the properly selected patient. To achieve a clear peripheral margin requires the use of ancillary manoeuvres. At a minimum, Leong et al. advocate a superficial parotidectomy in conjunction with a LTBR to ensure resection of SCCs that involve the EAC [22]. The rationale for this is that often invasion through the fissure of santorini is not detected on radiology. The operation is extended to a radical parotidectomy if evidence exists of facial nerve involvement, or if there is obvious tumour medial to the facial nerve, as in this scenario facial nerve involvement is inevitable. Our group does not advocate a prophylactic comprehensive neck dissection, as only 7.5–15 % of patients will have occult cervical lymphadenopathy at the time of presentation [6, 26, 27]. Often, a limited level 1B-III neck dissection is performed to facilitate vascular access for free-flap reconstruction. Further spread medially is often limited by the tough capsule of the TMJ. This can be resected as a medial margin with a parotidectomy. If involvement of the TMJ or mandible is evident on preoperative staging, then this is sacrificed to gain clear margins (Figs. 10.4 and 10.5). In the experience of the senior author, the incorporation of the TMJ or mandible *en bloc* with the temporal bone resection was required in 30 % of the advanced cases seen in our institution [1].

A significant factor in the evolution of skull base surgery has been the use of radiology to improve surgical planning. Specifically, in lateral skull base surgery a 3 T MRI neurogram allows for proper surgical planning to address PNS and soft-tissue extension. In obvious facial nerve PNS to the stylomastoid foramen, the facial nerve is sacrificed to the second genu and frozen section assessment is carried out to determine clearance. If PNS exists, then further resection with a STBR is required and more frozen sections are taken to achieve at least a 5 mm clearance margin. Surgery for perineural spread beyond the geniculate ganglion is controversial. However, as the tumour is often confined within the perineurium [28] and the nerve is surrounded by the cerebrospinal fluid, the senior author will resect the facial nerve back to the brainstem to achieve a clear margin. Perineural spread along the

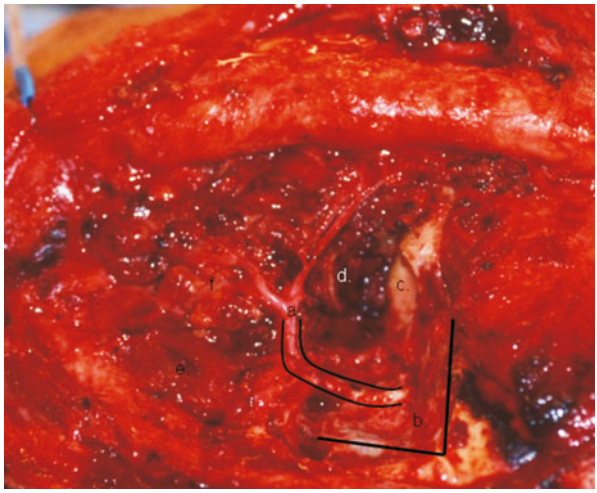

Fig. 10.4 Clinical photo after lateral temporal bone resection with extended resection to include the temporo-mandibular joint demonstrating (*a*) intact facial nerve from second genu to periphery, (*b*) sino-dural angle, (*c*) glenoid fossa, (*d*) cut end of the neck of mandible, (*e*) posterior belly of digastric with removal of mastoid tip, and (*f*) remaining deep lobe of parotid

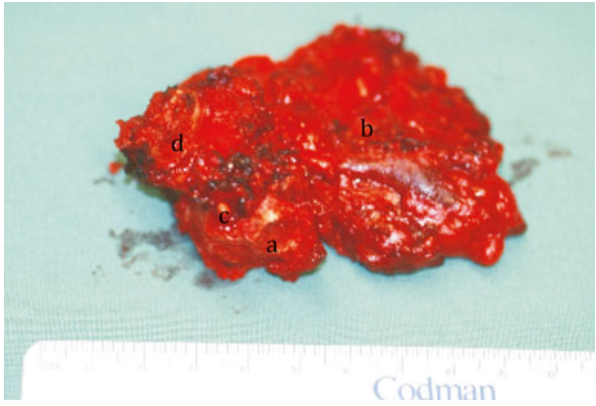

Fig. 10.5 Clinical photo of *en-bloc* specimen demonstrating (*a*) lateral temporal bone, (*b*) conservative parotid, (*c*) head of malleus, and (*d*) neck of mandible

auriculotemporal branch of the mandibular nerve requires clearance of the infratemporal fossa. This is achieved by resecting the TMJ, ascending mandible and pterygoid muscles. Again, frozen section control of the mandibular nerve at the foramen ovale is performed. The philosophy of preserving the facial nerve, if it is fully functioning, is well documented in parotid malignancy [29]. Whereas every effort is made to preserve the facial nerve if it is functioning, this is not done at the expense of involved margins, in temporal bone resection.

Using this philosophy, the senior author has achieved a 2-year overall survival rate of 79 % for all stages of disease. This compares favourably to other cohorts in which large tumours of the parotid (>6 cm or involving skull base) treated with a radical parotidectomy have achieved survival rates for 2 years of only 37 % [30]. Although these cohorts are small it shows the importance of aggressive surgery for this disease.

Lateral Temporal Bone Resection (LTBR)

The surgical goal of a LTBR is to completely excise the EAC with its bony margins. The medial boundary of resection is the tympanic membrane which is excised with the specimen, leaving the intact facial nerve.

Soft-tissue incisions are dictated by the location of the primary lesion but require enough exposure to identify the sigmoid sinus posteriorly and the tegmen superiorly. Once this is achieved a standard mastoidectomy is performed to expose the bony plate over the sigmoid sinus and the middle cranial fossa dura. A posterior tympanotomy is formed and the incudostapedial joint is disarticulated. The posterior tympanotomy is extended inferiorly into the hypotympanum, sacrificing the chorda tympani with sharp dissection; this is extended into the hypotympanum and extended anteriorly into the TMJ. The mastoidectomy is extended into the epitympanum to the root of the zygoma to reach the superior TMJ. The specimen can be rolled forward with digital pressure on the remaining EAC bone. Sometimes an osteotome passed though the posterior tympanotomy is required to liberate the tympanic plate [26]. This manoeuvre can also be used to gain access if the posterior tympanotomy is narrow (for a detailed description of this procedure, *see* Ref. [26]).

Subtotal Temporal Bone Resection (STBR) and Total Temporal Bone Resection (TTBR)

The STBR describes resection of the medial surface of the mesotympanum, leaving only the air cells of the petrous apex and internal carotid artery. Before proceeding, it is important to inspect both the middle and posterior cranial fossa dura for invasion before committing to a large resection. Invasion through the dura results in meningeal carcinomatosis and is irresectable.

Again, soft-tissue exposure is dictated by the location of the primary lesion but needs to be sufficient for access to the middle and posterior cranial fossa. A middle and posterior temporal craniotomy is performed, extending to the root of the zygoma. The middle cranial fossa is dissected from the petrous bone to ensure that the dura is not involved. Posteriorly the dissection is extended to identify the sigmoid sinus and jugular bulb before exposing the pre- and post-sigmoid dura. It is at this point a decision is made to proceed, if the dura is not breached. Once this decision is made, venous control is gained by incising the sigmoid sinus with packing proximally and opening the jugular bulb and packing the inferior petrosal sinus to gain inflow control. A preoperative MRI venogram is performed to assess the torcula for patency. If the contralateral flow is inadequate, sacrificing the jugular bulb may lead to venous infarction. To free the temporal bone, a diamond drill is used to make a cut along the superomedial aspect of the jugular bulb into the hypotympanum and up to the posterior wall of the carotid canal. Next, the middle cranial fossa is dissected free to the foramen ovale. The middle meningeal artery is coagulated at the foramen spinosum and the dissection is continued to the connective tissue of the

posterolateral margin of the foramen lacerum. The roof and lateral wall of the internal carotid canal are removed using a diamond drill all the way to the cochlea. A diamond drill is then used to create an osteotomy via the middle cranial fossa from the petrous bone just lateral to the porus acousticus anteriorly to the carotid canal and inferiorly to the jugular foramen. Finally, a cut is made from the root of the zygoma, across the floor of the middle cranial fossa immediately behind the foramen ovale to the carotid canal. The specimen is then freed by pushing anteroinferiorly and using sharp dissection to free any soft-tissue attachments, including the nerves of the internal acoustic meatus. Haemostasis is achieved and the dural defect is repaired. This can be completed with primary closure or the use of a fascial graft, such as tensor fascia lata followed by free-flap reconstruction (for a detailed description, *see* Ref. [26]).

The morbidity of a STBR is limited to facial nerve palsy, loss of hearing and balance, which most patients can compensate. However, a TTBR involves extending the dissection to include the petrous apex with resection of the internal carotid artery. The potential damage to the cavernous sinus, internal carotid artery and postoperative cranial nerve 3–6 palsy makes this procedure extremely morbid. The consensus is that TTBR is unjustified because of the increased morbidity with no proven survival benefit [20].

Postoperative Radiotherapy

In the experience of our group and that of others, the greatest chance of survival occurs with clear margins and postoperative radiotherapy [1, 8, 20, 29]. In the setting of positive margins, postoperative radiotherapy improves survival [20]. Several groups advocate surgery alone for limited T1 surgical disease [2, 20] and radiation in the event of adverse histological markers only [22].

We advocate adjuvant radiation for stage T2 – 3 cancers. In the setting of T4 temporal bone cancer, the outcomes are dismal, with 5-year DSS varying between 10 and 50 % [11, 20]. In the setting of T4 disease, our group advocates surgery with radiotherapy only if clear margins can be achieved or if debulking will aid in palliation. If clear margins cannot be achieved, several workers advocate radiotherapy alone, giving comparable results to palliative surgery [20, 31].

Conclusion

Typically in Australia, temporal bone carcinoma occurs in the setting of metastatic cSCC to the parotid bed with tumour abutting the EAC. In order to achieve satisfactory cure rates this requires an aggressive surgical resection with a focus on clear margins. The temporal bone resection allows for posterior and medial clearance of the temporal bone. In association with ancillary manoeuvres, such as parotidectomy, TMJ resection, infratemporal fossa resection and neck dissection, temporal bone resection also allows for anterior and inferior control. Our group advocates the use of postoperative radiotherapy to improve survival.

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