Lateral Skull Base Pathologies

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17.1 Introduction

The temporal bone, petrous apex, and jugular foramen are complex anatomic regions of the lateral skull base with distinct pathologies. The diversity and complexity of pathologies arising in the skull base need a multidisciplinary skull base team approach to facilitate management and to avoid significant morbidity and mortality. In this chapter, we discuss the anatomy, common pathologies, diagnostics, and management of jugular foramen, petrous apex, and other temporal bone lesions. The most common lesion seen involving the jugular fossa is a glomus jugulare paraganglioma followed by schwannomas and meningiomas [1]. Cholesterol granuloma is the most common pathologic lesion found in the petrous apex.

Thorough knowledge of the jugular foramen, petrous apex, and temporal bone anatomy is essential for the diagnosis and proper management of their pathologies.

Jugular foramen is one of the most important foramina of the lateral skull base. It contains the lower cranial nerves and major vascular structures. Tumors that develop within it, or extend into it, provide significant diagnostic and surgical challenges. The jugular foramen is separated by the jugular spine and a fibrous band into two fibro-osseous compartments (Fig. 17.1).

1. *Pars vascularis*: posterolateral vascular compartment, which is larger and receives the internal jugular vein, vagus nerve (CN X) with Arnold's branch, the spinal nerve (CN XI), and posterior meningeal artery. The cranial nerves are medial to the internal jugular.
2. *Pars nervosa*: anteromedial nervous compartment, which is smaller and receives the glossopharyngeal nerve (CN IX), the Jacobson's branch, and the inferior petrosal sinus.
   (a) Posterior to the jugular fossa lies the small canal of Arnold's nerve.
   (b) Medial to the jugular fossa, there is the groove of the inferior petrosal sinus and the opening of the cochlear aqueduct.

The petrous apex is a pyramid-shaped structure formed by the medial portion on the temporal bone. It is obliquely positioned within the skull base, with its apex pointing anteromedially and its base located posterolaterally. It is bounded by inner ear structures laterally, the petro-occipital fissure medially, the petrosphenoidal fissure and the internal carotid artery (ICA) anteriorly, and the posterior cranial fossa posteriorly. The middle cranial

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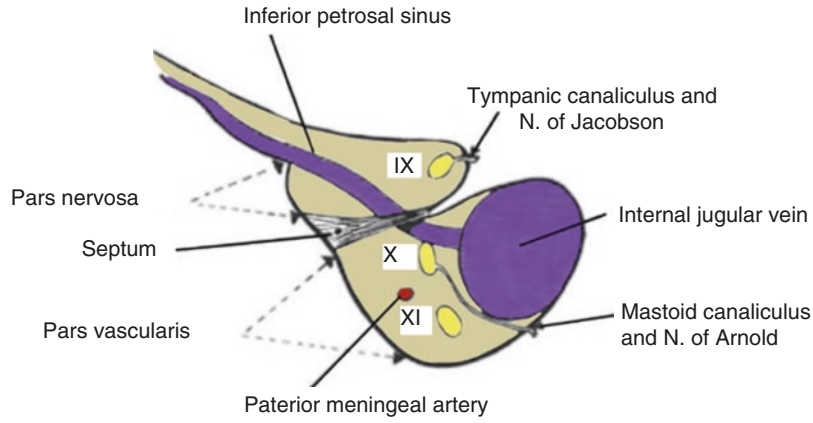

Fig. 17.1 Jugular foramen compartments anatomy and its contents

fossa, Meckel's cave, and ICA form the superior surface. The inferior surface is formed by the jugular bulb and the inferior petrosal sinus. The anterior portion of the petrous apex is filled with marrow in approx. 60% of temporal bones, pneumatized in 33% and sclerotic in 7%.

17.2 Jugular Foramen Tumors

The most common lesion seen involving the jugular fossa is a glomus paragangliomas, other lesions include meningiomas, schwannoma of lower cranial nerves, metastatic tumors, chordoma, and chondrosarcomas. Most of the jugular fossa tumors are slow growing. The clinical manifestations are arising by the compressing effect on the nerves that lie within the fossa and the tumor extension. The signs and symptoms are usually unilateral and can be related to the affected cranial nerves.

17.2.1 Paraganglioma

Paragangliomas, also called glomus tumors, are rare benign, slow-growing tumors, although they are locally destructive, that arise from parasympathetic ganglia chief cells which are arranged in well-defined nests "zellballen." Embryologically it is derived from the neural crest.

They occur more frequently on the left side, females are more frequently affected than males (3:1), and two-third of the patients are in their fourth to fifth decade at the time of presentation. Bilateral in 10% of patients, and multicentric in 5–15%. It has the ability to secrete catecholamines in 1–3%. Malignancy defined by the presence of metastasis occurs in 3–5% of the cases [2]. In 10% it is a familial form of an autosomal dominant disorder, multicentricity may occur in as many as 78% [3]. The hereditary susceptibility is carried on chromosomes 11 and 1 by the PGL loci that are responsible for the SDH gene (succinate dehydrogenase), where the SDH subunit D gene (SDHD) is a critical component of a cellular oxygen-sensing system in the mitochondrial matrix, and its mutation leads to chronic hypoxia stimulation cell proliferation [4]. Glomus tumors considered the most common benign tumor of the temporal bone and middle ear of adults.

They occur in a variety of places in the head and neck. Most common types include:

1. *Carotid body*: the most common type of paragangliomas (65%).
2. *Glomus tympanicum*: usually originate on the promontory of the cochlea. It arises from the tympanic branch of CN IX (Jacobson nerve).
3. *Glomus jugulare*: the most common type of jugular foramen paragangliomas. The incidence, in general, is 0.1 per 100,000. It arises from the adventitia of the jugular bulb, and glomus jugulare tumors are frequently supplied by the ascending pharyngeal artery.
4. *Glomus vagale*: it arises from paraganglia around the vagus nerve at the base of skull.

17.2.1.1 Clinical Presentation

They are usually large at the time of presentation. The symptoms vary and depend on the involvement and extent of the tumor which can affect the lower cranial nerves including VII through XII. Pulsatile tinnitus is the most common presenting symptom [5]. Other symptoms are summarized in Fig. 17.2. Secretory glomus tumors cause flushing, diarrhea, palpitations, hypertension, and headache. If suspect secreting tumor, do 24-h urine catecholamines (vanillylmandelic acid, metanephrine) or plasma-free metanephrines (most sensitive, but high false positives); and order abdominal CT for positive lab tests to rule out pheochromocytoma.

In at least 10% of those with jugular paragangliomas can have multiple ipsilateral cranial nerves involvement. Table 17.1 shows different types of the associated cranial nerve syndromes.

The special signs which might be found with glomus tumors include:

- **Brown's sign**: reddish-blue hue of glomus tympanicum behind intact eardrum, blanches on pneumatic otoscopy. Presents in 10–30%.
- **Aquino sign**: the pulsatile nature of the tumor can be diminished with ipsilateral carotid artery compression.
- **Objective tinnitus** may be apparent if auscultation over the mastoid or infra-auricular area reveals an audible bruit.
- **Rising sun sign**: reddish middle ear mass.

17.2.1.2 Diagnostic Tests

Imaging scans provide essential information to diagnose paragangliomas of all types. Magnetic resonance imaging (MRI) is the best diagnostic test for paragangliomas with the characteristic appearance of "*salt and pepper*"; pepper refers to the signal voids of huge feeding arteries, and salt represents subacute hemorrhage within the tumor [6]. The MRI provides information about tumor size, local extent, regional anatomy, and intracranial extension that are essential in planning surgery.

High-resolution computed tomography (HRCT) of the temporal bone can be used as an initial image. It appears as follows:

- **Glomus tympanicum**: temporal bone CT shows an enhancing mass in middle ear/mastoid/hypotympanum (see Fig. 17.3).
- **Glomus jugulare**: temporal bone CT shows "*Phelp sign*" as a destruction of the carotico-jugular spine that separates the jugular bulb from the petrous carotid artery. The tumor can have irregularly eroded margins with a "*moth-eaten*" appearance. Figure 17.4 shows the MRI and CT appearance of glomus jugulare.

Angiography is not routinely ordered for diagnosis, but it demonstrates the vascularity of paragangliomas and may be used for preoperative embolization. These tumors are highly vascular for which biopsy is contraindicated in most circumstances.

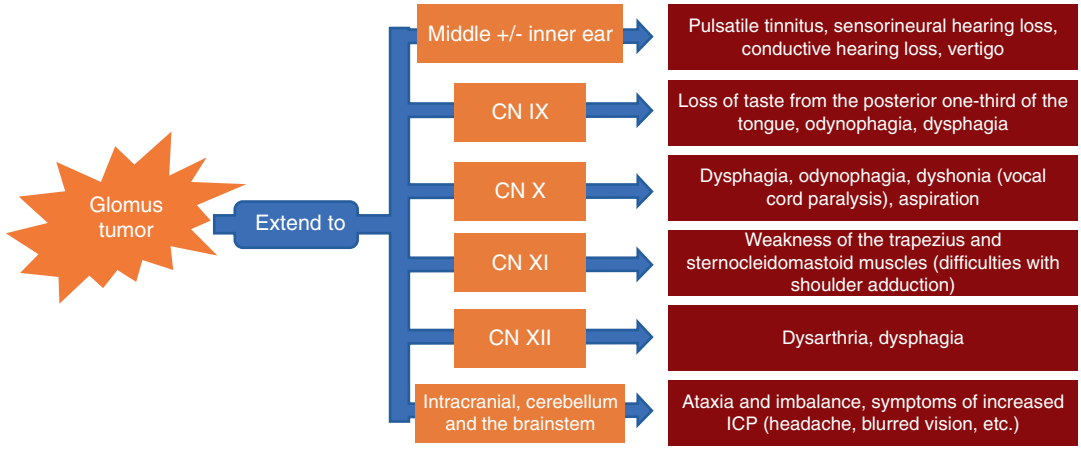

Fig. 17.2 Symptoms of glomus tumor depending on its extension. *CN* cranial nerve, *ICP* intracranial nerve

Table 17.1 Cranial nerves syndromes associated with glomus tumors

Syndrome	Involved cranial nerves (CN)	Clinical manifestations
Vernet syndrome	CN IX–XI	Ipsilateral palsy lead to: dysphonia/hoarseness; soft palate dropping, deviation of the uvula towards the normal side; dysphagia; loss of sensory function from the posterior 1/3 of the tongue; decrease in the parotid gland secretion; loss of gag reflex; sternocleidomastoid and trapezius muscles paresis
Collet–Sicard syndrome	CN IX–XII	Same as Vernet syndrome with addition of CN XII palsy which lead to: dysarthria; unilateral tongue weakness, atrophy and fasciculations
Villaret syndrome	CN IX–XII and sympathetic chain	Ipsilateral paralysis of the last four cranial nerves (IX, X, XI, XII) and Horner syndrome (enophthalmos, ptosis, and miosis)

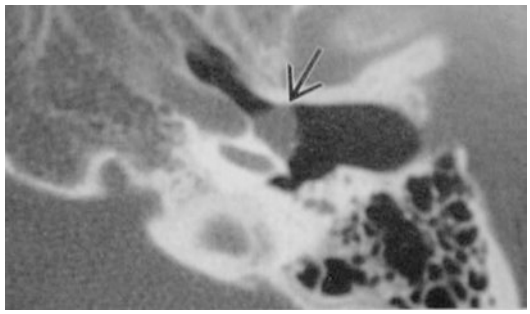

Fig. 17.3 Axial CT section of the left temporal bone showing small glomus tympanicum

17.2.1.3 Staging

Glomus tumors are classified by Fisch classifications, which is described in Fig. 17.5.

17.2.1.4 Treatment

The treatment modalities of glomus tumor include:

- **Surgery**: The principal treatment of glomus tumors is typically surgical excision. Favored for younger patients with cranial nerve deficits. It can be achieved by different approaches, as discussed below.
- **Radiotherapy** (stereotactic or external beam radiotherapy): as a primary or adjuvant therapy with subtotal resection. It can arrest tumor growth rate by causing obliterative endarteritis in the vessels [7].
- **Observation**: may observe if not growing, older patients, and poor surgical candidates. Alpha- and beta-blockers drugs used for secreting tumors as an adjunctive treatment.

Surgery

Complete excision is the favorable outcome after the surgical intervention, but a trend to do a subtotal resection in extensive tumors to spare functioning cranial nerves with possible adjuvant radiation therapy if subsequent tumor progression.

A variety of surgical approaches have been described for the management of glomus tumor that depends mainly on the localization, size, and extension of the tumor. Preoperative preparation and different approaches include the following [1]:

- All approaches need neck dissection for proximal carotid artery and internal jugular vein control.
- Preoperative embolization 1 or 2 days before surgery to decrease intraoperative blood loss, shorten the duration of the procedure, and reduce postoperative morbidity; embolize external carotid artery branches commonly including ascending pharyngeal artery.
- Glomus tympanicum approaches → Transcanal or hypotympanotomy approach via an extended facial recess approach.
  - *Transcanal*: mainly for small localized glomus tympanicum tumors, where the entire edges of the tumor can be identified.
  - *Extended facial recess (hypotympanotomy) approach*: For larger glomus tumors with hypotympanum and mastoid extension. It includes a complete mastoidectomy via a postauricular incision, then facial recess (posterior tympanotomy) opened with a sacrifice of the chorda tympani nerve to

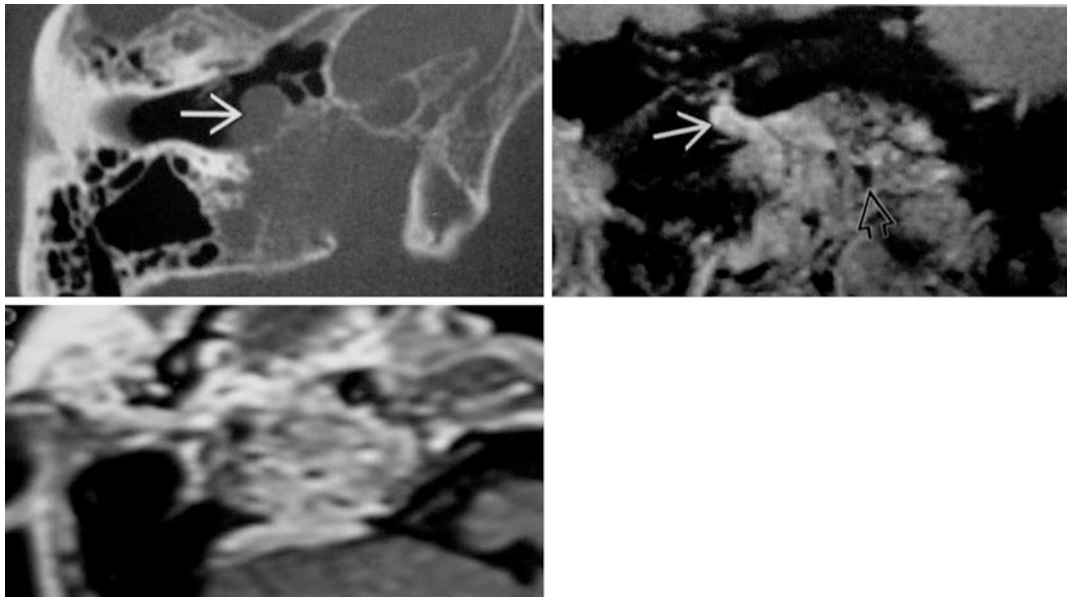

Fig. 17.4 Axial CT, coronal T2WI, and contrast-enhanced axial T1 MRI showing permeative bony destructing lesion centered into the right jugular fossa with erosion of the jugular spine and wall of the petrous carotid canal and extension into the hypotympanic space while showing characteristic salt and pepper pattern in MR images

Fig. 17.5 Fisch staging system (includes both glomus tympanicum and jugulare)

Fisch staging system (Includes both glomus tympanicum and jugulare)

- **Type A involves promontory only.**
- **Type B involves hypotympanum but no erosion over jugular bulb.**
- **Type C erodes bone over jugular bulb:**
  - **C1 erodes carotid foramen.**
  - **C2 involves vertical carotid canal.**
  - **C3 extension to horizontal carotid.**
  - **C4 extension to cavernous sinus.**
- **Type D intracranial extension:**
  - **De extradural, Di intradural**
  - **D1 <2 cm intracranial.**
  - **D2 >2 cm intracranial.**
  - **D3 unresectable.**

allow full exposure to the posterior mesotympanum and hypotympanum with skeletonization of the sigmoid sinus and exposure of the jugular bulb through the retrofacial air cells.

- Glomus jugulare approaches → transmastoid-transcervical exposure ± Fisch type A infratemporal fossa approach with anterior rerouting of the facial nerve to further increase exposure (can cause some degree of permanent facial weakness).
  – *Infratemporal fossa Type (ITF) A Approach*: The development of this approach, as pioneered by Fisch, has

allowed the excision of lateral skull base lesions which were previously deemed unresectable. This approach is used for the removal of tumors where the jugular foramen involves the vertical segment of the petrous internal carotid artery, primarily class C and D glomus temporal tumors. The key point of this approach is the anterior transposition of the facial nerve, which provides optimal control of the infralabyrinthine and jugular foramen regions, as well as the vertical portion of the internal carotid artery (see Fig. 17.6).

- For intracranial extension, a combined otologic and neurosurgical approach is recommended.

17.2.2 Other Jugular Foramen Tumors

- Schwannoma of lower cranial nerves.
- Meningiomas (see Fig. 17.7).
- Metastatic tumors.
- Chordoma.
- Chondrosarcomas.

17.3 Petrous Apex Lesions

The most common lesion seen involving the petrous apex is a cholesterol granuloma [8], and other findings and lesions include asymmetric marrow, effusion, epidermoid cyst, petrous apicitis, metastatic tumors, chordoma, and chondrosarcomas. Petrous apex presents symptoms including hearing loss, facial paresis and paralysis, dizziness, otorrhea, trigeminal paresthesia, and diplopia.

17.3.1 Cholesterol Granuloma

The most common cystic lesion of the petrous apex. Defined as a slowly expansive pathogenesis from obstruction of air cell drainage →

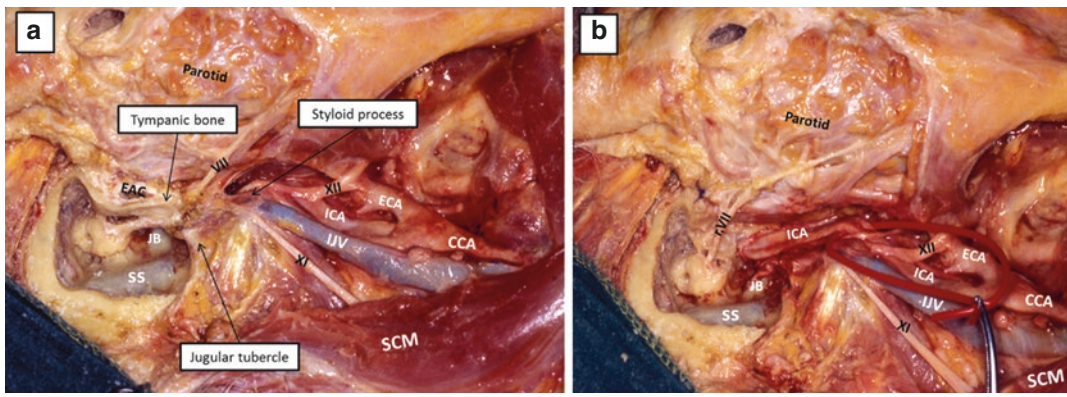

Fig. 17.6 Left side ITF type A. (**a**) A standard, curvilinear postauricular incision extended into the upper neck. The anterior flap is elevated superficial to periosteum over the mastoid and deep to platysma in the neck. The external auditory canal (EAC) is transected at the bony cartilaginous junction, and the flap continued forward over the parotid for 2–3 cm. The facial nerve (VII) is dissected from the parotid. The upper neck is next dissected and great vessels (*IJV* internal jugular vein, *CCA* common carotid artery, *ECA* external carotid artery, *ICA* internal carotid artery) and cranial nerves exposed in the neck. The vagus and accessory nerves (XI) are identified as they exit the jugular foramen and the hypoglossal (XII) is noted as it crosses the carotid bifurcation. The sternocleidomastoid muscle (SCM) is dissected from the lateral and medial mastoid tip and mobilized with the postauricular flap. A well-beveled canal wall down mastoidectomy is next performed and the sigmoid sinus (SS) and jugular bulb (JB) are completely skeletonized. The entire tympanic and mastoid course of the facial nerve is identified and decompressed to 270° of its circumference, from the geniculate ganglion to the stylomastoid foramen. (**b**) The bony EAC and the tympanic bone are removed, the facial nerve is anteriorly transposed, and the styloid process removed to allow complete exposure of internal carotid artery (ICA)

hemorrhage → RBC breakdown → foreign body reaction to cholesterol crystals [9]. Other features include the following:

- Clinical findings: Usually asymptomatic, it may expand to compress CNs and brainstem.
- MRI findings show nonenhancing lesion with T1 and T2 hyperintensity from both high fluid and fat content (see Fig. 17.8).

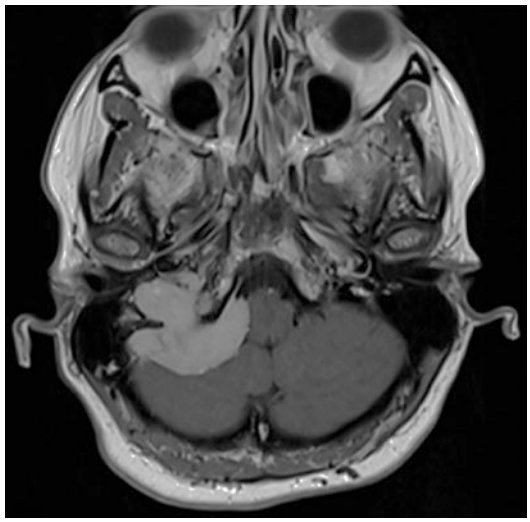

Fig. 17.7 MRI T1 with contrast shows dumbbell shape meningioma of the right jugular foramen

- Management:
  - Observe if not causing symptoms.
  - Surgical decompression for cranial neuropathies, brainstem compression
    - Transnasal approaches provide the widest access for lesions with extension medial to the carotid.
    - Lateral approaches: Infracochlear and infralabyrinthine afford only narrow access but provides a route for aeration and drainage through connection with middle ear/mastoid.

17.3.2 Asymmetric Marrow

It is a variant of normal anatomy; it is found in around 5–10% of individuals as an asymptomatic imaging finding. It appears on MRI as nonenhancing T1 hyperintense asymmetry. CT scan demonstrates normal, noneroded bone. No follow-up or treatment is required.

17.3.3 Effusion/Trapped Fluid

It occurs in a pneumatized petrous apex that trapped effusion. It is seen on imaging studies and in most cases is asymptomatic.

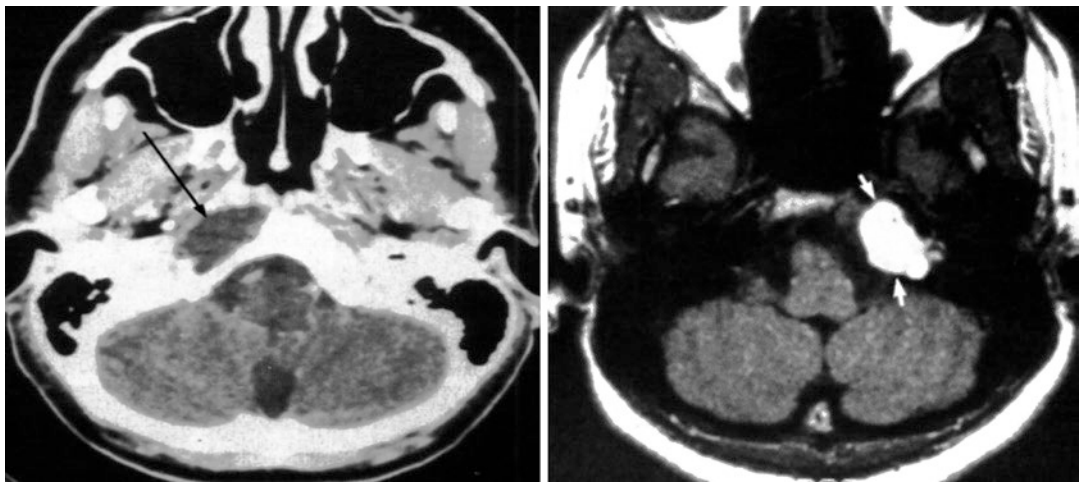

Fig. 17.8 Axial CT and T1 MRI in two different patients showing expansile lesion in the right and left petrous apices respectively displaying CT hypodensity and high T1-WI signal intensity due to fatty contents representing cholesterol granuloma

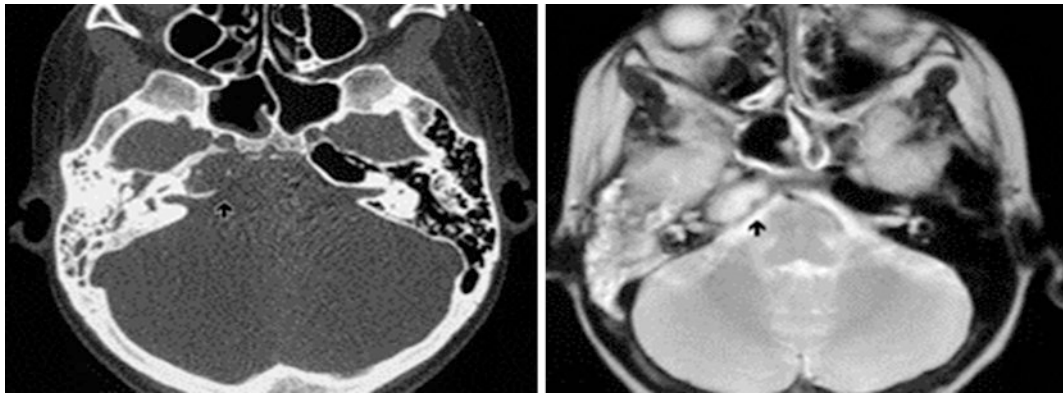

Fig. 17.9 Axial HRCT and T2WI MR showing evidence of right-sided otomastoiditis with opacification of pneumatized right petrous apex and erosion of its posteromedial wall denoting petrous apicitis

CT shows petrous apex air cell opacification with present septations; MRI demonstrates a T1 hypointense, T2 hyperintense area with no enhancement.

Most cases are asymptomatic with no required intervention. For symptomatic patients, prolonged antibiotics with a corticosteroid can be used, and rarely surgical drainage is required.

17.3.4 Cholesteatoma/Epidermoid Cyst

Epithelial cyst within the petrous apex. Imaging studies demonstrate bone destruction on CT and restricted diffusion on DWI MRI.

Surgical excision is quite challenging in this area, if patient has a dead ear. A translabyrinthine or transcochlear approach will give optimal access for the eradication of the cholesteatoma.

17.3.5 Petrous Apicitis

Petrous apicitis is a rare complication of otomastoiditis especially in individuals with a pneumatized petrous apex. There is a classic triad of sixth nerve palsy, deep facial pain, and ipsilateral otorrhea, which is referred to as Gradenigo syndrome, although it may not be present in every patient [8].

CT scan demonstrates air cell coalescence; T1 intermediate signal may have an enhancing ring with abscess; otherwise does not enhance; T2 high signal (see Fig. 17.9).

Treatment is achieved by antibiotics if no improvement surgical drainage should occur by mastoidectomy and drainage.

17.3.6 Chordoma

It is a malignancy of the primitive notochord that usually arises from the midline with extension from clivus to petrous apex. It has a locally aggressive behavior. Skull base chordomas account for only 0.1–0.2% of all intracranial tumors [10]. CT shows a destructive lesion with calcification foci and enhances on T1 with contrast, which may be difficult to differentiate from chondrosarcoma.

The optimal treatment involves radical surgery plus adjuvant radiotherapy. Chordoma carries a poor prognosis with a high rate of recurrence, even after extensive surgery and adjuvant radiotherapy [10].

17.3.7 Chondrosarcoma

It is a rare cartilaginous tumor. It arises from skull base synchondroses, most commonly from the petroclival region [10]. It presents as a destructive lesion that invades bone and extends into soft tissues and can manifest with headache

and diplopia. The lesion is located in the central or paramedian skull base. The diagnosis of chondrosarcoma is radiological; CT shows irregular bone destruction, may have "popcorn" calcifications, and enhances on T1 with contrast.

Treatment is surgical resection; both radiation therapy and chemotherapy are of no benefit as primary therapy, but radiotherapy (proton beam) may be of benefit in cases of subtotal resection and recurrent tumor [11].

17.3.8 Metastasis

The petrous apex is the most common site of metastatic spread within the temporal bone [8]. Breast cancer is the most common primary metastatic cancer, followed by lung, prostate, kidney, and melanoma primary cancers.

17.4 Diffuse Temporal Bone/ Other Skull Base Lesions

17.4.1 Fibrous Dysplasia

It is a focal condition where medullary bone is replaced by fibro-osseous tissue. Usually monostotic (localized to one site), progressive external auditory canal occlusion with conductive hearing loss rarely causes sensorineural hearing loss. Diagnosis is usually made by CT scan, which shows "ground glass" expansive mass; treatment is mainly observation, may consider canaloplasty for conductive hearing loss or cholesteatoma formation behind canal stenosis, and radiotherapy should be avoided.

17.4.2 Eosinophilic Granuloma

It is the most common form of Langerhans cell histiocytosis, known as the localized form, it carries the best prognosis [12]. It typically affects mastoid, the external auditory canal (EAC), and petrous apex, and may involve entire temporal bone; it affects older children and young adults. It presents as painful postauricular swelling, or with granulation and otorrhea of EAC. CT scan shows areas of bony destruction, and MRI T1 with contrast shows enhancement. Treatment is conservative, surgical excision, or low-dose radiotherapy for inaccessible or recurrent lesions.

17.4.3 Rhabdomyosarcoma

It is the most common temporal bone malignancy of children. It affects the middle ear and mastoid. It is divided into four histologic types: embryonal, botryoid, alveolar, and pleomorphic. The embryonal type is the most common. It presents with chronic otalgia and otorrhea that fails to respond to appropriate medical therapy. Treatment includes limited surgical intervention, external beam radiotherapy, and chemotherapy.

17.4.4 Endolymphatic Sac Tumor

It is a rare malignancy of the inner ear, originating from the endolymphatic sac on the posteromedial wall of the temporal bone. It is slow growing, but locally destructive tumor with a low risk of distant metastases.

It is histologically shown as papillary adenomas. It has an association with Von-Hippel–Lindau (VHL) syndrome (seen in 10–30% of VHL patients); therefore, these patients require screening [10].

The diagnosis is mainly by imaging; CT scan shows a soft tissue mass on the posterior petrous face with a destruction of adjacent regions of the temporal bone with a characterizing expansile appearance. MRI shows retrolabyrinthine lesion with hyperintense T1, heterogeneous T2 signal, and T1 with contrast reveals heterogeneous enhancement (see Fig. 17.10).

The treatment is best achieved with early and complete tumor resection. Different lateral skull base approaches can be used according to tumor localization, tumor size, and invasion. It is a highly vascularized tumor with a higher risk of bleeding. Radiotherapy can be utilized depending on the disease stage [10].

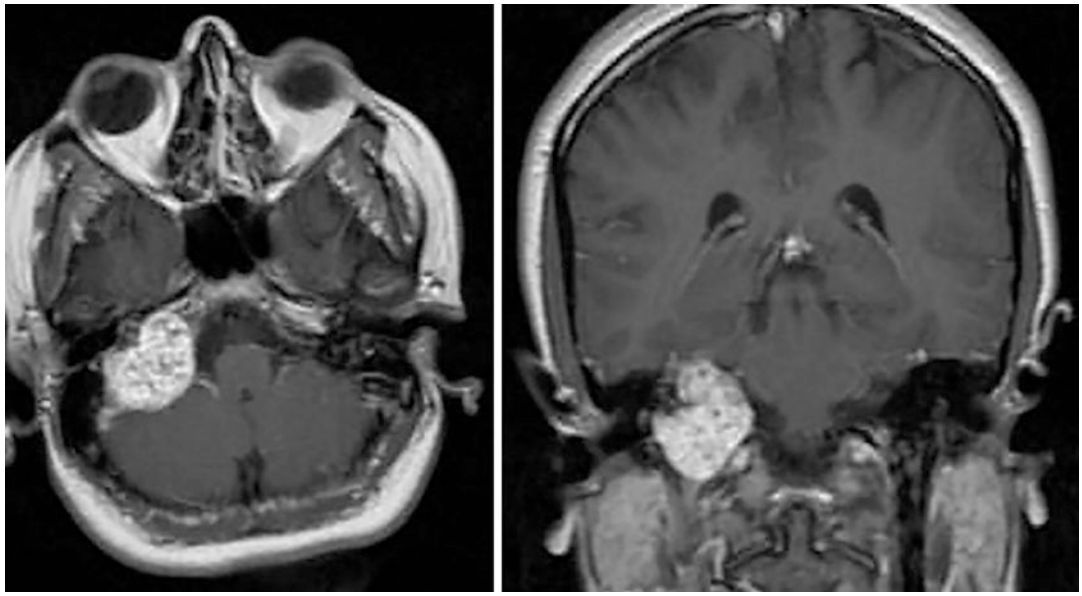

Fig. 17.10 Axial and coronal MRI T1 with contrast reveals heterogeneous enhancement of right ear endolymphatic sac tumor

17.4.5 Osteopetrosis

It is a rare disorder of bone remodeling with a symmetrical increase in bone density because of defective function of the osteoclasts. It presents in two types, an autosomal dominant (AD) type and an autosomal recessive (AR) type. The AD, also known as Albers-Schonberg disease, is the more common type, affecting the head and mandible including the temporal bone and ossicles but it spars the otic capsule. It presents with cranial neuropathies due to compression at neural foramina. The AR, also known as malignant infantile osteopetrosis, is more severe with higher morbidity and mortality.

The treatment focuses on symptomatic management, and decompression interventions of the facial nerve and the cochlear nerve may be of benefit. Bone marrow transplantation also has been described for malignant osteopetrosis.

Take-Home Messages

- Glomus tumors considered the most common benign tumor of the temporal bone and middle ear of adults. Glomus jugulare is the most common type involving the jugular fossa.
- Glomus tumors can present with different types of associated cranial nerve syndromes, including Vernet syndrome, Collet–Sicard syndrome, and Villaret syndrome.
- Jugular foramen lesions surgical excision is quite challenging and might require advance approaches such as transmastoid-transcervical, and infratemporal fossa approach.
- Cholesterol granuloma is the most common pathologic lesion found in the petrous apex.

- Petrous apicitis can present with classic triad of sixth nerve palsy, deep facial pain, and ipsilateral otorrhea, which is referred to as Gradenigo syndrome.
- The petrous apex is the most common site of metastatic spread within the temporal bone, and most commonly metastasize from breast cancer.
- Fibrous dysplasia is characterized by "ground glass" on CT scan. Radiotherapy should be avoided as it carries a risk of malignant transformation.

References

1. Ramina R, Maniglia JJ, Fernandes YB, Paschoal JR, Pfeilsticker LN, Neto MC, Borges G. Jugular foramen tumors: diagnosis and treatment. Neurosurg Focus. 2004;17(2):E5.
2. Borba LA, Araújo JC, de Oliveira JG, Filho MG, Moro MS, Tirapelli LF, Colli BO. Surgical management of glomus jugulare tumors: a proposal for approach selection based on tumor relationships with the facial nerve. J Neurosurg. 2010;112(1):88–98.
3. Fish JH, Klein-Weigel P, Biebl M, et al. Systematic screening and treatment evaluation of hereditary neck paragangliomas. Head Neck. 2007;29(9):864–73.
4. Drovdlic CM, Myers EN, Peters JA, et al. Proportion of heritable paraganglioma cases and assodated clinical characteristics. Laryngoscope. 2001;111:1822–7.
5. Fayad JN, Keles B, Brackmann DE. Jugular foramen tumors: clinical characteristics and treatment outcomes. Otol Neurotol. 2010;31:299–305.
6. Weissman JL, Hirsch BE. Imaging of tinnitus: a review. Radiology. 2000;216:342–9.
7. Willen SN, Einstein DB, Maciunas RJ, et al. Treatment of glomus jugulare tumors in patients with advanced age: planned limited surgical resection followed by staged gamma knife radiosurgery: a preliminary report. Otol Neurotol. 2005;26:1229–34.
8. Isaacson B, Kutz JW, Roland PS. Lesions of the petrous apex: diagnosis and management. Otolaryngol Clin N Am. 2007;40(3):479–519.
9. Jadder RK, Cho M. A new theory to explain the genesis of petrous apex cholesterol granuloma. Otol Neurotol. 2003;24:96–106.
10. Zanoletti E, Mazzoni A, Martini A, et al. Surgery of the lateral skull base: a 50-year endeavour. Acta Otorhinolaryngol Ital. 2019;39(Suppl 1):S1–S146. https://doi.org/10.14639/0392-100X-suppl.1-39-2019.
11. Lustig LR, Sciubba J, Holliday MJ. Chondrosarcoma of the skull base and temporal bone. J Laryngol Otol. 2007;121:725–35.
12. Bayazit Y, Sirikci A, Bayaram M, Kanlikama M, Demir A, Bakir K. Eosinophilic granuloma of the temporal bone. Auris Nasus Larynx. 2001;28(1):99–102.