Management of lateral skull base tumours

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Abstract

Lateral skull base tumours arise from overlying skin, parotid, temporal bone, mandible, muscle or the vasculature and they spread in midst of important neurovascular structures. This makes their diagnosis and management challenging. These tumours are primarily dealt with surgery which can be extensive, functionally disabling and cosmetically disfiguring. Tumour biology, involvement of the internal carotid artery and cranial nerves play key roles in management of these tumours. This review will focus on management of common tumours afflicting the lateral skull base.

Keywords Lateral Skull Base · parotid · glomus · temporal bone · carotid body tumour

Introduction

The skull base is a junction between the viscerocranium and the neurocranium and can be divided into the anterior, lateral and posterior skull base. The lateral skull base comprises of the region in the vicinity of the temporal bone and the greater wing of the sphenoid.

Tumour diagnosis, treatment planning and rehabilitation require multidisciplinary care and a dedicated team effort. Over the past half a century our understanding of the skull base anatomy has vastly improved along with advancement in imaging technology making way for improved more direct approaches to the skull base tumour. Chemotherapy and image guided radiation therapy have contributed towards improvement in treatment outcomes. With surgery being the key modality of treatment optimal disease clearance with minimal morbidity and mortality continues to challenge further development of skull base surgery.

Clinical presentation

It is an area filled with important neurovascular structures dealing with hearing, facial movements, swallowing and

speech. Patients with lateral skull base tumours present in advanced stages due to location of these tumours deep within. This reinforces the need for high index of suspicion for lateral skull base involvement when presented with uncommon symptoms or those which out of proportion to visible disease.

Tumours in the lateral skull base arise from the parotid, the temporomandibular joint, infratemporal fossa and the temporal bone.

Clinical anatomy

Regions

The lateral skull base can be roughly divided into the anterior temporal-infratemporal fossa region and the posterior temporal bone region. The temporal – infratemporal region is intimately related to the middle cranial fossa base, paracavernous area and orbital apex on its cranial aspect and the infratemporal fossa (ITF), parapharyngeal space, pterygopalatine fossa, and the TM joint on its subcranial aspect. The temporal bone region is again related to the MCF base and forms petrous ridge bordering the posterior cranial fossa on its cranial aspect, and subcra-

nially is related to the infralabyrinthine space and the jugular fossa. Though regional classification facilitates categorization of masses having epicentres in each of these distinct regions, tumors often transgress boundaries to involve multiple regions.

Foramina

The lateral skull base comprises of many foramina starting from the stylomastoid foramen just beyond the mastoid tip medial to the digastric belly. Parotid tumours can grow along the facial nerve which traverses this canal and tends to widen the canal. The other foramina are the foramen spinosum and foramen ovale which lie in a straight line behind the lateral pterygoid plate. Emissary veins along with the middle meningeal branch of the internal maxillary artery traverse the foramen spinosum while the mandibular branch of the trigeminal nerve, accessory meningeal artery, lesser superficial petrosal nerve and emissary veins traverse the foramen ovale and serve as conduit for perineural intracranial spread. Troublesome venous ooze can be encountered from these foramina which can be tackled by packing with hemostatic agents, pressure and by sealing them with bone wax.

Styloid process: Medial to the stylomastoid foramen is the styloid process of the temporal bone which gives attachments to the Riolsan bouquet of muscles and ligaments. The styloid process is an important landmark dividing the parapharyngeal space into the pre styloid and post styloid compartments. The pre styloid compartment contains the deep parotid gland, lymph nodes and fibrofatty tissue. The post styloid compartment contains the main neurovascular bundles traversing skullbase via the carotid canal and the jugular foramen. The styloid overlies the internal carotid artery which courses directly deep to it to enter the carotid canal medial to the jugular foramen. The styloid process is a good landmark for protecting the internal carotid artery from damage during surgery. The external carotid artery ascends above the styloid muscle and goes on to give terminal branches within the deep parotid gland.

Internal Carotid Artery (ICA) (Fig. 1): After its entry into the carotid canal at the skullbase, the midportion of the vertical ICA is anteromedial to the basal turn of the cochlea. In the distal part of the vertical segment near the genu, the ICA is just deep to the eustachian tube orifice, posteromedial to the glenoid fossa, and inferomedial to the tensor tympani muscle⁸. The horizontal segment of the ICA travels overlies the foramen lacerum travelling obliquely in a lateral-to-medial direction from the genu to the point of intracranial intradural entry at the anterior clinoid process. This part lies within the cavernous sinus

and is intimately related to the cranial nerves III, IV and VI.

Temporal bone: The mastoid emissary vein is a landmark for the position of the sigmoid sinus which can be used to begin drilling of the mastoid in order to remain lateral to the sigmoid to ensure enbloc resection of temporal bone. The glenoid fossa is formed by the tympanic part and the squamous part of the temporal bone. Complete exposure of the glenoid fossa will be required to delineate the internal carotid artery during petrous apex dissection and subtotal temporal bone resection.

Cranial Nerves: VII – The terminal branches of the facial nerve course from deeper part of the parotid gland to surface at its edge. The position of the facial nerves at level of the zygoma is within the superficial temporalis fascia and then at the level of the temporal fossa between the layers of the deep temporalis fascia. During exposure of the zygoma the plane of dissection should be subperiosteal to avoid damaging the facial nerve. Exposing the temporalis via a communicating incision between the facial and the fronto-temporal incisions care must be taken to remain above the superficial temporalis fascia. The facial nerve branches can be tagged and then either the nerves are divided or strips of the temporalis fascia and muscle beneath the facial nerve branches are separated prior to exposure of the temporal calvarium for craniotomy.

Fig. 1 Parts of internal carotid artery.

Fig. 2 Jugular Foramen – Neurovascular Structures.

IX, X, XI (Fig 2): These nerves lie in middle compartment of the jugular fossa (pars nervosa) enmeshed in a tough fibrous septum between the medial wall of the jugular venous bulb and the internal carotid artery. Preserving the medial wall of the jugular fossa during dissection will prevent these nerves from damage.

Tumour biology

Lateral skull base tumours have unique histology. Unlike other sites in the head and neck region, tumours arise from mesenchymal structures such as bone, parotid tissue, nerves and vasculature. Squamous cell carcinoma (SCC) is not the most common histology here. It is very important to understand this unique difference since surgical extirpation is a key issue which determines disease control. Control of squamous cell tumours involving skull base with adjacent natural neurovascular conduits is futile due to its propensity for perineural and perivascular spread. Unlike SCC, most tumours arising in the parotid, bone and nerves are amenable to control with narrow margin resections. Histology and tumour biology are therefore important considerations in management of lateral skull base tumours.

Parotid tumours

The most common parotid tumours are the mucoepidermoid and the pleomorphic adenomas. However the high grade variants of these tumours invade the adjacent struc-

tures necessitating lateral skull base resection. The high grade mucoepidermoid carcinoma, adenoid cystic carcinoma, salivary duct carcinoma and carcinoma ex pleomorphic adenoma are found to be more aggressive as compared to acinic cell tumour, low grade mucoepidermoid carcinoma and the polymorphous low grade adenoma.

High grade mucoepidermoid carcinomas behave like squamous cell carcinoma with aggressive spread. Survival rates are around 60% at 10 years. Adenoid cystic carcinoma is a slow growing tumour with late recurrences and distant metastasis. Amongst the different variants tubular have the least recurrence rates at 59% while cribriform at 89% and solid with almost 100% recurrences are the most aggressive end of the spectrum.¹ ACC are known for perineural spread and hematogenous distant metastasis to the lungs.

Bone and soft tissue tumours

The petromastoid part of the temporal bone, most parts of the sphenoid bone and the mandible are formed by endochondral ossification just as in the long bones. It is therefore not surprising to find chondrosarcoma, osteogenic sarcoma and giant cell rich lesions occurring here similar to that in long bones. Unlike in the long bones osteogenic sarcomas of the head and neck region are primarily treated with surgery followed by radiation. The overall survival is around 46% with a median follow up of 17 months.² Chondrosarcoma are also surgically treated with adjuvant radiation reserved for close or involved margins. 5 year survival rates are more than 70% but they do tend to manifest with local recurrences later than other disease and need long term monitoring.³

Temporal bone tumours

Amongst the rare tumours afflicting the temporal bone, tumours such as squamous cell carcinoma (SCC) in adults and mesenchymal tumours such as rhabdomyosarcoma (RMS) in paediatric age group are relatively more frequent.

SCC of the temporal bone staged by the universally accepted Pittsburgh Staging are primarily treated with surgery.⁴ Middle ear involvement, facial nerve involvement, nodal disease and intracranial extension of tumour portend a poorer survival.

RMS usually seen in the paediatric population are commonly parameningeal site of RMS and have poor survival. Treatment in RMS remains induction chemotherapy followed by surgical salvage when feasible followed by radiation therapy and maintenance chemotherapy. Prognosis of parameningeal RMS is extremely poor compared to other sites.

Paragangliomas: (carotid body tumour, glomus jugulare)

Glomus tumors of the head and neck arise from the various paraganglia. The paraganglia are part of the extra-adrenal neuroendocrine system and include the chemoreceptive areas of the carotid bifurcations, the aortic arch, and the temporal bone. Mafee et al.⁵ noted that paraganglionic cells are also found, although less frequently, in areas of the orbit (in association with the ciliary ganglion), pterygopalatine fossa (in association with the pterygopalatine ganglion), buccal mucosa, nasopharynx, larynx and dermis. However, progressive involution, which lasts until puberty, normally obliterates the paraganglionic cells of these and other extraadrenal locations. The major paraganglia that do not involute are the carotid bodies lining the medial wall of the carotid bulb. These are responsible for detecting the arterial pressure of oxygen and carbon dioxide. The most common site for glomus tumours in head neck region are in the Jugular bulb (glomus jugulare) arising in the adventitia of the dome of the jugular bulb followed by in the middle ear cavity (glomus tympanicum) arising from the glomus bodies that run with the tympanic branch of the glossopharyngeal nerve. Both of these usually present as intratemporal masses with variable intracranial and infratemporal extensions. In the vagus nerve (glomus vagale) these tumours arise infratemporally along the course of the cervical vagus nerve while in the Carotid body (Carotid body tumors) they occur at the bifurcation of the common carotid artery. Catecholamine non secreting glomus tumours present late with either large tumours or cranial nerve (VII, IX, X, XI) palsies while those patients who are secretors manifest early with symptoms of headaches, flushing and hypertension. There is a female predilection and present in the 4th to 6th decade. Multiple and bilateral paragangliomas should be ruled out prior to treatment.

Imaging of lateral skull base

CT scan and MRI scan is mandatory and complimentary for complete presurgical assessment of skull base tumours. Complete evaluation warrants contrast enhanced studies (CT and MRI) without which valuable information regarding tumor extent and involvement of vital structures will be lacking. Most tumours of the lateral skull base need primary surgery in their management. It is important to document regions involved to serve not only as a road map for the surgical team but also for radiation therapy planning. With all important structures in this region it is vital to evaluate involvement of internal carotid artery, orbit and intracranial structures. Internal carotid artery proximity or involvement is a sign which needs further assessment with cerebral blood flow evalua-

tion in the eventuality of ligation of the ICA. In temporal bone cancers involvement of venous sinuses requires documentation of cerebral venous drainage with MR venogram or a conventional venogram. In histologies such as adenoid cystic carcinoma it is vital to evaluate nerves in the vicinity of tumour for perineural spread i.e. the facial nerve and the trigeminal nerve. For intracranial spread it is important to document dural involvement, dural enhancement and brain involvement. In benign tumours bony rehabilitation with prefabricated implants is possible with stereolithography and it requires images generated with CT scans preferably 3D scan. Thus imaging in lateral skull base tumours guides us:

1. For evaluation of involved structures and extent of disease.
2. Status of the cerebrovascular circulation – ICA and venous drainage.
3. Perineural spread.
4. Dural Enhancement and Brain involvement.
5. Image information can be gathered for rapid prototyping with stereolithography.

Internal carotid artery

Skull base surgery has evolved with multidisciplinary teams to remove extensive lesions. However the internal carotid artery (ICA) remains a barrier towards the goal of complete removal of tumours. Before surgical procedure is undertaken we have to ensure that cerebral circulation is maintained in the event of total occlusion or ligation of the ICA which should be avoided at all costs. The sequelae of ICA ligation is stroke in more than 25% of patients⁶ (4). The brain is intolerant to reduced oxygenation for more than a few minutes. It is important to assess for adequacy of crossflow across the Circle of Willis formed by the anterior and posterior communicating arteries. Manual compression of the internal carotid artery is unreliable. Balloon test occlusion (BTO) is standard of care to occlude the ICA with certainty, measure proximal back pressure and check for neurological signs after complete occlusion. Patient should have at least 60–70 mmHg of back pressure and have no neurological deficits for 15–20 min which ensures that there is adequate cerebral blood flow. This is fairly accurate in predicting reduced risk of stroke by 90%. There still remains risk of delayed ischaemia which can be ascertained with cerebral blood flow reserve studies such as xenon CT scan or Technetium –99 m hexamethylpropyleneamine oxime (^{99m}Tc-HMPAO).^{7–9} Others techniques include PET scan, single photon emission CT (SPECT) scan, and functional MR imaging testing, none of which have gained broad acceptance but show great promise. Proper planning and decision on management of the ICA should be done preoperatively. For those patients who

might need ICA occlusion and do not withstand the BTO, a revascularisation procedure should be available with a vascular surgeon as standby.

Dural/intracranial involvement

Dural enhancement extending beyond surgical margins and gross brain involvement are poor prognostic indicators. Much controversy exists regarding the significance of radiologically enhancing dura. Often reactive changes may mimic tumor infiltration and complicate therapeutic decision making. Though evidence is equivocal dural enhancement more than 5 mm thick, especially discontinuous enhancement, nodular enhancement and pial invasion are better predictors of dural involvement by tumor.^{10,11}

Treatment decisions

Incision planning

As with all incisions the most important consideration is of access to the tumour. With malignant tumours another issue is the involvement of the overlying skin. The involved skin is marked for excision and incorporated into the standard incisions. The incision should be placed in a vascular area and if needed be extendable to account for disease extensions. Finally the incision should be cosmetic.

Access

The epicentre of the lateral skull base tumour determines the access. As a rough guide, the location of the tumor in relation to the ICA determines the route of entry. Broadly the access centres around the pinna.

1. Preauricular frontotemporal access: For tumours located anterior to the ICA which includes tumors of the parotid, mandible and the temporal and infratemporal fossae, including orbital apex.
2. Post auricular transmastoid access: For tumors located posterior to the ICA viz. tumours of the temporal bone and infratemporal region. This trajectory allows for a better control of the ICA in its intratemporal course.

The incision is placed around the pinna. The pre auricular access incorporates the modified Blair's and a frontotemporal incision while the post auricular access incorporates mastoidectomy with a cranial and a neck access.

Operative objectives

Parotid tumours: Parotid tumours which encroach on the temporal bone and the infratemporal fossa require

extended radical parotidectomy with adjacent structures. The most common involvement is that of the temporal bone. Following a radical parotidectomy the temporal bone is removed as per involvement. The algorithm followed is the same as that of the temporal bone tumours. The objective remains to remove the base of the tumour which involves adjacent bone with wide margins. Bone cannot be assessed for margin by frozen section and hence removal of involved structure will ensure a complete tumour clearance.

Temporo-mandibular joint and temporal fossa tumours: Lesion involving these regions can be encompassed with resection of the tumour along with removal of the parotid gland, glenoid fossa and temporomandibular joint, temporalis muscle and the vertical ramus of the mandible with masseter and the pterygoid muscles. A temporal craniotomy can be undertaken with a frontotemporal access for tumours with intracranial extension. Further medially the zygoma and maxilla can be resected for tumour extensions on the posterolateral wall of the maxilla or the pterygoid plates.

Temporal bone tumours: Temporal bone tumours can involve either the external canal or extend to the middle/inner ear (Fig. 3). In tumours limited to the external canal, lateral temporal bone resection (LTBR) is the procedure of choice where essentially the entire bony external canal is resected lateral to the facial nerve along with the tympanic membrane, incus and malleus. When the middle ear is involved, subtotal temporal bone resection (STBR) can encompass the tumour. STBR involves resection of the external auditory canal with tympanic membrane and the ossicles along with part of the inner ear and sacrifice of the facial nerve. Following LTBR if the disease is not infiltrating the bone, the histology is low grade and adjuvant radiation therapy is not indicated then a type III tympanoplasty can be done and hearing preserved. In all other resections of the temporal bone adjuvant radiation therapy is indicated and a proper cul-de-sac closure of the mastoid bowl using abdominal fat and temporalis muscle cover is mandatory to avoid osteoradionecrosis.

Carotid body tumours (CBT) (Table 1) are accessed directly via a cervical approach. Shamblin classified CBT according to the involvement of the carotid artery. The Type I and II tumours can be resected with least morbidity. They type III which can involve the intima of the vessels requires a bypass and graft after resecting the tumour.

Glomus tumours (Table 2) which are Type A can be resected with a regular post aural access and a transcanal resection. For Type B tumours a mastoidectomy may be sufficient. For Type C and D infratemporal fossa approach as described by Fisch (Fisch A) will be required. It involves complete delineation of the carotids and jugular vein and the cranial nerves. This is followed by a complete mastoidectomy and facial nerve decompression and rerouting.

Table 1. Paragangliomas – carotid body tumour, glomus jugulare

Carotid Body Tumour – Shamblin ¹² based on the size and the difficulty of surgical resection	Glomus – Fisch Based on involved structures
Grade-1 Small tumour easily resected from vascular elements.	Type A Limited to the middle ear cleft (glomus tympanicum)
Grade 2 Medium-sized tumour closely associated with vascular structures, which can be resected by careful subadventitial dissection.	Type B Limited to the tympanomastoid area with no infralabyrinthine compartment involvement
Grade-3 Large tumor encasing the carotid arteries and can be resected only by partial or total vascular resection requiring vascular replacement.	Type C Involving the infralabyrinthine compartment of the temporal bone and extending into the petrous apex C1 – Limited involvement of the vertical portion of the carotid canal C2 – Invading the vertical portion of the carotid canal C3 – Invasion of the horizontal portion of the carotid canal
	Type D1 Intracranial extension < 2 cm in diameter
	Type D2 Intracranial extension > 2 cm in diameter

Fig. 3 Algorithm for management of cancers involving the temporal bone.

The carotid canal and petrous ICA is exposed and the tumour is resected trying to preserve the medial wall of the jugular bulb. This will ensure that the cranial nerves that exit between the internal carotid and the jugular bulb are protected from the dissection and thermal damage.

Schwannomas: A combined cervicomastoidectomy approach gives a good access to schwannomas expanding the jugular fossa. The cervical access exposes the tumour in the parapharyngeal space while the mastoidectomy

along with removal of the mastoid tip exposes the jugular bulb. This gives access to the IX, X, XI nerves as they arise in the medial wall of the jugular fossa.

Reconstruction

The lateral skull base tumours involve parotid with or without facial nerve, temporomandibular joint, temporal bone with or without pinna and calvarium of the temporal fossa. Lateral skull base tumour resection usually causes

Table 2. Reconstruction of lateral skull base defect

Site	Reconstruction – local flaps	Free tissue transfer
Pinna and/or periauricular skin	Axial pattern Scalp flap Cervico-delto-pectoral advancement Pedicled flaps: Submental artery platysmal flap Pectoralis major myocutaneous flap Trapezius myocutaneous flap	Free forearm radial flap
Mastoid cavity	Temporalis muscle flap	Abdominal fat
Calvarial defect	–	Anterolateral thigh flap
Vertical Ramus of mandible		Rectus abdominus flap
Zygoma/Infratemporal fossa		

large volume defects. The objective of reconstruction in skull base surgery is to repair or seal the dura. Recreate the barrier between the intra and extra cranial structures and restore form and function. These objectives are tough but we should strive to achieve them.

Precautions during reconstruction of lateral skull base defects

Dural seal should be achieved adequately with water tight closure and effective support. The dura is a very resilient barrier and must be treated with utmost respect. Unnecessary breach of the dura should be avoided. Studies have shown that risk of infection is higher once the dura is breached even if it is appropriately closed.

Temporal lobe of the brain should be enclosed adequately and supported with soft tissue cover. Peroperatively undue retraction on the lobe should be avoided. CSF drainage may be used to facilitate retraction. Vein of Labbe coursing obliquely along the temporal cortex must be preserved to avoid fatal venous infarction. If adjuvant radiation is not contemplated then calvarial defect should be reconstructed with split calvarial graft or titanium plate made with help of rapid prototyping.

Eustachian tube opening is packed with muscle prior to filling the mastoid bowl with abdominal fat. All epithelium is cleared off to avoid cholesteatoma formation.

Facial nerve grafting should be attempted if two ends of the nerve are visualised.

Rehabilitation

Lateral skull base surgery affects form and function. Hearing, balance, mastication, swallowing and airway protection can be affected.

Immediate post operative period

- Following temporal bone procedures the patient can have giddiness due to excessive handling of the stapes or injury to the inner ear. Labyrinthine sedatives and entiemetics have to be given round the clock till compensation occurs.
- Following jugular fossa dissection lower cranial nerves are in jeopardy and the patient can manifest hoarseness, aspiration, shoulder dysfunction and dysarthria. Nasogastric feeding, fat thyroplasty and if required even tracheostomy for airway protection should be done to ensure adequate nutrition and protection of the lungs.
- TM joint dysfunction: Removal of the TM joint causes minimal morbidity with exaggerated lateral movement of the jaw during mastication. Most patients recover function with a guide bite plane prosthesis which is recommended for use for 3–6 months.

Long term rehabilitation

- Facial reanimation with static or dynamic slings, gold implant in the upper eyelid in case the facial nerve continuity is not restored during surgery.
- Voice rehabilitation may be done with thyroplasty with damage to the X nerve.
- For persistent aspiration arytenoid and vocal fold medialisation and cricothyroid tensing (triple) procedure is undertaken.
- Dysphagia can be aided with percutaneous endoscopic gastrostomy and nasal regurgitation prevented with palatal adhesion procedures.

Outcomes

There are few reports of outcomes of lateral skull base malignant tumours. McGrew et al.¹³ from the Otolaryngology

group at Nashville, Tennessee, USA published a retrospective review of 95 patients treated for lateral skull base malignancies. The mean age of the patients was 49.4 years. There were 44 females and 51 males. The mean follow-up period was 50 months. Resection was performed in all patients, and postoperative radiotherapy was undertaken in 54% of the cases. Local disease control was maintained in 73% of the patients. Tumour involvement of the facial nerve and intracranial tumor extension did not jeopardize the rate of local control. Manolidis et al.¹⁴ published their series of 81 cases dividing them into three groups, epithelial malignancies, 30 patients; malignancies of salivary gland origin, 23 patients; and malignancies of mesenchymal origin, 28 patients. With a mean follow-up time was 54 months (1–180 months) 43 patients were alive without evidence of recurrent disease, giving a n overall survival of 58%. Epithelial and salivary malignancy had a poorer prognosis, and pain and facial nerve dysfunction at presentation carried a worse prognosis.

At the Tata Memorial Hospital, a retrospective study from 1989 to 2001 had 47 lateral skull base tumours in-

cluding 33 temporal bone tumours, 9 orbit tumours, 5 infra temporal tumours. With a median follow up of 27 months our 5 year overall survival is 51%. There was one incidence of meningitis and that settled down with antibiotics. With a dedicated skull base clinic with multidisciplinary tumour board our prospective data has 110 patients of lateral skull base (71 temporal bone tumours, 19 parotid tumours, 10 orbit tumours, 3 infra temporal tumours and 7 parapharyngeal space tumours). Long term results of the management of these tumours will be published in the coming years.

Conclusions

Lateral skull base tumours are rare entities that continue to challenge cranial base surgeons. With improved imaging, powered instruments, better understanding of tumours biology and free tissue transfer, the overall morbidity has reduced. Unlike in the past, cranial nerve deficits and complications are less frequent. Proper planning and meticulous surgery is the key to success.

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