Temporal Bone Malignancies

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KEYWORDS

• Temporal bone cancer • Temporal bone resection • Squamous cell carcinoma
• Basal cell carcinoma

KEY POINTS

- Squamous cell carcinoma accounts for 60% to 80% of the tumors that arise in the ear canal, middle ear, or mastoid cavity.
- Otorrhea, otalgia, and hearing loss are the most common symptoms of temporal bone tumors and can be confused with benign disease.
- Preoperative staging requires computed tomography, magnetic resonance imaging, or both. The University of Pittsburgh staging system is useful for treatment planning and prognostication.
- Lateral temporal bone resection is required to treat most tumors. Auriculectomy, parotidectomy, mandibulectomy, craniotomy, and neck dissection are performed based on staging and location of tumor. Temporalis muscle flap or microvascular free flap are reconstruction options.
- Adjuvant radiotherapy is recommended for temporal bone tumors staged T2 and higher. Adjuvant chemotherapy has an emerging role for T3 and T4 tumors.

EPIDEMIOLOGY

Primary tumors that affect the temporal bone are rare. Primary ear canal cancers or middle ear cancers occur at an estimated rate of 1 person per million people per year.[1–3] It is estimated that cancer is the underlying cause in only 1 in every 5000 to 20,000 patients with an otologic complaint.[4] Temporal bone carcinomas account for only about 0.2% of all head and neck cancers.[5] Sun exposure is linked with skin cancers, and radiotherapy has been linked with squamous cell cancer of middle ear and ear canal.[6] Some investigators have linked chronic otitis media and cholesteatoma to ear canal and middle ear cancer,[6–9] but this cause probably accounts for only a few tumors in most modern studies. For most patients, the cause is unknown.

Because primary ear canal and middle ear cancers are so rare, the temporal bone is more likely to be affected secondarily from advanced periauricular skin cancer or the parotid gland tumors than from primary tumors.[10]

Tumors affecting the temporal bone can occur in all age groups, but typically occur in older patients, especially in men. In a large series of temporal bone cancers, 75% of patients were men, and the average age was 65 years.[11] The tumor histologies tend to trend with age, so that younger patients are likely to have sarcomas and older patients are likely to have carcinomas.

SIGNS AND SYMPTOMS

The signs and symptoms of these tumors are vague and can be confused with benign disease. Otorrhea, otalgia, and hearing loss are the most common symptoms of temporal bone tumors.[12] However, these symptoms are frequently seen in patients with otitis externa, otitis media, or cholesteatoma. Most patients with benign disease respond to aural toilet and eardrops or oral

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medications. Suspicion should arise when patients with these symptoms do not respond to standard therapy.

Otorrhea, otalgia, and hearing loss make up a classic triad for temporal bone cancer, but this classic triad is seen in only 10% of patients who have temporal bone cancer. Other symptoms, such as trismus, facial weakness, dysphagia, and hoarseness, are seen less commonly and are usually associated with advanced-stage disease.

The duration of symptoms can vary from months to several years.[12,13] Survival has been linked to symptom duration.[13] For this reason, a high index of suspicion should be maintained when symptoms do not resolve with standard therapy for benign diseases.

The physical examination of these patients demands close scrutiny of the external ear, ear canals, tympanic membranes, parotid gland, periauricular skin, cervical lymph nodes, and cranial nerves. Microscopic examination of the ear canal is important to determine the extent of disease into the ear canal. Tumors of the external ear that do not extend medially past the bony-cartilaginous junction in the ear canal can often be dealt with by local excision. However, tumors that involve the bony ear canal require, at a minimum, lateral temporal bone resection (LTBR) to achieve a negative medial margin.

Skin cancers involving the ear canal have an exophytic or ulcerated appearance (**Fig. 1**).

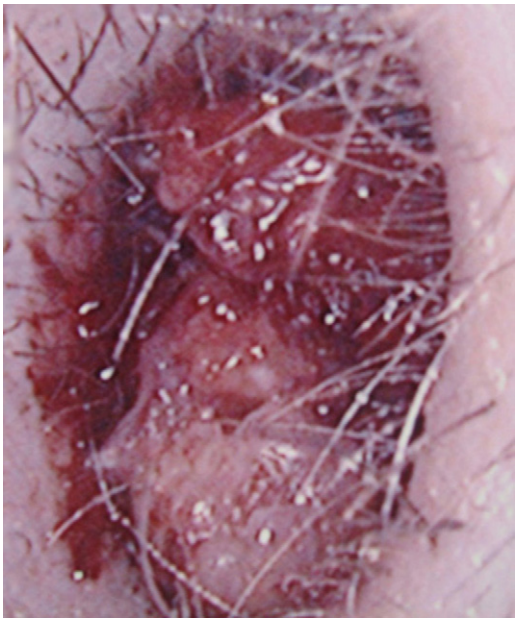

Fig. 1. Squamous cell carcinoma filling the left ear canal. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

Squamous cell carcinoma (SCCa) can be heralded by erythematous skin and granulation tissue. Basal cell carcinoma usually has an ulcerated appearance with rolled edges. Adenoid cystic carcinoma (ACCa) in its early stage is often subcutaneous. Occasionally, some tumors have a subcutaneous spread and a cursory examination of the canal might miss the ear canal involvement (**Fig. 2**).

When patients with these symptoms do not respond to standard therapy, then any suspicious tissue should be sent for pathologic evaluation. The differential diagnosis for disease in the ear canal should include skull base osteomyelitis (also called malignant otitis externa), pseudoepitheliamotous hyperplasia, and carcinoma.[14] The temporal bone and ear canal are a rare location for metastatic lesions, usually from breast, lung, or kidney primaries.[15,16]

Facial paralysis, when it occurs, it is an ominous finding. It is linked with a poor prognosis.[17] In a series from MD Anderson Cancer Center, approximately 40% of patients had various degrees of facial weakness or paralysis at presentation.[11]

Cervical lymphadenopathy is a particularly poor prognostic sign associated with worse survival.

LOCATION OF PRIMARY TUMOR

Primary tumors of the ear canal or temporal bone are rare. In our patient population, those 2 primary sites account for only 25% of our total patient population (**Fig. 3**).[11] Instead, the temporal bone is more likely to be invaded by cancers of the parotid

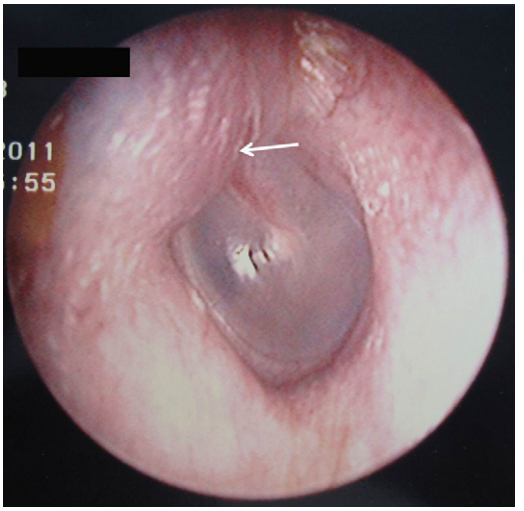

Fig. 2. Subtle left anterior-superior ear canal swelling (*arrow*) caused by infratemporal fossa chondrosarcomas. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

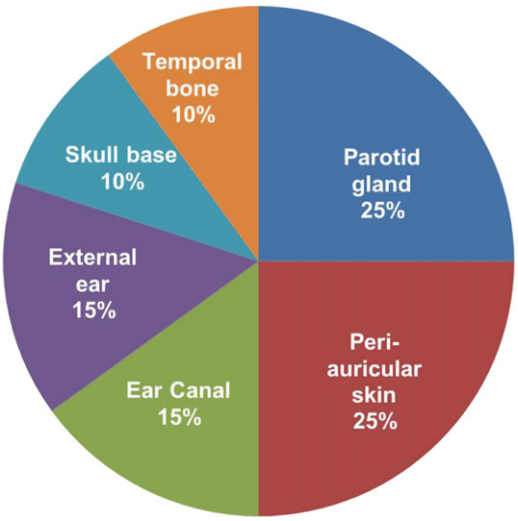

Fig. 3. Location of temporal bone primary tumors. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

gland or periauricular skin. These 2 primary sites combine for 50% of our temporal bone patient population. Advanced, neglected external ear cancers that have grown into the ear canal and skull-based cancers, primarily sarcomas, make up the remaining 25% of our patient population.

HISTOLOGY

A long list of tumor types has been described as affecting the temporal bone (**Box 1**).[11] Squamous cell and basal cell carcinoma account for more than 50% of the tumors if all primary tumor sites are considered. When primary locations outside the temporal bone are excluded, SCCa accounts for 60% to 80% of the tumors that arise in the ear canal, middle ear, or mastoid cavity.[18–20] Basal cell carcinoma and ACCa are the next 2 most common tumors found in the ear canal.

Box 1
List of malignant tumor types found affecting the temporal bone

Epithelial
SCCa
Basal cell carcinoma
ACCa
Basosquamous carcinoma
Hidradenocarcinoma
Melanoma
Sarcomatoid carcinoma
Sebaceous cell carcinoma

Sarcomas
Chondrosarcoma
Osteosarcoma
Pleomorphic sarcoma
Spindle cell sarcoma

Salivary
ACCa
Acinic cell carcinoma
Adenocarcinoma
Basal cell adenocarcinoma
Carcinoma ex-pleomorphic adenoma
Malignant mixed carcinoma
Mucoepidermoid carcinoma
Salivary ductal carcinoma

Other
Clivus chordomas
Hemangiopericytoma
Neuroendocrine carcinoma
Peripheral nerve sheath tumor

DIAGNOSTIC IMAGING

Diagnostic imaging is imperative for understanding the three-dimensional anatomy of these tumors. Computed tomography (CT) and magnetic resonance imaging (MRI) provide complimentary details.[21] A CT scan is generally obtained as an initial study. It gives an excellent view of soft tissue and bony anatomy. Because surgery is the mainstay of temporal bone cancer treatment, the bony anatomy is especially important for temporal bone surgeons. A high-riding jugular bulb and anomalous carotid artery are important to recognize and are readily identifiable on CT imaging (**Figs. 4** and **5**).

Given that MRI lacks bony detail, its use is reserved as an adjunct especially in cases in which dural involvement or perineural invasion is suspected (**Figs. 6** and **7**).

Arriaga and colleagues[22] considered 12 important sites of involvement to identify and study on CT and MRI. These sites include the 4 quadrants of the ear canal, the infratemporal fossa, middle ear, otic capsule, mastoid, jugular foramen, carotid canal, tegmen, middle fossa, and posterior fossa.

Leonetti and colleagues[23] looked at different paths of invasion of temporal bone tumors. They identified that tumors grow superiorly through the tegmen, anteriorly through the glenoid fossa and

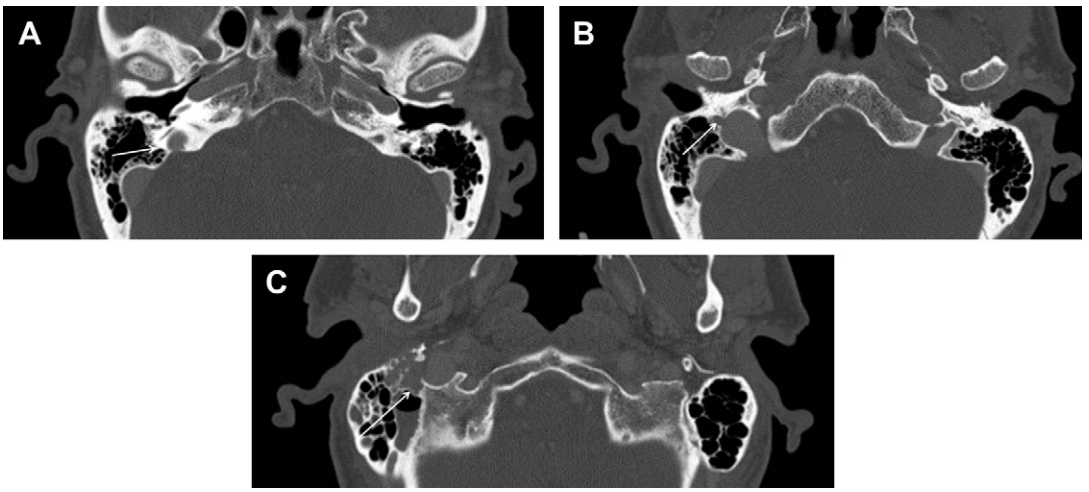

Fig. 4. CT of right parotid tumor with perineural invasion of facial nerve destroying stylomastoid foramen. (*A*) High-riding jugular bulb present at level of posterior semicircular canal (*arrow*). (*B*) Facial canal contiguous with jugular bulb (*arrow*). (*C*) Stylomastoid foramen destroyed by tumor. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

infratemporal space, inferiorly through the hypotympanum and jugular foramen, posteriorly into the mastoid, and medially through the carotid canal and inner ear. They noted that anterior and inferior spread is accurately assessed by CT scan and MRI; however, they found that there was underestimation of disease when it involved mastoid and middle ear mucosa, the tegmen tympani, the middle fossa dural, or the carotid canal. Local recurrences were found in these 4 areas on retrospective review.[23]

No staging system has been approved for temporal bone cancers by the American Joint Committee on Cancer. Several staging systems have been proposed.[3,24–27] The first iteration of the University of Pittsburgh staging system was proposed by Arriaga and colleagues[25] in 1990 and was modified more recently by Moody and colleagues[5] (**Table 1**). This staging system provides a comprehensive means of assessing these tumors, and the Pittsburgh T stage has been shown to predict overall survival.[12]

Early-stage T1 tumors are limited to the ear canal without any bony erosion or soft tissue involvement. T2 tumors have limited ear canal bony erosion or limited soft tissue involvement.

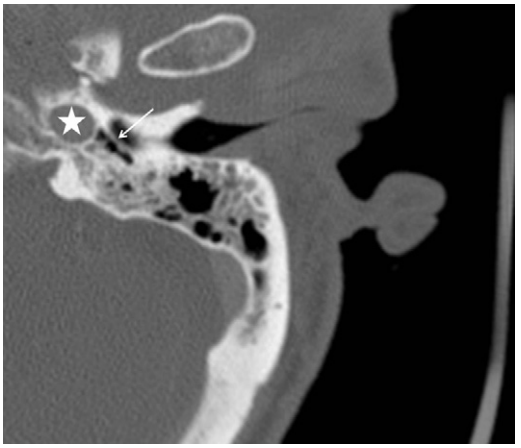

Fig. 5. CT of left temporal bone showing inferior bony annulus (*arrow*) at level of carotid canal (*star*). Patient has large parotid tumor necessitating LTBR. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

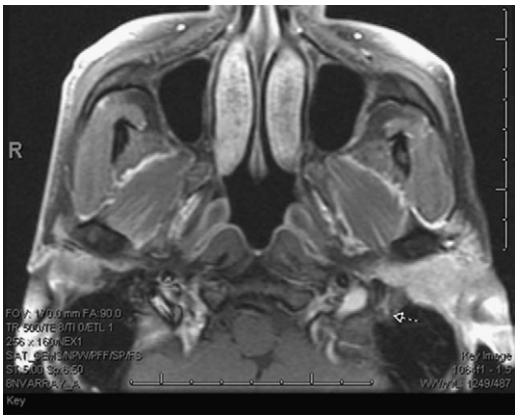

Fig. 6. Axial T1 with contrast MRI showing enhancement of intratemporal facial nerve (*arrow*) caused by perineural invasion from parotid tumor. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

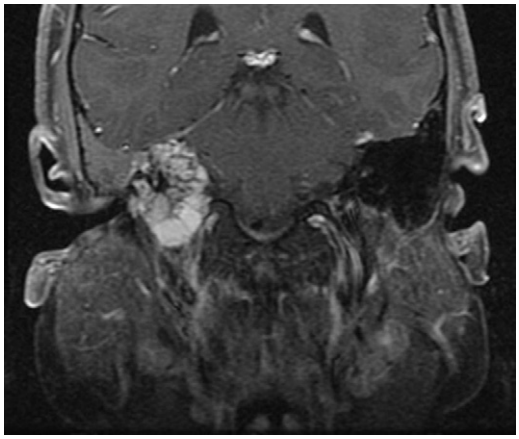

Fig. 7. Coronal T1 enhanced MRI of large right endolymphatic sac tumor showing tumor largely contained by the dura. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

T3 tumors are larger tumors that erode the bony ear canal or have limited soft tissue involvement or begin to involve the middle ear or mastoid. T4 are large tumors that involve the inner ear, the carotid canal, the jugular foramen, and the dura or have evidence of facial paresis. The presence of facial paralysis is an important change in the current system of staging when compared with the original iteration.[28]

Given that cervical lymphadenopathy carries a poor prognosis, overall staging for temporal bone tumors is different from staging for other head and neck tumor sites. T1N0 tumors are stage 1, T2N0 tumors are stage 2, and T3N0 tumors are stage 3. However, stage 4 tumors include T4N0 and any T with N+ status.[25]

TREATMENT: SURGERY

Although single modality radiotherapy has been reported as a treatment of temporal bone tumors,[29–31] surgical resection has been considered the standard of care. Surgical management of these tumors is predicated on and tailored to the extent of disease. The goal of surgery is to extirpate disease, achieving a negative margin and minimizing morbidity or mortality.

Local Canal Excision

Small, early staged tumors that are confined to the cartilaginous outer ear canal can be managed with a wide local excision.[19] This situation occasionally arises in the case of a tragal-based or conchal-based tumor that extends into the membranous ear canal. This procedure is usually performed under general anesthesia using the operative microscope. An endaural incision helps with visualizing tumor extent. Skin and underlying cartilage are excised using frozen-section pathology for margin evaluation. Reconstruction is performed with a split-thickness skin graft.

Table 1
Modified Pittsburgh staging system[5] for SCCa of the temporal bone

T Classification	Description
T1	Limited to the EAC without bony erosion or evidence of soft tissue involvement
T2	Limited to the EAC with bone erosion (not full thickness) or limited soft tissue involvement (<0.5 cm)
T3	Erosion through the osseous EAC (full thickness), with limited soft tissue involvement (<0.5 cm) or tumor involvement in the middle ear or mastoid
T4	Erosion of the cochlea, petrous apex, medial wall of the middle ear, carotid canal, jugular foramen, or dura, with extensive soft tissue involvement (>0.5 cm, such as involvement of the TMJ or styloid process), or evidence of facial paresis
N classification	
N0	No regional nodes involved
N1	Single metastatic regional node <3 cm
N2	
N2a	Single ipsilateral metastatic node 3–6 cm
N2b	Multiple ipsilateral metastatic lymph nodes
N2c	Contralateral metastatic lymph node
N3	Metastatic lymph node >6 cm
Overall Stage	
I	T1N0
II	T2N0
III	T3N0
IV	T4N0 and T1-4N+

Abbreviations: EAC, external auditory canal; N+, any positive lymph node; TMJ, temporomandibular joint.

The skin of the bony ear canal is thin, and achieving a negative deep margin is difficult once the tumor reaches the bony ear canal. If the medial (or bony canal) extent of the tumor cannot be cleared, then the procedure should be converted to an LTBR. Anecdotally, surgeons have tried to remove the skin from the bony ear canal, the so-called sleeve resection, reconstructed the defect with a split-thickness skin graft and used radiotherapy as a salvage therapy. This situation usually develops into a narrow ear canal with exposed bone and chronic drainage and can lead to osteoradionecrosis of the temporal bone. LTBR provides better tumor control and overall survival for these patients.

LTBR

The real workhorse of otologic oncologic surgery is the LTBR. This procedure removes the bony canal en bloc lateral to the facial nerve (**Fig. 8**). The specimen includes the tympanic membrane, malleus, and incus. The stapes, facial nerve, and inner ear structures are preserved. The amount of cartilaginous ear canal and pinna resected depends on the tumor extent in these structures. LTBR can be combined with parotidectomy, neck dissection, mandibulectomy, and craniotomy. Reconstruction technique depends on the defect and is influenced by previous irradiation.

General considerations LTBR with parotidectomy, neck dissection, and possible microvascular free-flap reconstruction usually requires 8 to 12 hours of general anesthesia. The patient's general health must allow such a surgical procedure. Intraoperative facial nerve monitoring is always used, and this fact must be communicated to the anesthetist to avoid use of long-term paralytics. Antithromboembolism techniques, thromboembolic deterrent hoses, and sequential compression devices are also used.

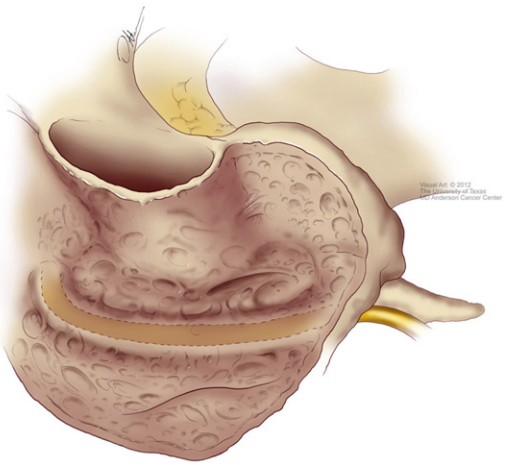

Fig. 8. Line facial nerve dissection as part of right LTBR. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

Skin incision The surgeon has a choice of incisions for temporal bone resection. This decision is based on location of tumor, the need for auriculectomy, parotidectomy or neck dissection, previous incisions, and the need for craniotomy. A large postauricular C-shaped incision is perhaps the most versatile incision and is used when the auricle is normal. The auricle can be elevated with a wide anteriorly based skin flap. This incision gives access for parotidectomy, neck dissection, and craniotomy.

When auriculectomy is required, an incision that circumscribes the tumor and auricle is used. Surrounding skin can be undermined, giving enough exposure for LTBR, parotidectomy, and neck dissection. Limited middle fossa craniectomy can be performed through this approach. This situation produces the largest soft tissue defect and generally requires a microvascular free flap for closure.

When the tumor is confined to the ear canal, a modified parotidectomy incision can be combined with an elliptical incision around the external meatus. This incision is especially advantageous in the circumstance of recurrent disease after previous parotidectomy. The incision can be extended anterior to the helix and superiorly into the temporal scalp. The remaining pinna is elevated with a broad posteriorly based skin flap. This incision is probably best for T1 and T2 ear canal cancers and parotid-based tumors that involve the ear canal. Parotidectomy and neck dissection are performed, and reconstruction can be with either a temporalis muscle flap or a microvascular free flap. The posteriorly based skin flap containing the pinna has some disadvantages: (1) the pinna is folded or retracted, which could compromise its blood supply, and (2) the surgeon has to work over the flap containing the pinna.

Bony dissection The goal of LTBR is en bloc resection of tumors involving the external auditory canal. The strategy to achieve this goal is bony dissection to free the external canal. After skin incisions, a complete mastoidectomy is performed. The tegmen is thinned and used as a guide to judge the level of the middle fossa dura. The antrum is opened, the attic is widened, and the incus and malleus head are identified. The horizontal semicircular canal is preserved throughout this dissection.

Bone at the root of the zygoma is removed, and the temporomandibular joint is identified at its

lateral bony margin. Drilling continues through zygomatic air cells, working lateral to the ossicular chain and between the bony ear canal and the tegmen. Care is taken to avoid a dural tear. Equally, care is taken to avoid violating the external ear canal bone and spilling tumor into the operative field. This dissection is complete when the entire temporomandibular joint capsule is exposed from medial to lateral.

The facial recess is opened, and the middle ear is checked for disease. (If disease is found in the middle ear, the surgical procedure needs to expand beyond an LTBR.) The facial nerve is identified at its tympanic segment and followed through its mastoid course to the stylomastoid foramen. The digastric ridge is drilled away to identify the underlying digastric muscle. A cut is made in the anterior wall of the mastoid tip, lateral to the facial nerve and inferior to the tympanic ring. This maneuver liberates the mastoid tip, and its soft tissue attachments are cut with electrocautery. Removal of the mastoid tip unifies the mastoid with the neck, and it allows the facial nerve to be traced from the mastoid into the parotid gland.

The facial recess is then extended, and the chorda tympani nerve is divided. The annulus is identified, and drilling continues between the annulus and the facial nerve until the hypotympanum is reached. The inferior tympanic bone is removed, and the soft tissue anterior to the tympanic ring is identified. In making the inferior final cut, care must be exercised to avoid having the shaft of the drill rest on the facial nerve. Only prudent surgical technique can avoid injury to the facial nerve here, because the facial nerve monitor does not alarm because injury to the facial nerve from the drill shaft is thermal.

The final bony cuts are made to free the anterior-inferior extent of the tympanic ring. Occasionally, the carotid artery is in contact with the annulus here. Vigilance is required to avoid injury to a laterally placed carotid artery.

The incus is disarticulated. Thumb pressure on the canal causes it to fracture anteriorly. A Freer elevator can be used to ensure that the canal is entirely mobilized, but care must be taken to avoid using the facial canal or middle fossa dura as a fulcrum.

Facial nerve management The mastoid segment of the facial nerve can be decompressed, and the nerve traced into the parotid gland. The facial nerve can be assessed for perineural disease. Perineural disease can produce a nerve that is thicker and redder than usual. If the patient has normal facial function preoperatively, then efforts should be made to preserve this function. If the patient has preexisting facial paralysis and facial nerve sacrifice is planned, then the nerve can be divided and the proximal end sent for frozen-section evaluation. A tumor-free margin should be sought; however, a labyrinthectomy is not performed for the reason of trying to achieve a negative facial nerve margin. Our philosophy has been to treat this remaining microscopic disease with postoperative radiotherapy. Successful facial nerve grafting and reasonable survival can be achieved even when microscopic disease remains in the proximal segment of the facial nerve.[32]

If nerve sacrifice is required and if usable proximal and distal segments are available, then an attempt should be made at facial nerve grafting.[33,34] The great auricular nerve is a reasonable choice for short defects; however, the facial nerve defects that result from LTBR and parotidectomy are usually too long to be grafted with the greater auricular nerve. Sural nerve and the cutaneous branch of the anterior femoral nerve, which is in the field of the anterior lateral thigh flap, are better options.[33]

Some tumors can be adequately treated with LTBR alone; however, most cases require concomitant parotidectomy and upper neck dissection (level II and III). These procedures commence after LTBR. Large auricular cancers and large parotid tumors can be resected en bloc along with the ear canal. This surgery creates a composite resection that includes the external ear, ear canal, parotid, and neck dissection and occasionally the upper mandible as 1 single specimen (**Fig. 9**).

Reconstruction Historically, surgeons reconstructed the lateral temporal bone defect by covering the cavity with a split-thickness skin graft.[35] This technique offered patients an option for hearing reconstruction. However, these cavities can develop serious, bothersome drainage and infection, especially after radiotherapy. Furthermore, these patients tended to have poor hearing outcomes, given the possible chronic infection and sensorineural hearing loss from radiotherapy.

More modern techniques of reconstruction emphasize closing off the cavity from the outside world. The type of reconstruction depends on several factors: size of defect, history of preoperative radiotherapy, vascular and dural coverage, and cosmesis. Small, uncomplicated defects can be reconstructed with temporalis muscle flap.[36] A split-thickness skin graft can be used to cover the flap and to close a small cutaneous defect. However, microvascular free-flap reconstruction is required in most circumstances, especially in

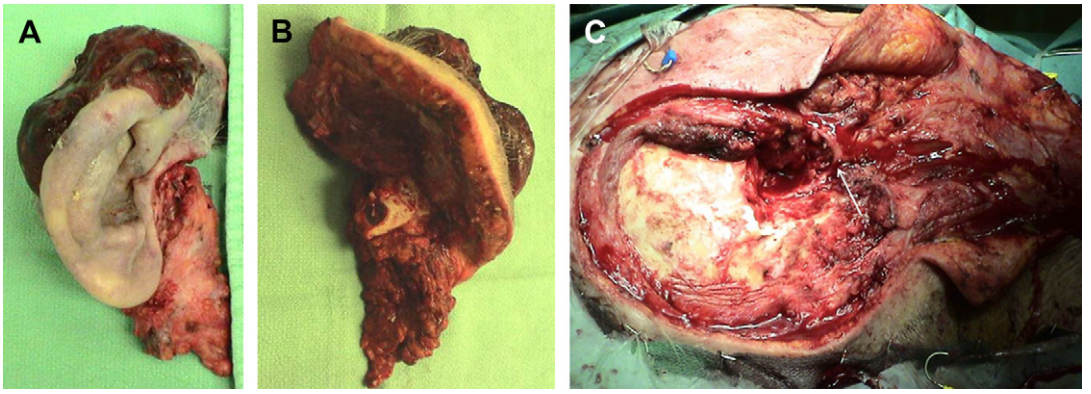

Fig. 9. Surgical specimen and defect after composite temporal bone resection. (*A*) Lateral surface showing large periauricular tumor. (*B*) Medial surface of specimen showing intact ear canal and eardrum. (*C*) Surgical defect after total auriculectomy, LTBR, neck dissection, and parotidectomy. Note the course of the facial nerve (*arrow*). This defect was closed with an anterolateral thigh flap. (*From* the Department of Head and Neck Surgery, MD Anderson Cancer Center; with permission.)

patients with large skin defects or previous radiotherapy or when the dura or vascular structures need coverage.[34,37,38]

When facial nerve sacrifice has occurred, techniques for facial rehabilitation and eye protection should be considered at the time of the primary surgery. Gold weights, tarsorrhaphy, canthoplasty, brow lifts, and static slings each have a role to play. Oculoplastic surgeons are invaluable members of the team to help manage eye complications related to facial paralysis.

Osseointegrated implants offer patients an option for hearing restoration and for prosthesis fixation. Implants can be placed either at the time of primary surgery or at a time after completion of therapy. Loading can begin in 3 months in patients not treated with radiotherapy. However, 6 months are allowed to pass before using an implant in an irradiated field.

Subtotal temporal bone resection

Tumors that extend into the middle ear need surgery beyond the LTBR.[26] Subtotal temporal bone resection (STBR) extends the dissection into the labyrinth, the cochlea, or both. Often, LTBR is required as an initial first step to permit access to deeper structures.

Skin incisions These tumors tend to have their epicenter in the middle ear or mastoid and thus have limited ear canal involvement. A large C-shaped postauricular incision works well. Frozen section is used to confirm a negative ear canal margin, and the ear canal is oversewn.

Bony dissection LTBR is performed to remove the disease contained within the ear canal. This procedure gives an unimpeded view of the middle ear, inner ear, and carotid-jugular triangle. Dissection is directed by disease extension. Labyrinthectomy, jugular foramen dissection, and cochlectomy are performed as needed depending on disease extent. The eustachian tube mucosa can be involved, and the carotid artery often must be dissected and decompressed. Tumor dissection is performed in a piecemeal fashion. Tissue sampling for frozen section is used liberally to determine the full extent of disease.

The dura of either the posterior or middle fossa is often involved in these tumors. Dural resection is again directed by extent of disease, and frozen section is used to control the margin.

Facial nerve management These patients generally present with facial nerve dysfunction, and these tumors generally involve the facial nerve in its mastoid or tympanic segments. Facial nerve sacrifice is required in this circumstance. Because labyrinthectomy is being performed, the facial nerve can be traced proximally and a tumor-free margin can be obtained. Occasionally, a low-grade tumor, such as an endolymphatic sac tumor or middle ear neuroendocrine tumor, can be adequately dissected away from the nerve and facial function preserved.

Reconstruction techniques These procedures generally do not produce large cutaneous defects, but reconstruction is complicated by the presence of a dural defect and the potential for cerebrospinal fluid (CSF) leak. A water-tight dural repair should be attempted using allograft duraplasty, but this can be impossible for some defects. Overlay temporalis fascia and abdominal fat graft, as is used in acoustic neuroma surgery, is another option for a small dural defect. Microvascular

free flaps can be used, especially in the setting of previous radiotherapy.

Total temporal bone resection

Although en bloc total temporal bone resection has been reported,[39–42] our team does not use this technique. Carotid resection is common to en bloc total temporal bone resection (TTBR), and this carries with it a high morbidity. Our philosophy has been to avoid carotid artery resection for malignant disease, because it produces significant morbidity and does not carry an improvement in overall disease-free survival.

TTBR extends the dissection as described earlier for STBR and includes removal of the internal auditory canal and petrous apex. Disease removal is piecemeal and directed by frozen-section pathology. Reconstruction considerations are similar to those described for STBR.

The role of parotidectomy and neck dissection

The parotid gland can be involved by either direct extension through the fissures of Santorini or by metastatic spread to intraparotid lymph nodes.[19,43] Morris and colleagues[19] described a series of 72 patients with temporal bone cancer, 36% of whom had direct tumor invasion into the parotid and 25% of whom had metastatic intraparotid lymph nodes. Gidley and colleagues[11] found that about 11% of their series of 157 patients with temporal bone cancer had salivary gland invasion. For these reasons, superficial parotidectomy, at a minimum, should be performed with LTBR.

Primary tumors of the ear canal, middle ear, and mastoid rarely (around 10%) present with cervical lymphadenopathy.[19,44–46] Level II and III are most commonly involved.[11] Although overall nodal involvement is low, neck dissection and parotidectomy permit accurate tumor staging.[12,47] Neck dissection also facilitates vessel exposure when microvascular free-flap reconstruction is needed.

Because the ear canal is more commonly involved by parotid primaries or periauricular skin cancers, parotidectomy and neck dissection are required to address the primary tumor.[48]

Complications of surgery

Temporal bone resection can be associated with high complication rates. All patients are counseled about the risks of surgery: hearing loss, tinnitus, dizziness, facial weakness or paralysis, loss of taste on the tongue, loss of the outer ear, CSF leak, and meningitis. Major complications, defined as requiring additional surgery or additional intensive medical therapy, have remained less than 10%. CSF leak and meningitis occur at rates consistent with transtemporal skull base surgery. Pulmonary embolism, myocardial infarction, and death have been reported after temporal bone resection.[11,49] For these reasons, patients must be in good general health to be able to tolerate such surgery.

A maximal conductive hearing loss is the expected side effect of LTBR. This hearing loss can be overcome with an osseointegrated bone conducting hearing aid. Single-sided deafness occurs from STBR and TTBR. Osseointegrated bone conducting hearing aids and a contralateral routing of sound hearing aid are options for hearing rehabilitation for these patients.

Facial paralysis is a disappointing, but often unavoidable, outcome of temporal bone surgery. Rates of facial nerve sacrifice might be nearly 50%.[11] Plastic reconstructive surgeons and oculoplastic surgeons are invaluable in helping to manage facial paralysis, to restore cosmesis, and to protect vision.

Contraindications to surgery

Surgery is not indicated for patients with unresectable disease, distant metastasis, or poor general health status. Tumors that encase the carotid or vertebral artery, that erode into the cervical spine, or that have significant brain invasion are not considered for surgical treatment. Although the use of carotid artery bypass has been reported for skull base cancers,[50] the long-term results for this technique are disappointing, yielding only a 20% 2-year survival and the attendant risks of postoperative stroke or death.[51] Our team has avoided such surgery and relied on palliative chemotherapy and radiotherapy for these patients.

Isolated and limited temporal lobe involvement can be resected.[41,52] Moffat and colleagues[53,54] have reported reasonable results after resection of temporal bone tumors with brain invasion. However, in our patient population, we have rarely found isolated brain invasion from temporal bone tumors that did not have concomitant carotid artery involvement or metastatic disease.[11]

TREATMENT: RADIOTHERAPY

Radiotherapy plays a significant role as adjuvant therapy for temporal bone cancers or as a treatment of patients who are not candidates for surgery. Primary radiotherapy was used to treat temporal bone cancers up to the 1970s[20,55,56]; however, this technique had a low overall cure rate. Only a few articles have reviewed the role of radiotherapy as single modality therapy. Kang and colleagues[57] concluded that radiotherapy alone was not inferior to combined surgery and

radiotherapy for disease-specific survival (DSS), but they found that local control was worse when radiotherapy alone was used.

Advances in skull base techniques vaulted surgery into its current role as primary therapy, using radiotherapy as a postoperative adjuvant.[19,58] The combination of these 2 modalities has improved overall survival for patients who have temporal bone cancer.[9,38]

Currently, radiotherapy is recommended for T2 and higher staged tumors.[12,54,59,60] Other indications for postoperative radiotherapy include recurrent tumors, positive margins, perineural spread, positive lymph nodes, or extracapsular spread.[38]

Intensity modulated radiotherapy (IMRT) allows the radiation oncologist the ability to adequately treat the tumor site and minimize dose to surrounding structures, especially the temporal lobe and brainstem. Dosages vary widely in the literature. Pfreundner and colleagues[61] recommended 54 to 60 Gy in patients with negative margins and a minimum of 66 Gy with positive margins. Prabhu and colleagues[62] gave doses between 60 and 66 Gy for patients with negative margins and doses between 68 and 72 Gy for patients with positive or close margins.

TREATMENT: CHEMOTHERAPY

Advancements in chemotherapeutic agents have ushered in a new era for head and neck tumors.[63–68] Data from these studies have been extrapolated from mucosal epithelial tumors of the head and neck to other sites. Only a few isolated studies have examined the role of chemotherapy for temporal bone cancers.[69–71]

Nakagawa and colleagues[69] described a series of 25 patients with primary SCCa of the ear canal and middle ear. Six patients (T2: 1 patient; T3: 3 patients; T4: 2 patients) received preoperative chemotherapy followed by surgery and radiotherapy. Five of these 6 patients achieved mean survival of 60 months. Chemotherapy and radiotherapy alone were used in 7 patients with T4 disease; 3 of these 7 patients had no evidence of disease at mean of 31.6 months.

In a pilot study, Shiga and colleagues[70] described a series of 14 patients with SCCa of the temporal bone, of whom 9 had stage IV disease and were treated with concomitant chemoradiotherapy. Their chemotherapy regimen included docetaxel, cisplatin, and 5-fluorouracil (TPF). Eight of 9 patients achieved complete response. These investigators concluded that the use of concomitant chemotherapy with TPF was safe and effective as a treatment of patients with cancer of the temporal bone.[70]

Intra-arterial chemotherapy has been proposed and tested in a few patients. Sugimoto and colleagues[71] published a small series of 5 patients with T3 and T4 SCCa of the temporal bone who were treated with radiotherapy and intra-arterial chemotherapy consisting of cisplatin and thiosulfate. Three patients obtained a complete response and had mean survival of 28 months.

OUTCOMES: RECURRENCE AND SURVIVAL

Historically, temporal bone tumors were associated with dismal results and a poor prognosis.[72,73] However, advancements in multiplanar imaging, skull base surgical techniques, IMRT, and chemotherapy have combined to improve overall survival. A cogent staging system, as proposed initially by Arriaga and colleagues[25] and later modified by Moody and colleagues,[5] has allowed comparison of results across time and institutions. This later iteration of the Pittsburgh staging has shown that survival is progressively worse with increasing T stage,[17] and this finding is supported by other studies.[12,45,74] The Pittsburgh tumor staging is an important, independent factor for prognosis, and a reliable predictor for outcomes for SCCa.[12]

Small tumors that are limited to the ear canal with minimal soft tissue involvement or bone erosion (ie, T1 and T2 tumors) can be completely excised with LTBR. Whereas T1 tumors can be treated with surgery alone, T2 tumors have improved outcomes when postoperative radiotherapy is added.[12,60] Those patients with early-stage disease have 80% to 100% 5-year survival rates.[7,75,76]

Larger tumors (T3 and T4) involve significantly more anatomic structures and present a significantly more difficult tumor to treat. These tumors can be conceptualized in 2 varieties: external ear canal tumors that erode past the eardrum into the middle ear, or tumors that arise primarily within the middle ear, inner ear, or mastoid. When tumors involve the middle ear, the LTBR is no longer an adequate or sufficient surgical treatment.[77] Multidisciplinary team management is required for these larger, advanced-stage tumors. These patients might require a combination of surgery, radiotherapy, and chemotherapy for treatment of their tumors. Treatment goals remain consistent despite the large size of the tumor: adequate disease resection to achieve negative margin and minimizing damage to surrounding normal structures. Despite adequate treatment, overall 5-year survival rates do not exceed 50% for this group of late-stage tumors (**Table 2**).

Facial nerve involvement, positive lymph nodes, extratemporal disease extension, and positive

Table 2
Comparison of recently published survival results for SCCa of the ear canal and temporal bone. Only studies with 20 or more patients are included

Authors, Year	Total Patients (n)	T Stage[a] (n)	Mean Fu (mo)	Overall 5-y Survival (%)	DSS (%)	Disease-Free Survival (%)
Yin et al,[45] 2006	95	NS	NS	66.8 (cohort) Stage I = 100 Stage II = 100 Stage III = 67.2 Stage IV = 29.5	NS	NS
Nakagawa et al,[69] 2006	25	T1 = 1 T2 = 3 T3 = 5 T4 = 16	39	T1 and T2 = 80 (estimated) T3 and T4 = 40 (estimated)	NS	NS
Kunst et al,[79] 2008	28	pT1 = 12 pT2 = 2 pT3 = 4 pT4 = 10	34	64 (cohort) T1 = 83 T4 = 25	NS	NS
Madsen et al,[74] 2008	47	T1 = 13 T2 = 7 T3 = 7 T4 = 19	48	31	42%	NS
Kang et al,[57] 2009	35	T1 = 10* T2 = 11 T3 = 14	34	NS	80 (3-y)	63 (3-y)
Prabhu et al,[62] 2009	30	T1 = 7* T2 = 5 T3 = 18	24	54	T1 and T2 = 70 T3 = 41	T1 and T2 = 73 T3 = 55
Gidley et al,[12] 2010	71	T1 = 20 T2 = 15 T3 = 5 T4 = 31	NS	38	NS	60
Chi et al,[7] 2011	72	T1 = 15 T2 = 3 T3 = 19 T4 = 35	NS	T1 = 100 T2 = 66.7 T3 = 21.1 T4 = 14.3	NS	NS

[a] Pittsburgh 2000 staging system[5] is used except where noted by an asterisk for Stell and McCormack staging system.[24]

surgical margins are all factors linked with poor overall survival.[6,7,11,19,45,74] Higgins and Moody[17] performed a systematic review of SCCa of the temporal bone to examine the effect of facial paralysis on survival outcomes. Their pooled data showed 5-year overall survival of 19.1% in patients with facial paralysis versus 59.4% in patients without facial paralysis, regardless of tumor stage.

Although lymph node metastases are uncommon, their presence is a significant risk factor for poor survival.[11] For patients with SCCa, Morris and colleagues[19] reported that 5-year DSS was 81% in node-negative patients and 19% in node-positive patients ($P<.0001$). A stark contrast in survival was also reported by Gidley and colleagues.[11]

Margin status is a strong predictor for recurrence, and rates of positive margins vary between 20% and 33%.[7,11,12,19,45,74] Morris and colleagues[19] describe 5-year DSS of 81.7% for patients with negative margins versus 50.0% for patients with positive margins ($P = .03$, long rank).

Other factors that are linked to high recurrence rates and poor survival include middle ear invasion,[74] need for mandibulectomy,[11,19] performance of craniotomy, facial nerve sacrifice, and parapharyngeal space or infratemporal fossa dissection.[78] Middle ear invasion is an important factor, because tumors that are confined to the ear canal can be resected en bloc with LTBR. Survival rates decrease to about 20% when the middle ear is involved with tumor, compared with 60% or higher when the middle ear is not involved.[20,74]

Intracranial disease extension can be successfully treated.[49,54] Dural involvement was seen in about 5% of patients from a series of 157 patients with temporal bone cancer reported by Gidley and colleagues.[11] Dean and colleagues[38] found intracranial disease in 16 of 65 patients, and they found that it did not have an effect on disease-free survival. The local control rates were similar with or without intracranial extension (76.9% vs 71.7%, respectively).[38]

Recurrences tend to occur within the first 2 years after completion of therapy.[11,19] A large study of 157 patients with temporal bone tumors showed a mean time to recurrence of 13 months.[11] In this study, recurrences were 12.7% local, 6.4% regional, and 13.4% distant. The most common sites for distant spread are lung, brain, and dermal metastases.[11] Morris and colleagues[19] showed recurrence rates of 20.5% for local disease, 5.5% for regional disease, and 22.9% for distant disease. Recurrence rates tended to be higher with salivary gland origin tumors.

Because temporal bone tumors are rare, many investigators have lumped several different tumor histologies into analysis.[11,19] This lumping can make analysis difficult because tumor biology and behavior vary widely among these histologies. A clear distinction in tumor behavior exists between SCCa and ACCa. Although SCCa is the most common tumor type, it has a lower overall 5-year survival rate than is reported for ACCa of the ear canal.[12,13] Although mean time to recurrence with SCCa is around 2 years, the mean time to recurrence for ACCa is reported to be nearly 8 years.[12,13]

SUMMARY

Primary temporal bone tumors are rare, and the temporal bone is more likely to be involved secondarily by tumors from the parotid gland or periauricular skin. Ear canal cancers are rare and do not have specific symptoms distinguishing them from benign ear canal conditions. Suspicious lesions of the ear canal should be biopsied for proper diagnosis. The most common tumor type is squamous cell cancer; however, a long list of tumor types have been described involving the temporal bone.

Surgical resection to achieve negative margins is the mainstay of treatment. Small tumors (T1 and T2) can often be treated with LTBR. Parotidectomy and neck dissection are added for disease extension and proper staging. Higher staged tumors, T3 and T4, generally require STBR or TTBR along with possible craniotomy, mandibulectomy, resection of the zygoma, and dissection of the infratemporal fossa.

Small defects can be adequately reconstructed with a temporalis muscle flap. Microvascular free flaps are used for large defects and for patients with a history of previous radiotherapy. Adjuvant postoperative radiotherapy has shown improved survival for patients with tumors staged T2 or higher. Chemotherapy has an emerging role for advanced-stage disease. Evaluation and management of patients with temporal bone tumors by a multidisciplinary team are critical to optimize outcomes in this group of patients.

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