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Osteoradionecrosis of the Temporal Bone: A Case Series

Jeffrey D Sharon, MDⁱ, Shariq S. Khwaja, MD, PhDⁱⁱ, Andrew Drescher, MDⁱ, Hiram Gay, MDⁱⁱ, and Richard A Chole, MD/PhDⁱ

ⁱDepartment of Otolaryngology, Washington University in St. Louis, MO

ⁱⁱDepartment of Radiation Oncology, Washington University in St. Louis, MO

Abstract

Objective—To study osteoradionecrosis (ORN) of the temporal bone

Study Design—Retrospective case review

Setting—Academic medical center

Patients—Patients were included who had previously undergone radiation to the head and neck and then developed exposed necrotic bone within the ear canal that persisted at least three months

Intervention(s)—Patients were treated with a variety of modalities, including conservative therapy with antibiotic ear drops and in-office debridements, hyperbaric oxygen therapy and surgery.

Main Outcome Measure(s)—To describe the presentation and management of patients with temporal bone osteoradionecrosis.

Results—33 patients with temporal bone osteoradionecrosis were included. The most common site of primary tumor was the parotid gland (n=11), followed by the nasopharynx (n=7). The time to development of ORN varied between 1 and 22 years, with mean 7.9 years. The mean radiation dose was 62.6 Gy to the primary tumor, 53.1 Gy to the affected temporal bone, and 65.2 Gy to the affected tympanic bone. The most common symptoms of ORN were otorrhea (n=15), hearing loss (n=13), and otalgia (n=12). 15 patients had bacterial superinfection, most commonly *S. aureus* (n=9). Conservative therapy was successful at managing symptoms but not in eradicating exposed bone in most patients. Surgery was used for recalcitrant pain, infection, cholesteatoma, cranial neuropathies, and intracranial complications.

Conclusions—Osteoradionecrosis is a rare complication of radiation to the temporal bone. Management should be aimed at relief of symptoms, eradication of superinfection, and treatment of other commonly present radiation effects like cholesteatoma and hearing loss.

Introduction

Radiotherapy (RT) has been widely used as primary or adjunctive therapy for a variety of head and neck malignancies. When the radiation field includes the temporal bone, there can be several acute and long-term adverse effects to the temporal bone and the structures contained within. Some have observed that the external ear canal (EAC) can be affected with ulceration, epithelial thickening with stenosis, fibrosis, atrophy of cerumen glands and cholesteatoma.¹ Radiation to the middle ear has been shown to cause mucosal hypertrophy, loss of ciliary function, ossicular necrosis and Eustachian tube obstruction; which in turn causes serous otitis media, tympanic membrane atelectasis, cholesteatoma and chronic inflammation.² Radiation damage to the inner ear was shown to cause sensorineural hearing loss and vestibular impairment, with histologic evidence of damage to the stria vascularis, spiral ligament, basilar membrane and destruction of cells within the Organ of Corti.³⁻⁵ Very rarely the facial nerve can be damaged, producing paralysis or paresis.

Radiation to the skull base can also result in bony necrosis of the temporal bone, termed osteoradionecrosis (ORN). There has been no consensus if higher doses of radiation cause more necrosis, why there is variability in the time to necrosis, and what is the optimal treatment.^{6,7} In this study we present a series of 33 patients with ORN of the temporal bone. We do so both to review the diagnosis and treatment of this rare entity, and to suggest an approach to the management of these patients based upon available evidence.

Methods and Materials

After obtaining approval from the institutional review board at Washington University in St. Louis, candidates for inclusion were identified in two ways. They were first identified by direct identification of patients by both the otologists and head and neck surgeons, one of whom kept a log of his patients with ORN. Secondly, candidates were identified by searching through the otology patients with the International Classification of Diseases (ICD)- 9 codes for chronic osteomyelitis of the temporal bone (730.1), personal history of malignant neoplasm (V10), effect of radiation therapy (990), and disorder of the external ear (380). The inclusion criteria were a history of radiation therapy and exposed necrotic bone within the temporal bone for at least a period of three months. There were no exclusion criteria. The patient's medical records were examined for the details of ORN and treatment, and the treating physician was contacted to make any necessary clarifications. When available, details of radiation therapy were reviewed and tabulated. Audiograms, when available, were examined as well to determine hearing levels in patients with ORN. Data collected included word recognition score (WRS), pure tone average (PTA), which was calculated as the mean air conduction pure tone for 500, 1,000, 2,000, and 3,000 Hertz, and speech recognition threshold (SRT). In addition, we examined data from radiology, including CT and MRI, pathology, microbiology and operative reports.

Radiation records were available for 26 patients in terms of total dose prescribed. For 14 patients, detailed radiation plans were available to determine the dose received by the total temporal bone as well as the tympanic bone. For these patients, segmentation with contours of the temporal and tympanic bones was added to their radiation plans and detailed dose

volume histograms were generated in order to ascertain the maximum and mean doses to these structures. For each patient with a detailed radiation plan available, dose to the affected and unaffected temporal and tympanic bone was acquired.

Statistical comparisons were performed using the Wilcoxon rank sum test to compare mean values for our data sets and Spearman's rank correlation coefficient to assess for correlation between data points. The significance level was set at 0.05, and two-tailed tests were used. Analysis was performed using GraphPad Prism version 5.00 for Mac OS X, GraphPad Software, San Diego California USA, www.graphpad.com.

Results

We identified 33 patients with ORN of the temporal bone, who are summarized in Table 1. The most common site of radiation was the parotid gland (11), followed by nasopharynx (7), and peri-auricular skin (6). The time between RT and the development of ORN varied widely, between 1 and 22 years, with average of 7.9 years (Figure 1). Four patients were radiated in the 1980s, 14 in the 1990s and 15 in the 2000s. The mean radiation dose was 62.6 Gy to the primary site (median dose 65.8 Gy, minimum 40 Gy, maximum 70.3 Gy). Fourteen patients had enough data in our system to calculate the dose to the affected and unaffected temporal bone. Analysis showed a mean of 53.1 Gy to the affected temporal bone (SD 10 Gy) (median dose 57.4 Gy, minimum 41.8 Gy, maximum 55.5 Gy), and 24.3 Gy to the unaffected temporal bone (SD 17.8 Gy) (median dose 16.35 Gy, minimum 2.2 Gy, maximum 54.6 Gy); (p-value 0.0015). In those patients the RT dose to the tympanic bone was also calculated, revealing a mean dose 65.2 Gy (SD= 6.8 Gy) (median dose 65.4 Gy, minimum 53.0 Gy, maximum 75.7 Gy), and 32.2 Gy to the contralateral tympanic bone (SD=22.8 Gy) (median dose 22.75 Gy, minimum 3.4 Gy, maximum 70.4 Gy); (p-value 0.005). No correlation was seen between radiation dose to the primary site and time to development of ORN (Spearman $r=-0.202$, $p=0.33$). However, there was a moderate correlation seen between dose to the temporal bone and time to development of ORN (Spearman $r=-0.552$, $p=0.0405$). There was no correlation seen between dose to the tympanic bone and time to development of ORN (Spearman $r=0.154$, $p=0.599$). There was no correlation seen between age at diagnosis and the interval between radiation and diagnosis of ORN (Spearman $r=0.152$, $p=0.40$). In terms of possible predisposing medical comorbidities, nine of our patients had vascular disease, defined as diabetes, stroke, coronary artery disease or peripheral vascular disease. The most common comorbidity was hypertension (14), and eight patients had hypothyroidism, likely resulting from radiation to the head and neck. There were two cases of bilateral ORN, one in a patient with nasopharyngeal carcinoma, and one with supraglottic squamous cell carcinoma (SCC).

The most common symptoms that led to the diagnosis of ORN were otorrhea (15), hearing loss (13), otalgia (12) and bloody otorrhea (3). There were a variety of other findings consistent with radiation damage in our series, including debris in EAC (8), tympanic membrane perforation (5), cholesteatoma (5), purulence (4), otitis externa (4), granulation tissue (4), canal stenosis (2) and middle ear effusion (1).

Physical findings in this group of patients varied but all had exposed, necrotic bone in the EAC (Figure 2). The amount of exposed bone varied, from a small exposed patch to a larger area of necrosis, with possible fistulae to the mastoid or temporomandibular joint (TMJ) (Figure 3). In one patient fluid bubbles were seen emanating from the temporomandibular joint fistula in the EAC (Figure 4). The necrotic bone was often infected, with drainage that was sometimes bloody, sometimes purulent, and oftentimes malodorous. Granulation tissue was commonly seen adjacent to the necrotic bone.

Bacterial infection was documented in 15 of the 33 patients, and five had two positive cultures. Nine cultures grew *S. aureus*, five grew mixed organisms, and one culture each grew *Corynebacterium jeikeium*, *Actinomyces* species, *Pseudomonas*, *Streptococcus viridians*, *Klebsilla oxytoca*, and *Enterococcus faecalis*. One patient grew rare *Candida albicans*. High-resolution temporal bone CT scanning was used to delineate the extent of disease. The most common findings on CT included bone erosion, bone sclerosis, soft tissue thickening and mastoid opacification (Figure 5). Magnetic resonance imaging (MRI) was less specific than computed tomography (CT) in showing the bony changes associated with ORN, but MRI did show a variety of other radiation effects like mastoid effusion or soft tissue within the middle ear and mastoid (Figure 6). Audiometric data was analyzed to characterize the hearing levels in patients with ORN (Figure 7). Pre-radiation audiograms were not available, and the time from diagnosis of ORN until audiogram varied from 0 to 10 years, with three years on average. Because of a lack of baseline data, the hearing in the ear that developed ORN was compared to the contralateral ear. There were 25 patients in whom audiometric data was available, and two were excluded because of bilateral ORN. The mean WRS in the affected ear was 62%, and the mean WRS in the contralateral ear was 79% ($p=0.0068$). The PTA in the affected ear was 58 dB versus 36 dB in the contralateral ear ($p=0.0011$). The SRT in the affected ear was 53 dB versus 32 dB in the contralateral ear ($p=0.0015$). There were 27 ears that developed ORN and had audiograms, of those 11 had sensorineural hearing loss, and 16 had mixed hearing loss.

The subset of seven patients with nasopharyngeal carcinoma was examined because theoretically they received similar doses of RT to both temporal bones. One patient developed bilateral ORN, and all except for one had clinical evidence of radiation damage to the contralateral ear, including severe to profound hearing loss ($n=3$, with one requiring cochlear implant), and chronic suppurative otitis media ($n=2$). In the patients in whom we had data who only developed ORN in one ear ($n=4$), the mean dose to the affected ear was 50 Gy, versus 46 Gy to the contralateral ear, and the difference was not statistically significant.

The treatment of ORN in these patients varied depending on the severity of disease. Most patients initially received conservative therapy, including topical antibiotics, office debridement of dead bone, gentian violet applications and analgesics for pain. Of the twenty patients who were originally treated with local care, two had complete resolution of signs and symptoms, eleven still had exposed bone at the last follow up visit, but symptoms were well controlled in eight of those eleven, two had insufficient follow up data, and five progressed to require surgery. Eighteen of 33 patients underwent surgery for either cholesteatoma or recalcitrant symptoms or pain and drainage. Long-term follow up data was

available for fourteen of those patients, which showed that seven patients were successfully treated with one procedure, five patients did not resolve with one surgery but either declined further surgery or had manageable symptoms despite exposed bone, and two patients eventually resolved with multiple surgeries. The procedures performed included canalplasty, mastoidectomy, lateral temporal bone resection, closure of EAC and temporalis rotational flap reconstruction. Pathology showed hyperkeratosis, granulation tissue, acute and chronic inflammation, abscesses, necrotic bone and fibrosis (Figure 8). Six patients received hyperbaric oxygen (HBO) therapy, with one of those treated prophylactically postoperatively to assist healing. Of the other five, all later progressed to require surgical therapy, which one declined to undergo.

Discussion

We have presented here the largest series of ORN of the temporal bone to date. Unlike prior studies, we did not include patients with idiopathic temporal bone necrosis, which is a rare condition characterized by necrotic bone without a clear cause, but believed to be caused by trauma and vascular disease.⁶ Our data correlate well with other large studies, in terms of patient presentation, time to development of ORN and rates of surgical therapy.^{6,7}

Temporal bone ORN refers to the necrosis of the temporal bone as a sequela of radiation therapy. It was originally described by Ewing in 1926 as a bony osteitis.⁸ Schuknecht described the histopathological findings in the temporal bone in 1966 by describing an “aseptic bone necrosis with compensatory reparative fibrosis.”⁹ The pathophysiology of ORN is unclear, but is believed to be due to radiation damage to blood vessels, causing osteocyte loss, fibrosis, hypocellularity and fatty degeneration of bone.¹⁰ However, there is also evidence that bacterial and fungal infections are common within radiation damaged bone, and bacterial biofilms have been shown to be present as well.¹¹⁻¹³ There is agreement that the tympanic bone is susceptible to the effects of radiation because of its superficial position, poor vascularity, thin epithelium and resident flora.^{7,14} In 2000, Ondrey analyzed the radiation dose to the inner ear during radiation therapy in 15 patients using treatment planning software.¹⁵ He found that in three patients radiated for nasopharyngeal cancers, the cochlea received 86-102% of the total radiation dose, with the Eustachian tube receiving full radiation dose. In patients radiated for oral cavity and oropharyngeal primaries, the Eustachian tube received greater than 60% percent of the total radiation dose in the majority of patients.

The diagnosis of ORN is based on finding necrotic bone, usually visible within the external auditory canal, in patients with a prior history of radiation. Symptoms can include pain, purulent or bloody otorrhea, and trismus with TMJ involvement. While the time between RT and the clinical diagnosis of ORN can vary widely, from six months to 40 years in one series,⁶ experimentally ORN of the temporal bone was produced in 12 weeks by exposing rats to proton beam irradiation.¹⁶ Ramsden presented a review of 29 cases of temporal bone ORN, and he divided them into localized ORN, with involvement of only the tympanic bone, and diffuse ORN, involving the rest of the temporal bone as well.⁷ In some cases ORN can be life threatening from intracranial complications, including with brain abscesses, carotid artery pseudoaneurysm, meningitis, CSF leak and sigmoid sinus thrombosis.¹⁷ In a

study of 221 patients treated for parotid tumors, Leonetti found the incidence of temporal bone ORN after parotidectomy and RT to be 1.9%. After combined parotidectomy, mastoidectomy and RT it was 12.5%, and after parotidectomy, subtotal petrosectomy, oversew of the meatus and flap obliteration of the mastoid cavity and radiation it was 0%.¹⁸ Vudiniabola found the incidence of ORN of any of the facial bones to be 1.2% after radiation for head and neck cancer.¹⁹

Reliable evidence on which to base the optimal treatment of ORN of the temporal bone is not available, but widespread agreement that conservative therapy is indicated for localized disease or minor symptoms, whereas patients with widespread disease or severe pain and/or drainage are more likely to require surgical therapy. A suggested treatment algorithm for ORN is presented (Figure 9). We suggest a trial of conservative therapy in all patients, with local debridement and topical antimicrobials. Gentian violet has a good rationale for application, as it may disrupt biofilms which have been shown to be present in ORN.^{13,20} One study suggested that surgery within the radiated temporal bone can be hazardous, with higher than expected rates of facial nerve dehiscence, oval window and lateral canal fistulae, dural exposure, CSF leak and lateral sinus or superior petrosal exposure.²¹ That study also suggested that if mastoidectomy is to be performed, removal of the posterior canal wall has a higher success rate than intact canal procedures, presumably because of a poor blood supply to the EAC that is further decreased by surgery. Imaging can be helpful preoperatively to define the extent of ORN to aid in surgical planning. In this study, a single surgery was successful in completely eliminating the ORN about half of the time, and conservative therapy was successful in controlling symptoms but not in eradicating exposed bone most of the time. However, it should be noted that the goal of treatment should be aimed at symptom control rather than complete removal of necrotic bone. We had many patients in whom either conservative therapy or surgery was successful in relieving pain or super-infection despite persistent ORN. We propose that the potential benefits in terms of symptoms relief must carefully weighed against the risks of surgery within the radiated temporal bone. It should also be mentioned that many of our patients were of advanced age at presentation, which did skew our treatments towards conservative therapy rather than surgical therapy.

The idea of reconstructing the surgical defect created by resection of necrotic bone with vascularized tissue is intuitively appealing, and local rotational and free flaps have been used for this purpose.²²⁻²⁴ In our series, two patients underwent temporalis rotational flap to help close defects in the irradiated temporal bone, both after lateral temporal bone resections. One of those patients who had a prior resection of the temporal bone and mandibular condyle had extensive ORN of not only the temporal bone but the ipsilateral skull base and temporal lobe. In this patient, a temporalis rotational flap was used to cover this large defect. However, she continued to have a draining fistula from the necrotic bone of her skull base. The second patient had a temporalis flap used to fill in a lateral temporal bone resection defect and underwent post operative HBO with good healing. In another patient an antero-lateral thigh free flap was used to cover a large temporal bone defect with good result.

A TMJ fistula is a rare condition, usually resulting from odontogenic infections, suppurative arthritis, otitis externa, trauma, or surgical complication.²⁵ Anatomically the TMJ and EAC are separated by the thin plate of the tympanic bone, with small foramina of Huschke in the anterior-inferior aspect of the plate. Sinn describes successful closure of an aural-TMJ fistula with a temporalis flap in a patient with an iatrogenic fistula.²⁵ Schwartz describes successful closure of an EAC-TMJ fistula in a previously irradiated temporal bone with fascia lata.²⁶ In this series three patients had fistulae from the EAC to the TMJ. These patients presented with pain around the TMJ, and hearing a crackling sound each time the jaw is opened. They present an interesting problem, because in addition to resection of necrotic bone, reconstruction is required to recreate the posterior wall of the glenoid fossa and anatomically separate the EAC and TMJ. In our series, one patient underwent hyperbaric oxygen therapy with 48 treatments and a skin graft; those procedures failed to close the fistula. A lateral temporal bone resection with condylectomy was considered but not pursued due to the patient's medical condition and proximity to a previous craniotomy. The second patient was offered a lateral temporal bone resection, plugging of the Eustachian tube, closure of the EAC, but declined to have surgery. The third patient was managed conservatively, with antibiotic drops and gentian violet, and this helped alleviate some of the pain with jaw movement.

Cochlear implantation within the irradiated temporal bone is controversial. On one hand, irradiated patients oftentimes have profound hearing loss, which can be conductive or sensorineural due to intracochlear fibrosis and hair cell loss.²⁷ On the other hand, placing hardware within a necrotic field which may harbor subclinical infection might predispose to further infectious complications. A study by Low evaluated patients radiated for nasopharyngeal carcinoma, and auditory brainstem response audiometry demonstrated an absence of retrocochlear effects.²⁸ This paved the way for Adunka to successfully perform a cochlear implant in a patient with ORN.²⁷ That patient had previously undergone labyrinth-sparing temporal bone resection with rectus abdominus free flap reconstruction and post-operative hyperbaric oxygen therapy. In our series, two patients underwent cochlear implantation in the setting of ORN. In the first patient, the procedure was performed in two stages, with a mastoidectomy and EAC obliteration first to eradicate chronic otitis externa, and then cochlear implantation in the second stage. He did well, with good function of the implant. The second patient underwent cochlear implantation along with mastoid obliteration with a fat graft in a single procedure, however only 11 electrodes could be inserted into the cochlea due to fibrosis. It has been suggested that magnetic resonance imaging (MRI) may be used preoperatively to ensure cochlear patency. Therefore, cochlear implantation after temporal bone ORN is feasible, but care must be taken to eradicate necrotic bone and infection beforehand, and the risk of cochlear fibrosis and incomplete electrode insertion should be considered.

Hyperbaric oxygen therapy has been used as therapy in irradiated tissue to help with oxygenation, which is thought to promote healing and eradication of chronic infection. Marx showed that irradiated bone, after exposure to HBO, showed increased fibroblastic stroma and angiogenesis.²⁹ Vudiniabola reported the successful use of HBO in three cases of ORN of the temporal bone, and Rudge reported its successful use in one case.^{19,30} In our series six patients underwent HBO, it did not result in resolution of the necrotic bone in any patients,

but possibly aided with postoperative healing in one patient. Overall it is difficult to assess the efficacy of HBO in treatment of temporal bone ORN, and in fact, a recent review questioned its efficacy in mandibular ORN.³¹ At this stage, HBO treatment of ORN should be considered rational but it lacks sufficient evidence to support its widespread use.

In current practice, there is no absolute constraint that has been put on the radiation dose to the temporal bone. Certainly, in day-to-day segmentation, the temporal bone is rarely included as a normal tissue contour. As such, there are no real contouring guidelines for the temporal bone. Here, we presented one of the first efforts to identify the dose to the temporal bone and its association with ORN. Our data showed a statistically significant higher dose received by the affected temporal bone in head and neck cancer patients receiving radiotherapy as part of their cancer care. Furthermore, our finding of a moderate correlation between dose to the temporal bone and time to development of ORN does suggest there is some causality between higher doses and the development of ORN. Further study is required to compare the RT doses to the temporal bone in these patients versus patient radiated for similar malignancies who did not develop ORN to see if they are receiving higher doses to the temporal bone. These results should be viewed with caution, however, as these results do not establish tolerance dose level for the temporal bone. While practicing radiation oncologists should work to establish proper tumor coverage in their radiation plans, we propose that dose to the temporal bone does matter and can result in late effects, including ORN.

There are several important limitations of this study. Firstly, as a retrospective study, there is danger of recall bias. Secondly, although these patients had significant decrements in hearing, the audiometric data is hard to interpret because there were no pre-treatment audiograms to compare with, and the timing of the audiograms was variable, and there were oftentimes several contributing factors to the hearing loss. Without a control group it is hard to interpret some of the information we present, like the doses to the temporal bone. Additionally, since our treatment algorithm was developed after looking through our data, we only argue that it is a logical approach to treatment based on our experience, but we do not have prospective data that proves its efficacy. Furthermore, since there is no consensus on what entails successful treatment, be it complete eradication of exposed bone, complete resolution of symptoms, or reduction of symptoms to acceptable levels, it is hard to draw conclusions on which of our treatments were successful.

In conclusion, ORN of the temporal bone is a delayed complication of radiation for the treatment of malignancies of the head and neck. The onset of ORN after RT is highly variable. Most patients with ORN should be managed conservatively with local debridement and topical antimicrobials. Progressive and extensive ORN can often be managed by ablative surgery and local reconstruction. We offer a paradigm for the management of these challenging cases.

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Figure 1.

Lag Years (years from radiation until diagnosis of ORN) plotted against XRT dose. Higher doses of XRT are not associated with quicker development of ORN.

Figure 2.

Arrows point to necrotic brown bone inferiorly in the EAC. Changes to the epithelium of the EAC can be seen including thinning and increased superficial vascularity.

Figure 3. More extensive exposed bone within the EAC. Squamous debris and thickening of the TM make it difficult to see any middle ear landmarks. The arrow points to a fistula to the TMJ.

Figure 4. Bubbles are seen in the EAC with jaw opening. CT shows a bony defect of the anterior wall of the EAC leading to the glenoid fossa.

Figure 5.
Coronal CT scan from the same patient as Figure 1, showing bone loss inferiorly in the EAC.

Figure 6.
Axial T2 MRI showing fluid throughout the right middle ear and mastoid cavity in association with ORN.

Figure 7. Audiometric data comparing pure tone average (PTA), speech recognition threshold (SRT), and word recognition score (WRS) in the ear with ORN versus the contralateral ear. The height of the bar is the mean, and the error bars show the standard deviation.

Figure 8.
H&E stain, 600x magnification, showing necrotic bone and inflammation with neutrophils.
Note the absence of osteocytes in osteocyte lacunae.

Figure 9.
Proposed treatment algorithm for osteoradionecrosis

Table 1

Summary of patients with temporal bone osteoradionecrosis

Age	Sex	Tumor	RT dose (cGy)	Dose affected temporal bone (cGy)	Lag Years	Treatment	Micro	Audiogram
57	F	Left Parotid	6000		18	gtts, abx, HBO, partial lateral temporal bone resection, 3 years later with exposed bone that responded to conservative therapy, NED	S. aureus	10 years, WRS 100% and PTA 18 dB
61	F	Right Parotid Basal Cell Adenocarcinoma	5760		6	gtts/wick, office debridement/gentian violet, still with exposed bone	S. aureus	9 years, WRS 92% and PTA 30 dB
59	M	NPC	7030	5940	8	Failed closure of TM perforation then radical mastoidectomy for cholesteatoma then mastoidectomy revision with mastoid and EAC obliteration, followed by cochlear implant on left, then cochlear implant on right, NED	S. aureus	8 years, WRS 0 and PTA NR
47	F	NPC	N/A		9	conservative care, BAHA, then CWD mastoidectomy for cholesteatoma, NED	No cultures	2 years, WRS 0%, PTA NR
83	M	Parotid Skin SCC	N/A		2	local care with debridement-had cholesteatoma that was not operated on due to medical condition	No growth	5 years, WRS 0 and PTA 95 dB
86	F	Right Glomus Jugulare	6000		9	tympanomastoidectomy for cholesteatoma then local care with office debridement, lost to f/u	No cultures	N/A
28	F	NPC	7000	4800	4	HBO, and 2 mastoidectomies, then local care.	No cultures	2 years, WRS 55 PTA 80 dB
43	F	Right Parotid Pleomorphic	4500		8	partial mastoidectomy and tympanic bone resection, meatoplasty, then local care then EAC soft tissue/ tympanic bone excision, NED	Corynebacterium jeikeium, Actinomyces species	0 years, WRS 100, PTA 10 dB
66	M	Left EAC SCC	N/A		22	local care, then mastoidectomy with canal wall reconstruction, NED	No Cultures	0 years, WRS 82%, PTA 40 dB
48	F	Metastatic breast	N/A		0	local care, HBO, unsuccessful skin graft to EAC, surgery offered for TMJ fistula but declined	No Cultures	No Audio
68	M	NPC	7000	4460	5	local care, then cochlear implant, second surgery for	Mixed microorganisms	2 years, 0% WRS and PTA NR

Age	Sex	Tumor	RT dose (cGy)	Dose affected temporal bone (cGy)	Lag Years	Treatment	Micro	Audiogram
						repositioning implant, NED		
68	F	Left Parotid Mucoepidermoid	6000		16	surgery offered with temporal bone resection, closure of EAC for TMJ fistula, declined by patient	No cultures	0 years, WRS 70%, PTA 50 dB
69	F	Left Parotid acinic cell carcinoma	5600		20	temporal bone resection, closure of EAC, temporalis rotational flap, post-op HBO, NED	S. viridans	0 years, WRS 4 and PTA 100 dB
65	F	Left Parotid SCC	6600	6290	1	lateral temporal bone resection with resection of glenoid fossa and reconstruction with temporalis muscle and fascia rotation flap, still with draining fistula	S. aureus, Pseudomonas	5 years, WRS 44%, PTA 60 dB
55	M	NPC	6995	5090	1	mastoidectomy with petrosectomy, NED	Mixed microorganisms	0 years, WRS 42%, PTA 85 dB
70	M	Parotid Skin SCC	6000	6150	0	local care with gtts, antibiotics, NED	No cultures	0 years, WRS 72%, PTA 50 dB
81	F	Left EAC SCC	5600		2	Local care, then meatoplasty and debridement of necrotic bone, still with exposed bone	Mixed microorganisms	No Audio
85	M	metastatic SCC left neck	6660	4180	13	local care, had TMJ fistula but surgery not offered due to medical condition	No cultures	0 years, WRS 78%, PTA 65 dB
79	F	NHL	4000		12	local care, still with exposed bone	No cultures	1 year, WRS 78%, PTA 45 dB
54	M	left conchal SCC	6600	6550	1	local care, mastoidectomy, NED	C. Albicans, mixed microorganisms	0 years, WRS 78%, PTA 85 dB
43	M	right parotid acinic cell carcinoma	6000		7	local care, exposed bone at last visit	No cultures	No Audio
62	F	left parotid acinic cell carcinoma	6600	6400	4	canalplasty, afterwards exposed bone treated with local care	S. aureus	0 years, WRS 90 %, PTA 40 dB
54	M	NPC	7000			local care, still with exposed bone	No cultures	0 years, 72% WRS, 45 db.
61	M	Right Parotid mucoepidermoid	6000	5830	7	local care, still with exposed bone	No cultures	2 years, 100% WRS, PTA 60 dB
62	F	right tongue/palate SCC	N/A		7	local care, no f/u data	No cultures	No Audio
59	M	right parotid skin SCC	6560		5	HBO, mastoidectomy, then more HBO, still with exposed bone	S. aureus	9 years, WRS 56%, PTA 55 dB

Age	Sex	Tumor	RT dose (cGy)	Dose affected temporal bone (cGy)	Lag Years	Treatment	Micro	Audiogram
48	F	NPC	6996	4620	2	local care, NED	S. aureus, mixed microorganisms	1 year, WRS 96%, PTA 20 dB
54	M	right SMG carcinoma expleomorphic adenoma	6650	5260	8	local care, exposed bone at last visit	S. aureus, Klebsiella oxytoca	No Audio
63	F	Parotid adenocarcinoma	N/A		11	Mastoidectomy, no f/u data	No growth	No Audio
58	M	Supraglottic SCC	6600	3040	9	local drops, exposed bone at last visit	No cultures	2 years, 0% WRS, PTA NR
71	M	parotid	4600		15	local care, exposed bone at last visit	Enterococcus faecalis	10 years, 96% WRS, PTA 20 dB
58	F	Right tonsil SCC	7000		3	local care	No culture	No Audio
65	M	Right ear BCC	6000	5660	1	Temporal bone resection with free flap reconstruction, NED	S. aureus	3 years, 0% WRS, PTA 100 dB

Legend: Age refers to age at diagnosis of ORN. Local care refers to antibiotic ear drops, topical applications like boric acid and gentian violet, and in-office debridement of necrotic bone. NED indicates no evidence of disease- i.e. no further exposed bone or symptoms referable to ORN. For the audiology data, presented are years after the diagnosis of ORN the audiogram was completed, and the word recognition score (WRS) and pure-tone average (PTA) in the affected ear.

Abbreviations: gtt = drops, abx = antibiotics, EAC = external auditory canal, SCC =squamous cell carcinoma, BCC = basal cell carcinoma, NHL = non-Hodgkins lymphoma, HBO = hyperbaric oxygen, TMJ = temporal-mandibular joint, NR= no response