Malignant External Otitis: CT Evaluation

Malignant external otitis is an aggressive infection caused by *Pseudomonas aeruginosa* that most often occurs in elderly diabetics. Malignant external otitis often spreads inferiorly from the external canal to involve the subtemporal area and progresses medially towards the petrous apex leading to multiple cranial nerve palsies. The computed tomographic (CT) findings in malignant external otitis include obliteration of the normal fat planes in the subtemporal area as well as patchy destruction of the bony cortex of the mastoid. The point of exit of the various cranial nerves can be identified on CT scans, and the extent of the inflammatory mass correlates well with the clinical findings. Four cases of malignant external otitis are presented. In each case CT provided a good demonstration of involvement of the soft tissues at the base of the skull.

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MALIGNANT external otitis, perhaps more appropriately called necrotizing or invasive external otitis, is an aggressive *Pseudomonas* infection that occurs almost solely in elderly diabetics (1). Beginning as an area of granulation on the floor of the external auditory canal, the infection can cause chondritis and osteomyelitis that extends posteriorly into the mastoid, anteriorly into the region of the temporomandibular joint, or medially to the apex of the petrous bone. More commonly, however, the infection extends inferiorly into the soft tissues in the subtemporal area apparently following the fissures of Santorini, which are small clefts in the cartilaginous portion of the external auditory canal.

Involvement of the cranial nerves, especially motor VII, is an ominous sign associated with high mortality. Although some reports have described involvement of the facial nerve at various points within the temporal bone (2-4), most descriptions have indicated that facial paralysis is a result of an infection in the subtemporal space affecting the nerve as it leaves the stylomastoid foramen (1, 5, 6-8). Computed tomography (CT) demonstrates well the subtemporal component of malignant external otitis (MEO). The disease obliterates the normal fat planes in the subtemporal space, including the small fat collections normally seen just below the stylomastoid foramen close to the extratemporal facial nerve.

Four cases that illustrate the usefulness of CT in staging MEO, and especially its usefulness in evaluation of the subtemporal component of the disease, are presented. In this paper we concentrate on the CT findings in the subtemporal area rather than describe all radiographic signs of the disease.

MATERIALS AND METHODS

All tomography was performed with trispiral motion. CT scans were obtained with a GE 8800 with slice thickness ranging from 1.5 to 5 mm. Contrast material was administered intravenously.

CASE STUDIES

CASE 1: An 84-year-old man without a history of diabetes presented with persistent pain despite three to four weeks of local therapy for external otitis. Left facial nerve paralysis began two days prior to admission. *Pseudomonas* was cultured from the external canal. Physical examination showed granulation tissue at the junction of the cartilaginous and bony portions of the external canal. There was peripheral seventh nerve palsy with the other cranial nerves functioning normally. Antibiotic therapy was begun with intravenous ticarcillin (Ticar, Beecham) and tobramycin (Bencin, Lilly); gentamicin (Garamycin, Schering) was later used when bacterial culture and sensitivity test results became available.

CT showed soft-tissue density in the external canal and a clear middle ear. The normal fascial planes around the mastoid tip, including the area between the styloid process and mastoid tip in the anatomic location of the facial nerve (Fig. 1), were obliterated. Many small areas of the external cortex of the mastoid were eroded with normal intervening bone. There was partial specification of the mastoid air cells (Fig. 2).

A modified radical mastoidectomy and decompression of the vertical
1. Malignant external otitis involving the right side. Increased soft-tissue density surrounds mastoid tip (m) (a). Note obliteration of fat planes, especially between mastoid and styloid process (s), and compare with comparable level shown in b. On the normal side fatty densities surround a dot of soft tissue (arrow) that we believe represents the location of the facial nerve. Other bony landmarks include pterygoid plate (p) and mandibular condyle (c).

2. Arrowheads denote discontinuous erosions of the mastoid.

CT scanning confirmed the tomographic findings. In addition, the fat planes around the mastoid process and between the mastoid and styloid processes were obliterated on the right. Multiple uneven areas of erosion were demonstrated in the external cortex of the mastoid process. The mastoid air cells were clear. A soft-tissue density was seen in the left external canal (Fig. 4).

The patient received gentamicin and carbenicillin and later moxalactam (Moxam, Eli Lilly). A right radical mastoidectomy and seventh nerve decompression were performed and followed five days later by a left radical mastoidectomy. The complicated postoperative course included poor diabetes control, gastrointestinal bleeding, a seizure, CVA, and acute renal failure. The patient died at home one month after his family removed him from the hospital against medical advice.

CASE II: A 76-year-old man presented with bilateral drainage of four months duration. The right external canal had been debrided once. The right temporomandibular joint was tender. His past medical history included diabetes for 25 years and a previous myocardial infarction and cerebrovascular accident (CVA). Physical examination showed tissue granulation in both external canals and right facial nerve paralysis. Tomography showed soft-tissue density in the right external canal and irregular erosion of the roof of the canal (Fig. 3). The middle ear was clear and the facial nerve canal was normal. *Pseudomonas* was grown from a culture of the external auditory canal. No *Pseudomonas* was cultured from the left external canal, but the patient was being treated with antibiotics at the time of this culture.

Portion of the seventh nerve were performed. The mastoid tip was removed. The patient continued four weeks of intravenous antibiotic treatment uneventfully and was discharged. There was no recurrence after six months.

CASE III: A 53-year-old man with a 20-year history of diabetes presented with right ear drainage and pain over the mastoid and parotid regions. There was right seventh nerve paralysis. The external canal was swollen and inflamed. CT showed soft-tissue density in the external auditory canal. Erosions of the outer cortex of the mastoid were seen both on plain radiographs and CT scans (Fig. 5). Soft-tissue density obliterated the normal fascial anatomy around the mastoid, including the area between the styloid and mastoid processes. The middle ear was clear although the mastoid air cells were partly opacified.

*Pseudomonas* was cultured and treatment with antibiotics begun. Modified radical mastoidectomy and facial nerve decompression were performed. The patient did well but returned eight weeks after surgery with increased pain. CT now showed a soft-tissue mass in the infratemporal fossa consistent with an abscess. There was also increased soft-tissue density obliterating the fat surrounding the carotid sheath and extending into and replacing the normal fascial planes in the parapharyngeal space. A superficial parotidectomy and debriement of the infratemporal fossa were performed and antibiotics continued.

One month later hoarseness and dys-
Figures 3 and 4. Case II.

3. AP tomogram with arrowheads indicating areas of erosion in roof of external auditory canal.

4a. Soft-tissue density seen in the right external canal (long arrow). Erosions are seen along the lateral aspect of the mastoid. Malignant external otitis can be seen on the right side.

4b. Increased density obliterating fat planes between the mastoid tip (m) and styloid process (s) on the right.

4c. Coronal CT scan on the right side showing clear middle ear (black arrow) and soft-tissue density in external canal with partial erosion of the roof (arrowhead). S is the styloid process.

4d. Coronal view of the right side showing multiple erosions of the mastoid (arrowheads). (o) indicates the odontoid.

4e. Coronal view of the left side showing soft-tissue density in the external canal at the osseous-cartilaginous junction. This is thought to represent granulation tissue on the floor of the canal that had been noted clinically (arrow).
phagia with paralysis of cranial nerves VIII, IX, X, XI, and XII developed in the patient. The patient was given intensive therapy that included the antibiotic moxalactam. CT now demonstrated the extension of edema slightly beyond the midline in the nasopharynx with distortion of the nasopharyngeal airway. It was unclear which portion of this progressive soft-tissue density occurred as a result of surgery. The patient survived with partial recovery of function of cranial nerves IX, X, XI, and XII at the time of discharge.

**Case IV:** A 58-year-old man with a 15-year history of diabetes presented with two months of pain and drainage from the left ear. His medical history included congestive heart failure, stroke, hypertension, retinopathy, and bilateral below knee amputation. The patient had left seventh nerve paralysis and pain on palpation of the temporomandibular joint. Tomography showed soft-tissue density in the external canal, middle ear, and mastoid antrum. There was destruction of the anterior portion of the tympanic ring creating a communication between the external canal and glenoid fossa (Fig. 6). The mandibular condyle was displaced anteriorly causing the mandible to deviate to the opposite side on closure. The facial nerve canal appeared normal. There was erosion of the roof of the glenoid fossa.

CT again showed the soft-tissue density in the external canal and mastoid air cells. The communication of the external canal and glenoid fossa was likewise demonstrated, and the erosion from glenoid to middle cranial fossa was confirmed (Fig. 7).
Soft-tissue swelling was noted anterior to the external canal and lateral to the mandible, and the fat planes around the facial nerve were obliterated. There was slight asymmetry in the lateral pterygoid muscles that may have resulted from the displacement of the mandibular condyle.

*Pseudomonas* was cultured from the external canal. Carbenicillin and tobramycin were administered for two weeks, after which a left radical mastoidectomy was performed. At surgery the communication with the glenoid fossa was debrided, and the osteitic bone from the superior aspect of the glenoid was removed leaving exposed dura at the floor of the middle cranial fossa. The patient recovered with a mild residual seventh nerve paresis.

**DISCUSSION**

Malignant external otitis is an aggressive *Pseudomonas* infection that begins in the external ear canal and progresses to involve surrounding bone and soft tissues. Initially reported by Meltzer and Kelemen (9) in 1959, the disease was described in considerable detail in 1968 by Chandler (1), who coined the term malignant external otitis (MEO).

Patients with MEO are almost exclusively diabetic. Some elderly nondiabetic patients have been described, as well as several pediatric patients who were either suffering from severe malnourishment or had some form of systemic disease making them compromised hosts (10, 11). Three of our four patients were diabetic.

MEO begins as a small granulation on the floor of the external auditory canal, most often at the osseous-cartilaginous junction. If the disease is limited to this area control can usually be obtained through antibiotics and minor surgery. Eradication is much more difficult when the disease extends beyond the external auditory canal. Because the tympanic membrane is resistant to the spread of the disease middle ear involvement is not typical and is usually the end result of involvement of the mastoid. The mastoid air cells become involved when the disease erodes through the cortex of the mastoid, which is also a considerable impediment to spread. This occurs with osteomyelitis, one of the manifestations of MEO. Osteomyelitis can involve the mastoid only or it can spread throughout the temporal bone, usually sparing the bony labyrinth, which is also resistant. The easiest means by which the disease spreads from the external auditory canal is thought to be the inferior route. The inflammatory process extends through...
small clefts in the cartilage of the external auditory canal. These clefts are called the fissures of Santorini and yield access to the subtemporal space. From here the disease can progress anteriorly into the region of the parotid but, more significantly, it can extend medially beneath the temporal bone toward the petrous apex. In this case the inflammatory process first encounters the facial nerve at its exit from the stylomastoid foramen followed by cranial nerves IX, X, and XI at the jugular foramen and finally nerve XII at the hypoglossal canal.

Involvement of these nerves indicates that the patient has a much lower chance of survival. Involvement of the jugular bulb can cause a sigmoid sinus thrombosis that along with meningitis can be a direct cause of death. Extension across the midline to the opposite side is not uncommon.

*Pseudomonas* tends to follow vascular and fascial planes (12, 13), which explains why the disease usually progresses through bone as osteomyelitis or through the subtemporal area as a fascitis, rather than the air spaces. The middle ear is often clear even in patients with cranial nerve abnormalities.

Some reports have described involvement of the facial nerve at various points along the nerve's circuital course through the temporal bone (2–4). Most papers have described involvement in or below the stylomastoid foramen (1, 5–8). This area is the subtemporal space and it is here that CT can play a major role in evaluation.

Tomography has been used to demonstrate both bony and mastoid involvement (14, 15) but it cannot visualize the subtemporal area. Technetium 99-labeled phosphate bone scanning agents (16) and gallium scanning (8) have also played a role in staging the disease. CT not only demonstrates bony erosion, but can provide a good demonstration of the extent of involvement in the mastoid air cells and aid in the evaluation of aeration of the middle ear. In addition, the ability of CT to image fat and muscle planes provides necessary information about the very important subtemporal area. The obliteration of the fat planes can be demonstrated without intravenous enhancement, but initial scanning is performed with enhancement to demonstrate the relationship of the pathology to the great vessels in the subtemporal area.

When serial CT scans are obtained through the temporal bone, the facial nerve can often be followed inferiorly toward the stylomastoid foramen. The nerve leaves the foramen and enters the subtemporal area between the styloid process and the mastoid tip. This is indicated by CT as a small area of fat density slightly posterior and lateral to the base of the styloid process and slightly anterior and medial to the mastoid tip. There is normally a small dot of soft-tissue density in the center of this small fat collection that may in fact be cranial nerve VII in combination with the stylomastoid artery. In all four of our patients, this small fatty lucency was obscured by the inflammatory process as it passed medial to or around the mastoid tip.

In CASE III there was progression of the disease after the initial CT scan. An abscess in the infratemporal fossa was identified on a repeat scan. There was also involvement of the area around the jugular bulb. The intravenously injected contrast agent showed the position of the jugular vein and the surrounding inflammatory tissue, marking the location of involvement of cranial nerves IX, X, and XI. Eventually the disease process extended across the midline, distorting the pharyngeal airway on yet a third CT examination. The patient had been explored several times after the first scan. Surgical procedures would, of course, make evaluation of the fat planes much more difficult, but in the case of the later CT scans the radiographic findings fit well with the clinical picture.

In addition to the information about the subtemporal area supplied by the CT scan, excellent visualization of other areas of involvement were also obtained. CT demonstrated bony erosion of the mastoid tip at least as well as pluridirectional tomography. Mastoid air space involvement was easier to visualize on CT. Pluridirectional tomography gave precise detail about the integrity of the vertical portion of the facial nerve canal immediately above the stylomastoid foramen. Middle ear involvement could be assessed by either pluridirectional tomography or CT.

In CASE IV, CT showed the erosion of the glenoid fossa and floor of the middle cranial fossa and provided information about the condition of the temporal lobe immediately above, showing that it was free of disease.

Malignant external otitis can be differentiated from other forms of external otitis by characteristic clinical information and identification of *Pseudomonas*. A carcinoma of the external auditory canal could have radiographic findings similar to malignant external otitis. There may be considerable bony destruction in both but the predominance of subtemporal extension is more characteristic of MEO. Definitive diagnosis is made by biopsy.