MR and CT of Squamous Cell Carcinoma of the Middle Ear and Mastoid Complex

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Squamous cell carcinoma infrequently arises in the middle ear and external auditory canal. Mastoid complex primary carcinoma is extremely rare. The site of origin of these tumors may be obscure when large destructive lesions are encountered at the initial presentation. Imaging techniques may provide a clue as to the origin of these carcinomas. The complementary role of CT and MR is illustrated in two cases: one with primary carcinoma of the mastoid complex and the other with carcinoma of the external auditory canal/middle ear. The sclerosis of bone evident on CT and the striking dural enhancement seen by MR in these cases is discussed.

Case Reports

Case 1

A 78-year-old woman presented with a 2-month history of a progressively enlarging, painful mass behind the left ear. She had no history of any otologic abnormality. Physical examination confirmed the presence of a large retroauricular mass. There was no otorrhea, and although the external auditory canal was severely narrowed, tumor was not identified within it. Hearing was diminished on the left side. Cervical adenopathy was absent. An MR scan was obtained on a 1.5-T superconducting magnet (General Electric, Milwaukee). T1-weighted (800/20/2 TR/TE/excitations), T2-weighted (2000/80/1) axial images (Fig. 1A) revealed a large destructive mass centered in the posteroinferior aspect of the left temporal bone that was isointense with brain. MR images obtained after administration of gadopentetate dimeglumine (0.1 mmol/kg weight) (Fig. 1B) revealed irregular enhancement of the mass. The external auditory canal was narrowed by the encroaching lesion but was patent. Tumor extending medially along the undersurface of the temporal bone was readily apparent on the MR images. Dural enhancement adjacent to the lesion was seen best on coronal images. A high-resolution CT scan (GE 9800; General Electric, Milwaukee) of the temporal bone with IV contrast demonstrated a hypodense, irregularly enhancing destructive lesion centered in the left mastoid complex. Tumor involvement along the undersurface of the temporal bone was less conspicuous (Fig. 1C). The external auditory canal appeared to be occluded (Fig. 1D). Although the middle ear cleft was filled with soft tissue, the ossicles were intact (Fig. 1E). The otic capsule was not invaded. Sclerosis of bone in the middle cranial fossa was also observed. The results of a biopsy of the lesion revealed squamous cell carcinoma. Thorough clinical and radiologic evaluation of the patient, including a head and neck CT scan, demonstrated no evidence of an occult carcinoma. Hence, the neoplasm was thought to have originated in the mastoid complex. She was treated with radiotherapy and chemotherapy; survival was less than 1 year.

Case 2

A 62-year-old man with a 30-year history of a draining left ear presented with several months of pain and swelling behind the left ear. He also gave a history of an "ear operation" (probably myringotomy and tympanoplasty) on that side in 1950. Physical examination revealed a left retroauricular mass and a mild yellowish discharge from the external meatus. No adenopathy was appreciated. The external auditory canal was filled with tumor and hearing was diminished on the left. An MR scan was obtained on a 1.5-T superconducting magnet (General Electric, Milwaukee). T1-weighted (800/25/2) and T2-weighted (3000/90/1) axial images revealed a destructive mass of the left temporal bone that was isointense with brain. Images obtained after administration of gadopentetate dimeglumine (0.1 mmol/kg weight) demonstrated invasion of the external auditory canal; the mass itself enhanced in a fairly uniform fashion. Prominent dural enhancement adjacent to the lesion was best seen on T1-weighted coronal images (500/20/2) (Fig. 2A). A high-resolution CT scan (GE 9800; General Electric, Milwaukee) demonstrated an isodense destructive temporal bone mass (contrast agent was not administered owing to mild renal insufficiency). The external auditory canal was occluded. The middle ear cleft was filled with soft tissue and the ossicular chain was almost completely destroyed (Fig. 2B). Prominent sclerosis of bone in the middle cranial fossa was observed (Fig. 2C). Partial resection of the lesion revealed squamous cell carcinoma probably arising in the middle ear or bony external canal. The patient received postoperative radiotherapy and chemotherapy.

Discussion

Squamous cell carcinoma of the ear, arising in the external auditory canal, middle ear cleft, or in a mastoid cavity is rare. Estimates of its frequency in the general population vary from 0.004% to 0.0006% [1, 2]; it accounts for approximately 1 in

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Fig. 1.—Case 1: 78-year-old woman with 2-month history of progressively enlarging, painful mass behind left ear.

A, T2-weighted (2000/80) axial MR image shows large destructive mass, isointense with brain, in left temporal bone. The mass extended 4 cm caudal to this level.

B, Enhanced T1-weighted (800/20) axial MR image reveals patchy enhancement of mass. External auditory canal is stenosed but patent (short arrows). Tumor extends along undersurface of temporal bone in the region of the jugular fossa (long arrows).

C, Tumor extension in the region of the jugular fossa is less apparent on the enhanced CT scan (arrows).

D, On enhanced CT scan, extrinsic compression cannot be distinguished from invasion of the external auditory canal. Tumor has extended into left temporomandibular joint (arrows).

E, Bone algorithm shows that ossicular chain is intact despite engulfment by soft tissue. There is thickening and sclerosis of bone in left middle cranial fossa (arrows).

Fig. 2.—Case 2: 62-year-old man with 30-year history of draining left ear and several months of pain and swelling behind left ear.

A, Enhanced T1-weighted (500/200) coronal MR image shows fairly uniform enhancement of a large destructive left temporal bone mass. The dural enhancement, which was seen on multiple contiguous sections in both axial and coronal planes, is of greater intensity than the tumor itself and tapers away from the lesion. There is enhancement between the tumor and the dura as well.

B, Bone algorithm shows disruption of ossicular chain (arrow).

C, Bone algorithm at slightly lower level shows striking thickening and sclerosis of bone ventral to tumor (arrows).
5000 otologic conditions [3]. Squamous cell carcinoma is the most common malignant tumor of the ear. Other neoplasms include adenocarcinoma, lymphoma, melanoma, malignant parangangioma, and metastases. Patients in the fifth to seventh decade are most often affected. The classic presentation of otalgia and persistent ear drainage (often bloody) in the setting of chronic ear infection is seen in at least one third of cases [3–5]. Cholesteatomas are occasionally present as well. Late clinical manifestations include facial palsy, deafness, vertigo, tinnitus, and trismus [5]. The disease is usually in an advanced state of invasion at the time of diagnosis. Tumors involving the external auditory canal, middle ear, and mastoid cannot usually be labeled as to the site of origin [3–8]. Primary mastoid carcinoma is most often diagnosed as an incidental finding during mastoidectomy to control chronic infection [6].

Carcinomas of the ear spread primarily by direct extension. Adenopathy is uncommon and involves retropharyngeal, preauricular, and upper cervical nodes [8]. Distant metastases are rare. Tumors arising in the cartilaginous auditory canal tend to spread into the parotid gland, posterior auricular sulcus, and pinna, since the walls of the cartilaginous external canal are easily infiltrated. Because the dense bone of the osseous external canal is a more effective barrier to tumor growth, tumors arising in this location initially spread into the middle ear and then into deeper structures. When the middle ear is primarily or secondarily involved, lesions may grow upward through the tegmen tympani and posteriorly through the mastoid air cells and eventually infiltrate the dura. Neoplastic cells can spread along the fascial planes of the eustachian tube and involve the lateral nasopharynx. Caudal growth may involve the carotic canal and jugular fossa [4, 7, 9]. The hard avascular bone of the otic capsule is relatively resistant to tumor, and direct invasion of the inner ear signifies far advanced disease [4]. Other findings that may be seen are erosion of the articular fossa of the temporomandibular joint and facial nerve involvement.

The MR and CT characteristics in our two cases demonstrate important similarities as well as differences. The patency of the external auditory canal, preservation of the ossicular chain, and more inferior location of the tumor in the first patient contrast with the external canal invasion, ossicular destruction, and more superiorly located lesion in the second patient. The constellation of findings in the former case strongly implicates tumor origin in the mastoid complex. Unlike CT, the improved soft-tissue contrast of MR imaging appears to permit more accurate assessment of the status of the external auditory canal; however, further cases would be required to confirm this. Although the patency of the external canal can be determined clinically, this information may not be available to the radiologist at the time of interpretation. In both cases, prominent sclerosis of bone in the middle cranial fossa was seen on CT. By MR, the tumors were isointense with brain on short and long TR/TE images. In addition to tumor enhancement, considerable dural enhancement after contrast administration was observed in both. All of these features could potentially cause confusion with malignant meningioma. Various authors [10, 11] have discussed the importance of the “flare” sign or dural “tail” associated with meningiomas. Criteria proposed by Goldsher et al. [11] to define a dural “tail” were met in one of our patients (Fig. 2A), although, in the experience of these authors, dural enhance-

ment was never seen between the meningioma and the underlying skull or dura mater (unlike our case). With regard to sclerosis of bone, this may in some instances be a manifestation of chronic inflammation (case 2), although our first patient gave no history of infection. We believe that the elegant bone detail provided by CT, combined with the superior soft-tissue contrast of MR, result in the most accurate definition of tumor extent.

The therapy of squamous cell carcinoma of the ear and temporal bone depends on the extent of disease. When limited to the cartilaginous or bony canal, it can be treated effectively by resection of the entire ear canal, including the tympanic membrane [12]. Middle ear and deeper temporal bone extension are associated with a much poorer prognosis [12–14]; subtotal temporal bone resection, possibly in association with radiotherapy, will occasionally result in long-term survival. In one large series, there was little chance for cure when tumor was left behind at the time of surgery [13]. Local recurrence, followed by intracranial extension, is the most common cause of treatment failure.

The differential diagnosis of destructive temporal bone lesions is extensive and includes both benign and malignant causes such as necrotizing external otitis, tuberculous otitis, Langerhans histiocytosis, osteoclastoma, epidermoid, cholesterol granuloma, primary bone neoplasms, as well as the malignant soft-tissue tumors previously mentioned. Both benign and malignant lesions can cause bone destruction consequent to pressure effect [9]. It is hoped that some of the features of squamous cell carcinoma described in this report will help in the identification and localization of this disease.

REFERENCES