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Pictorial Review

Masses and disease entities of the external auditory canal: Radiological and clinical correlation

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A wide spectrum of disease entities can affect the external auditory canal (EAC). This review describes the normal anatomy of the EAC. Congenital abnormalities, infections, neoplasms, and miscellaneous conditions, such as cholesteatoma and acquired stenosis, are shown with reference to clinical relevance and management. Cases have been histologically confirmed, where relevant. The EAC is frequently imaged — for example, on cross-sectional imaging of the brain — and this review should stimulate radiologists to include it as an important area for review.

Introduction

The external auditory canal (EAC) extends from the external auditory meatus to the tympanic membrane. Whilst amenable to direct clinical examination, radiology plays a significant role in the diagnosis of a variety of conditions (including congenital abnormalities, infection, and neoplasms). Imaging helps delineate the extent of disease and the presence of co-existing diseases.

This review demonstrates the normal imaging anatomy and highlights the range of disease entities and differential diagnoses for abnormalities of the EAC with clinical correlation.

Anatomy

The adult EAC measures 2–3 cm in length and forms a tunnel through the temporal bone. The lateral third of the EAC is directed posterosuperiorly and is cartilaginous, being continuous with the pinna. The narrowest part of the EAC, the isthmus, separates the cartilaginous EAC from the bony medial two-thirds, which courses anterosuperiorly. The floor, anterior wall, and inferoposterior wall are formed by the tympanic portion of the temporal bone, the roof, and superoposterior wall by the squamous portion and the mastoid portion lying posteriorly. The mandibular condyle and fossa are an anterior relation of the EAC (Fig 1). The EAC is limited medially by the attachment of the tympanic membrane at the tympanic annulus.

The EAC is lined by keratinizing stratified squamous epithelium. The skin over the lateral cartilaginous aspect is thicker, with a subcutaneous layer, hair follicles, sebaceous glands, and modified apocrine glands (ceruminous glands, which contribute to wax formation). These are absent in the skin over the bony canal, which closely adheres to the underlying periosteum.

Imaging

Thin-section (ideally <1 mm collimation) high-resolution computed tomography (HRCT) of the temporal bone is sufficient for demonstrating the anatomy of the EAC, and is the usual first-line radiological investigation in the evaluation of

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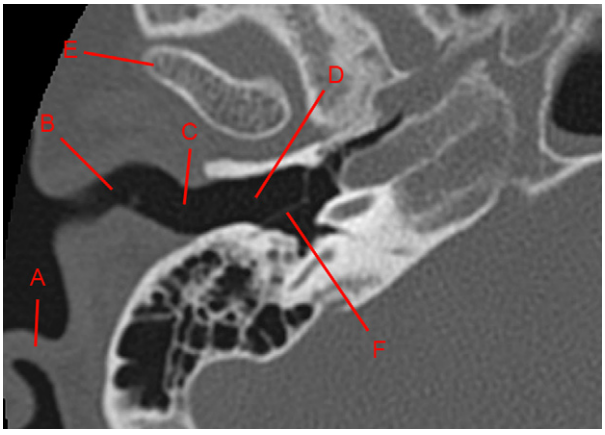

Figure 1 Axial HRCT of the right EAC and its relations. A = pinna; B = cartilaginous EAC; C = isthmus; D = bony EAC; E = mandibular condyle; F = tympanic membrane.

EAC masses and anomalies. The anterior and posterior walls are best visualized on axial images, with sagittal and coronal reformats useful for examining the roof and floor. Disease spread into the middle ear is best assessed using axial sections.

HRCT is a useful means of identifying bony involvement, a feature of many EAC conditions, although magnetic resonance imaging (MRI) is of value when skull base disease is suspected, for example. There are a limited number of indications for radionuclide imaging in EAC disease.

Congenital abnormalities

EAC atresia

Developmental abnormalities of the EAC arise due to defects of the first and second branchial arches and first branchial cleft during embryogenesis. EAC atresia (ranging from membranous atresia to complete bony occlusion) is uncommon, with a reported incidence of 1 in 10–20,000, and more commonly affects the right ear. It is bilateral in one-third of patients,[1] often forming part of syndromes with craniofacial anomalies, such as Goldenhaar syndrome or Treacher Collins syndrome.[2] Patients have conductive hearing loss, with microtia or hypoplastic pinna a common association. It is important to note that the outer and middle ear are embryologically separate from the inner ear. Therefore, associated inner ear anomaly is rare.

Management often includes the use of bone-anchored hearing aids (BAHA) although operative intervention is sometimes required. Preoperative evaluation with HRCT is vital to exclude associated abnormalities (e.g., cholesteatoma

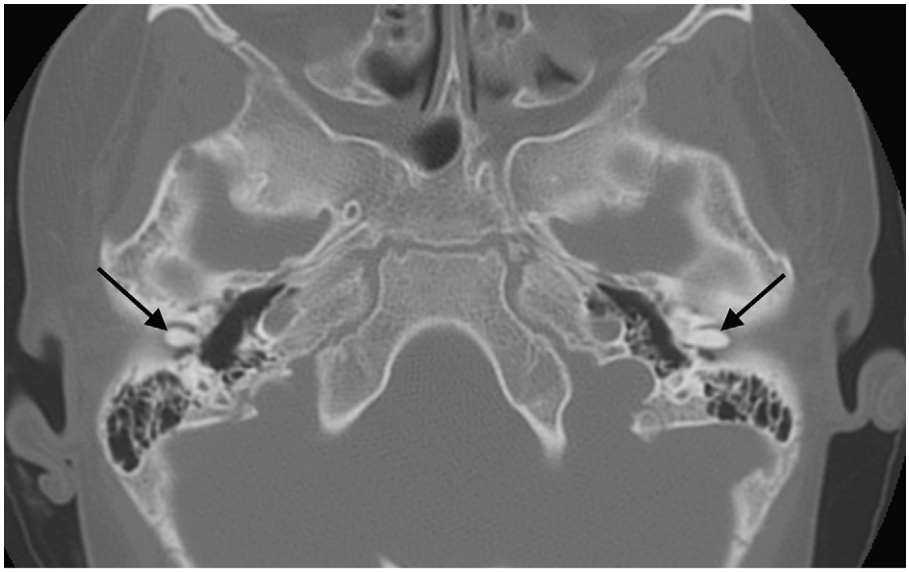

Figure 2 A 4-year-old patient with conductive hearing loss and microtia. Axial HRCT (a) demonstrates bilateral bony atretic plates (black arrows) lying medially in the EACs. A coronal reformat (b) shows fusion of the handle of the right malleus to the atretic plate (white arrow).The pinnae are seen to be hypoplastic. The patient was ultimately fitted with a BAHA.

or aberrant facial nerve course[1,3–6]) and facilitate surgical planning. This is best performed after the age of 5 years, once the temporal bone has sufficiently developed.

In congenital bony atresia, there is a bony plate at the level of the tympanic membrane, with variable EAC pneumatization (Fig 2). The malleus and incus may be fused to this bony plate. Patency of the eustachian tube orifice and aeration of the middle ear cavity must be determined, as reduced middle ear space is associated with a poor surgical outcome.[1,3–6] Therefore, it is important to determine whether or not the tegmen tympani is low-lying, and the distance between the promontory and the lateral attic wall should be greater than 3 mm. The incudomalleolar complex, incudostapedial articulation, and round and oval window patencies[3,4] must be carefully assessed for dysplasia. Identification of stapes has the greatest bearing on surgical outcome.[7]

First branchial anomalies

First branchial cleft anomalies are rare and variable. They can present as a recurrent abscess in the preauricular skin. The upper end can open into the EAC or middle ear and the lower end can open anywhere along the angle of the mandible[8,9] (Fig 3). Sinuses can be complex (e.g., EAC duplication) and have a variable relationship to the facial nerve; hence, preoperative MRI is required for further assessment. Sinograms may also be of value. Very careful dissection with intra-operative monitoring is required.[10]

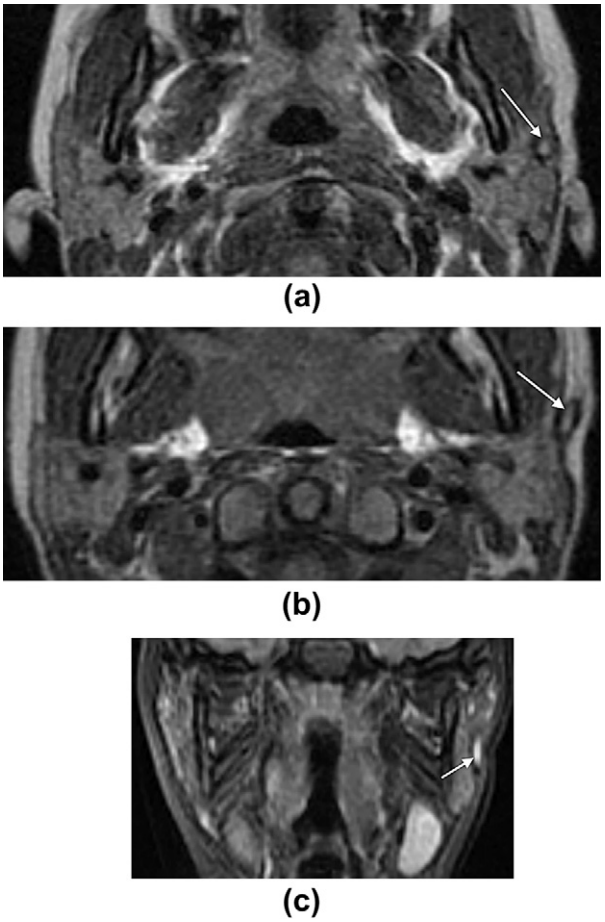

Figure 3 This adolescent patient presented with a discharging sinus from the left angle of mandible and pain radiating to the ear. Axial T1 (a and b) and coronal short tau inversion recovery (STIR) (c) MRI show a 2 mm sinus tract (white arrows) extending from the cartilaginous EAC to the skin over the angle of the mandible. This was excised via a parotidectomy and was a histologically confirmed first branchial cleft tract.

Infection

Malignant otitis externa

Radiological imaging is necessary if malignant otitis externa (MOE, also known as necrotizing otitis externa) is suspected. The differential diagnosis includes benign necrotizing externa, canal cholesteatoma, osteoradionecrosis, chronic inflammatory conditions, and tumours of, or metastases to, the temporal bone. MOE is an invasive, potentially life-threatening infection (mortality rate of approximately 20%). It is usually caused by *Pseudomonas aeruginosa* and involves the EAC and skull base, typically in immunocompromised or diabetic patients. Symptoms are non-specific, including otorrhoea, headache, and severe, persistent otalgia. Patients are often afebrile with raised inflammatory markers but a normal white cell count. Cranial nerve palsies (most commonly facial nerve) can occur in advanced disease along with osteomyelitis of the skull base and temporomandibular joint (TMJ; Fig 4). Diagnosis relies on a combination of clinical findings and radiological features.

CT and MRI play complementary roles in the evaluation of suspected MOE. CT delineates the extent of bony erosion, and MRI identifies cranial nerve and skull base involvement.[11–13] MRI is less useful in the follow-up of MOE as abnormal enhancement may persist for many months following symptomatic resolution.[14]

Radionuclide imaging is also used. Technetium 99 m bone scintigraphy yields a positive result when CT is normal due to the osteomyelitic process, and is useful to monitor the therapeutic response of the disease [as is positron-emission tomography (PET) or gallium 67 imaging with single photon-emission CT (SPECT),[12] which can be used to see whether MOE is still active].

Neoplasms

A range of primary benign and malignant neoplasms can involve the EAC. Direct spread from head and neck tumours and distant metastatic disease also occur.

Cutaneous malignancies

Basal cell carcinoma (BCC) and squamous cell carcinoma (SCC) comprise the most common group of EAC malignancies in Caucasian populations. Both malignancies can involve the pinna and periauricular tissues. EAC and

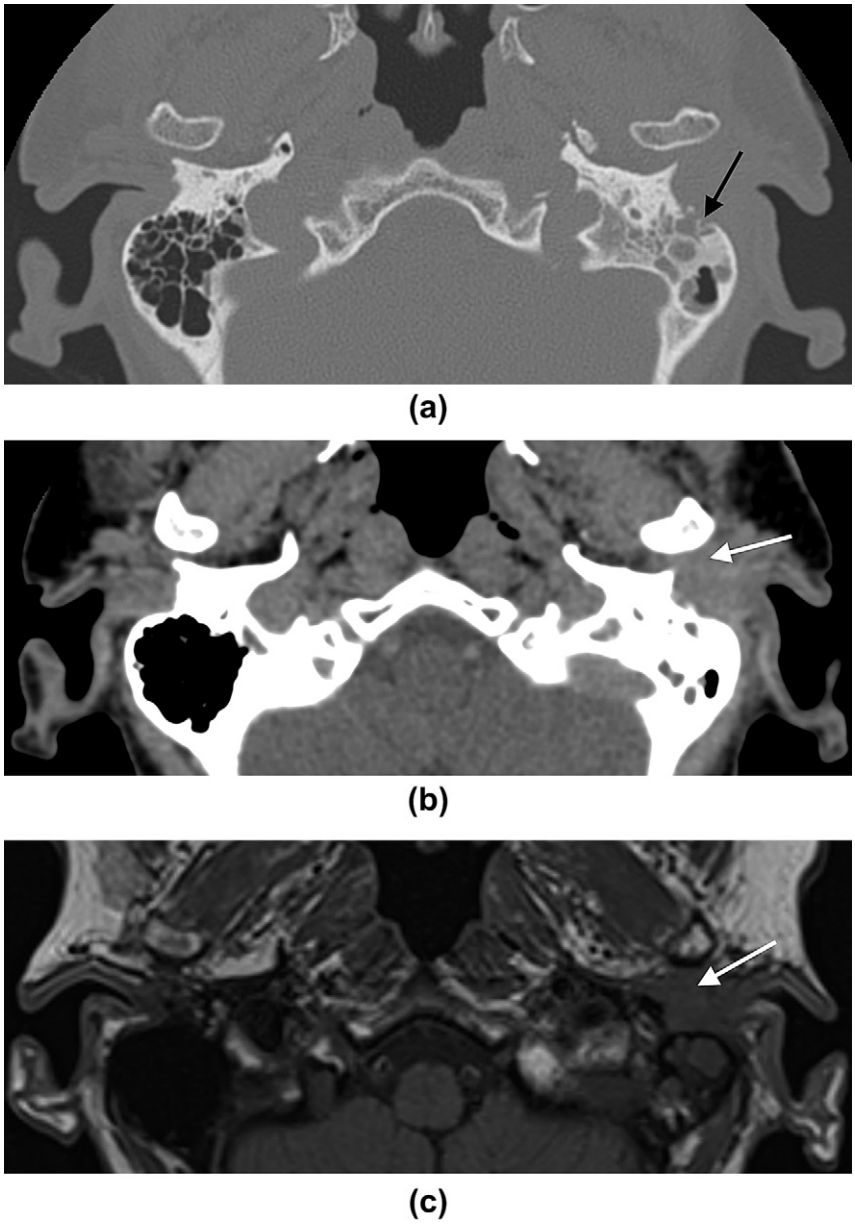

Figure 4 This patient presented with severe left otitis externa and underwent imaging to establish whether there was bony involvement. Axial CT on bony windows (a) shows erosion of the anterior wall of the mastoid air cells (black arrow), which are opacified. Axial CT on soft-tissue windows (b) and T1-weighted axial MRI (c) show circumferential soft-tissue thickening in the EAC, extending around the mandibular condyle (white arrows). This was a confirmed case of MOE.

temporal bone involvement can occur following incomplete excision of a primary pinnal or periauricular lesion.[15] BCC is more common and tends to involve sun-affected areas. SCC is more likely to be invasive, with higher morbidity and mortality. Imaging is required in BCC only if the EAC is involved (Fig 5). CT is useful in SCC (Fig 6) to assess nodal spread and local and bony invasion, which will determine the feasibility and extent of surgical resection. Radical neck dissection or parotidectomy may be necessary in addition to EAC sleeve resection. Surgery is not usually appropriate if the extent of malignancy necessitates complete temporal bone excision,[12] but localized EAC SCC has a 5 year survival of around 50%.[16]

Malignant melanoma of the pinna is not uncommon (up to 14% of head and neck melanomas[17]) and is related to sun exposure, and can involve the EAC by superficial spread. There are rare cases of primary EAC melanoma, presenting as a dark lesion.[17,18] Imaging defines the extent of disease, with bony involvement delineated by CT.

Metastases

The temporal bone (especially the vascular petrous portion) is a relatively common site for distant and local metastases. Breast, lung, and renal malignancies are the most common primary sources for distant metastases.

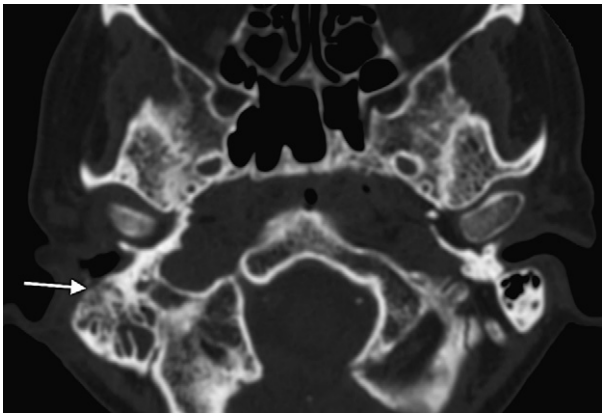

Figure 5 Axial HRCT showing bony erosion (white arrow) by a pathologically proven BCC.

Fig 7 shows metastatic prostate cancer involving the EAC. Local malignancies frequently involve the temporal bone. Neoplastic spread from inside the cranium, via the leptomeninges or subarachnoid space, is also well documented.[19,20] There have been reports of EAC metastases (from colorectal and renal primaries) which present as polypoid lesions within the canal, without temporal bone involvement.[21,22]

Temporal bone metastases can involve the EAC directly or indirectly, via lymphatic or vascular spread. The middle ear and EAC may be synchronously affected without tympanic membrane involvement.[19] Otological symptoms or facial nerve palsy may be the initial manifestation of a malignancy, and temporal bone metastases, whilst often asymptomatic, may mimic chronic inflammatory disease of the external ear canal.[19] It is the authors' practice to evaluate the temporal bone on bony windows CT, even on "routine" CT of the brain, to maximize chances of detection of these metastases.

Glandular tumours

Glandular EAC tumours are rare (around 150 cases in the literature[23]) and include ceruminous adenoma and

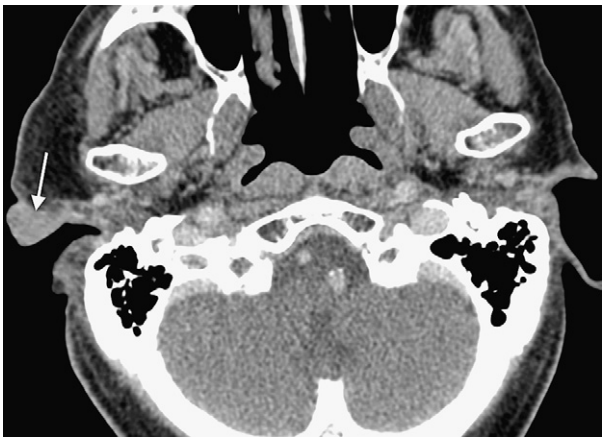

Figure 6 Axial CT demonstrates an enhancing mass centred on the tragus of the right ear (white arrow), extending along the cartilaginous EAC. This was found to be SCC histologically.

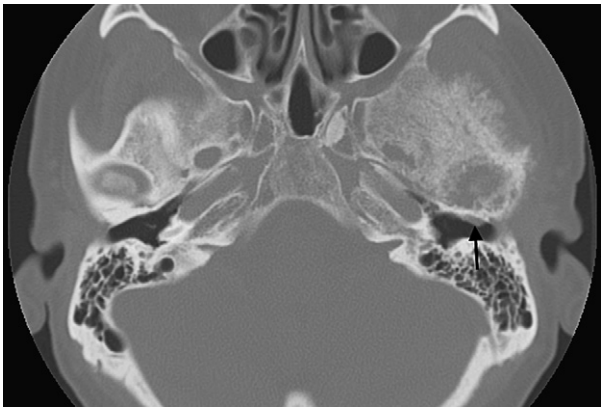

Figure 7 A patient with known primary prostate cancer was seen to have multiple osteoblastic metastases on bone scintigraphy, including one in the region of the left temporal bone. This was seen on CT as a destructive lesion of the greater wing of sphenoid and the squamous temporal bone, encroaching into the EAC with soft-tissue swelling evident in the canal (black arrow).

pleomorphic adenoma (PA; both benign) along with adenoid cystic carcinoma and adenocarcinoma.[24]

PA most commonly originates in the salivary glands. Rarely, it can develop from the EAC ceruminous glands (Fig 8), with around 30 cases reported.[24] It presents as

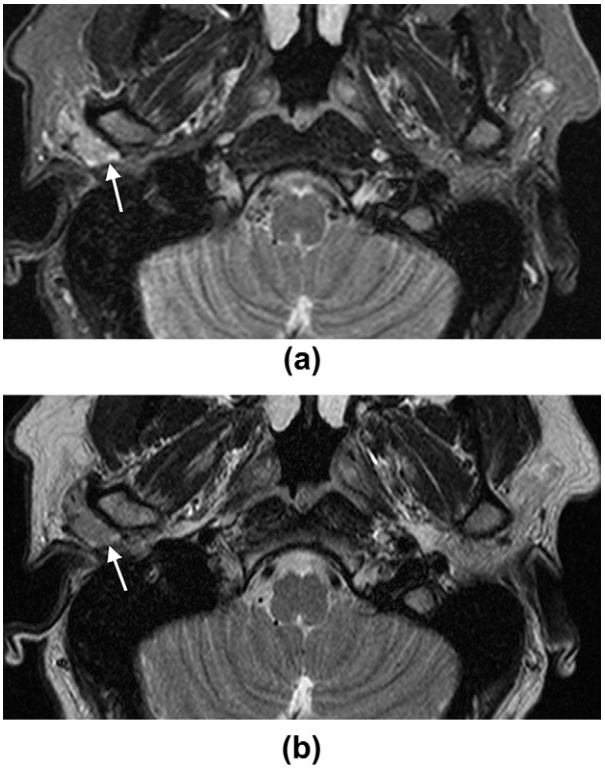

Figure 8 This patient presented with a 1 year history of painless swelling over the right TMJ in association with joint dislocation. Significant crepitus was evident clinically. (a) Axial STIR, (b) axial T2-weighted, and (c) coronal T1-weighted post-gadolinium MRI demonstrate a well-defined, enhancing mass closely related to the lateral aspect of the mandibular condyle (white arrows). Histology confirmed a ceruminous adenoma.

a smooth, round mass, which is asymptomatic if small but presents with non-specific symptoms (e.g., conductive hearing loss) if larger. Imaging aids surgical planning and helps clarify the nature of the mass, which is particularly important given the malignant differential diagnoses. PA itself has malignant potential, with carcinoma ex-PA representing the most common malignant mixed salivary gland tumour[25] (not yet been reported in relation to primary EAC PA). A well-defined enhancing homogeneous mass is seen on CT and MRI, returning a low signal on T1-weighted MRI and a high signal on T2-weighted MRI (relative to the parotid gland).[24,26] Imaging will clarify whether the PA extends from the parotid gland or arises from within the EAC.

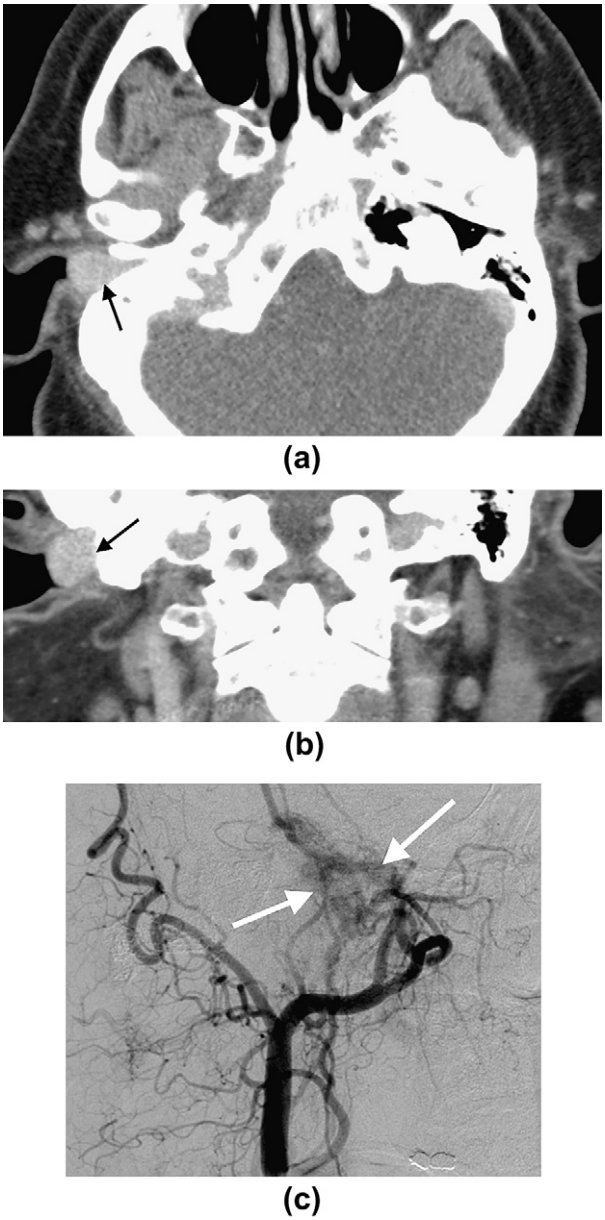

Figure 9 This patient presented with conductive hearing loss. (a) Axial and (b) coronal CT images show a homogeneously enhancing ovoid mass within the lumen of the right EAC (black arrows), causing minor erosion of the posterior wall. There is an intense vascular blush on angiography (c) (white arrows). This is a jugulotympanic paraganglioma eroding through the tympanic membrane. Bipolar excision was performed.

Paraganglioma

Paragangliomas (glomus tumours, chemodectomas) are uncommon neuroendocrine tumours. They are typically benign with locally aggressive features, and exhibit slow growth. In the head and neck, they are most often found at the carotid bifurcation or in the middle ear, originating from parasympathetic paraganglionic tissue.[27–29]

Rarely, paragangliomas can arise entirely within the EAC, with the tympanic membrane remaining intact.[30] This was initially suspected in the case in Fig 9, which was subsequently found to represent erosion of a jugulotympanic paraganglioma through the tympanic membrane into the EAC. CT demonstrates an avidly enhancing mass. MRI shows a characteristic “salt and pepper” appearance, with “salt” representing tumour stroma or areas of haemorrhage or slow flow on post-contrast T1-weighted imaging or on T2-weighted imaging and “pepper” indicating multiple flow voids from intratumoral vessels.[31–33] An intense blush is seen on catheter angiography.

Angiomyxoma

Superficial angiomyxomas are rare, benign tumours. They often form part of Carney’s complex, an autosomal dominant condition comprising multiple myxomas, skin hyperpigmentation, and endocrinopathy. They occasionally affect the EAC in isolation (Fig 10), with a polypoid mass occluding the canal and causing deafness. There are no characteristic imaging features, although avid contrast enhancement will occur due to its vascular nature. Lesions can recur following surgical excision, and pathological diagnosis of isolated angiomyxoma should prompt evaluation for further manifestations of Carney’s complex.[34]

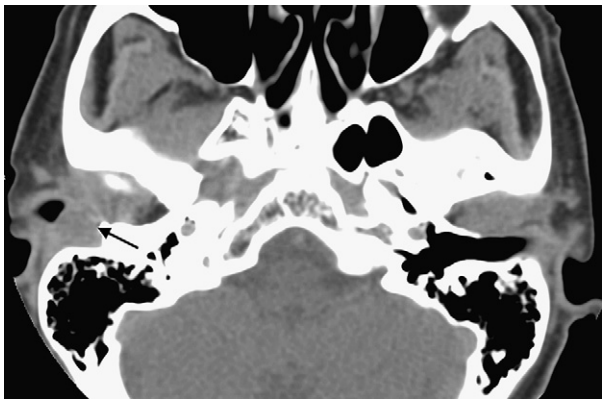

Figure 10 A broad-based mass arises from the posterior wall of the EAC (black arrow), abutting the isthmus and occluding the canal. Histopathological analysis confirmed angiomyxoma.

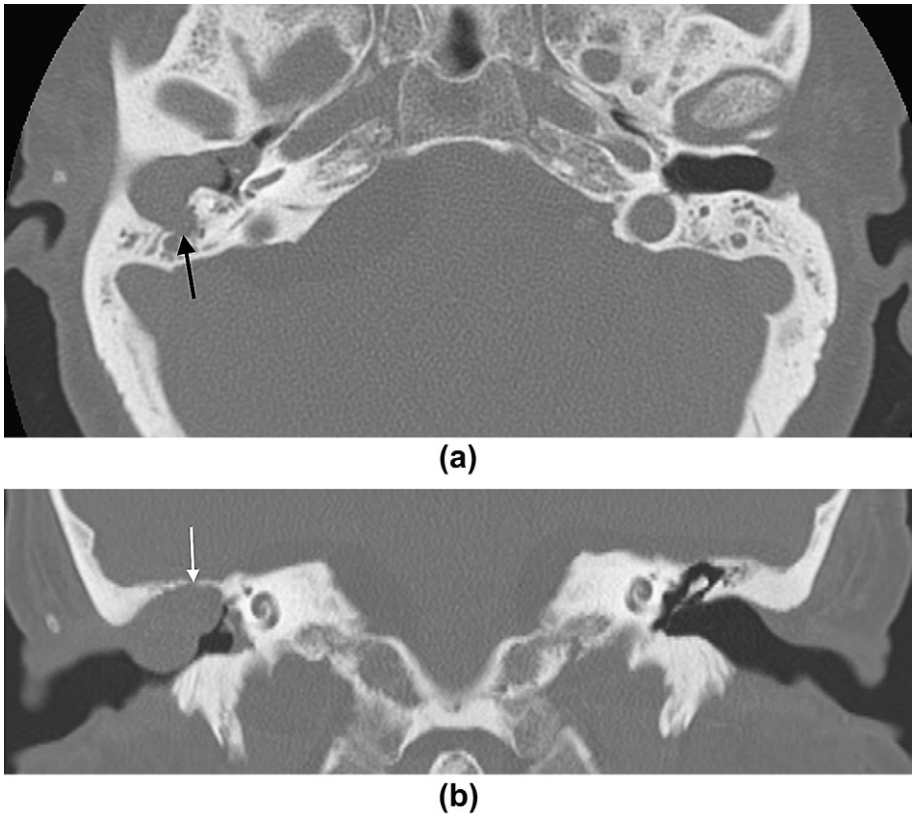

Figure 11 This patient had a prior history of right mastoidectomy. (a) Axial and (b) coronal HRCT show a lobulated, soft-tissue mass eroding the posterior wall of the EAC and mastoid (black arrow), extending into the middle ear with thinning of tegmen tympani (white arrow). Cholesteatoma was confirmed histologically.

Miscellaneous

Cholesteatoma

Acquired cholesteatoma is non-neoplastic condition of the petrous temporal bone. It may involve the EAC by spread from the middle ear through the tympanic membrane, but rarely arises in the EAC itself (Fig 11). Primary EAC cholesteatoma accounts for 0.1% of new presentations at otolaryngology clinics.[35] There is usually no identifiable cause, although it can occur following surgery, trauma, infection, or radiotherapy. Disruption of epithelial migration has been postulated as being causative.[36,37]

Presenting symptoms are non-specific, including chronic unilateral otorrhoea and otalgia. EAC cholesteatomas can cause significant bony destruction in the relative absence of symptoms.[36]

Imaging clarifies the diagnosis and defines the extent of the disease for surgical planning. On HRCT, EAC cholesteatoma is seen as a soft-tissue mass with bony erosion and associated intramural bony fragments. Invasion into the mastoid or through the tympanic membrane into the middle ear may be seen, and tegmen tympani or facial nerve involvement can occur. Diffusion-weighted imaging MRI has been shown to have a role in the evaluation of middle ear cholesteatoma,[38] especially in the assessment of residual or recurrent disease. MRI is rarely used in EAC cholesteatoma evaluation.

Keratosis obturans

Keratosis obturans (KO) is the most important differential diagnosis for EAC cholesteatoma, given the different management strategies. These entities were once incorrectly considered to represent the same disease process,[39] and there is a significant overlap between clinicoradiological features, but KO is considerably more common than primary EAC cholesteatoma. KO may be associated with bronchiectasis and sinusitis and tends to affect younger adults than cholesteatoma. It is typically bilateral and presents with severe, acute otalgia. On imaging, bony erosion is usually circumferential (rather than localized in EAC cholesteatoma) with a thickened tympanic membrane

Table 1
Features differentiating between osteoma and exostosis.

	Osteoma	Exostosis
Frequency	Less common	More common
Nature of lesion	True bone tumour	Reactive lesion
Lateralization	Unilateral	Bilateral
Number	Solitary	Multiple
Attachment to underlying EAC	Pedunculated	Broad based
Location	Lateral, near isthmus	Medial

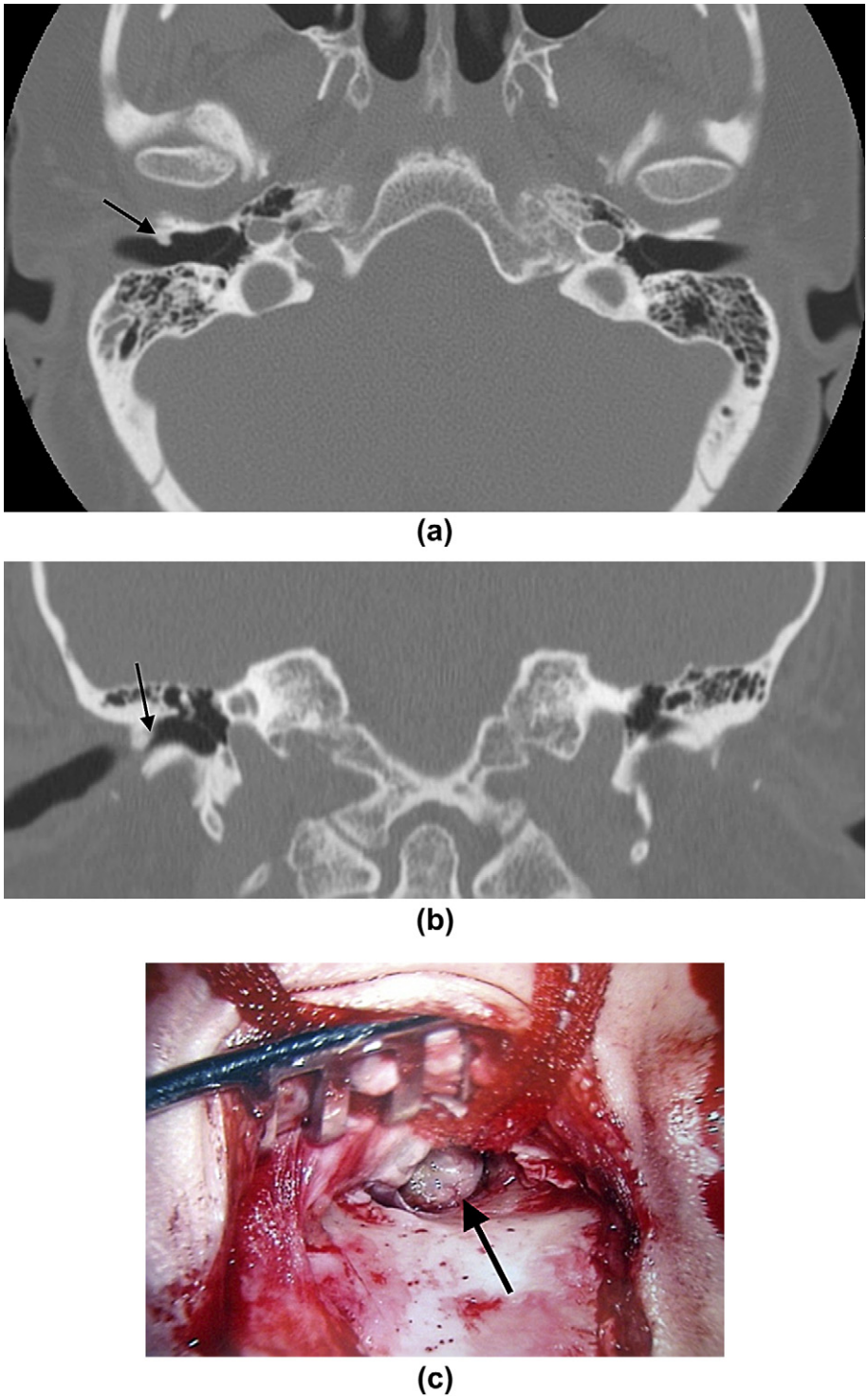

Figure 12 This 35-year-old with no prior ear problems presented with a bony growth in the right ear canal. (a) Axial and (b) coronal HRCT shows a well-circumscribed, pedunculate bony mass attached to the anterolateral bony EAC (black arrows). (c) Intra-operative clinical photography shows a bony mass (black arrow), consistent with osteoma.

(which is normal in EAC cholesteatoma) as a result of circumferential shedding of keratin squames.

Whereas permeatal or mastoid surgery is typically required for EAC cholesteatoma, a less invasive approach can be adopted for KO (often life-long microsuctioning of debris).

Cholesterol granuloma

Cholesterol granuloma (cholesterol cyst) is benign and most commonly arises in the temporal bone (particularly the petrous apex). It arises due to foreign body reaction, with haemoglobin breakdown leading to the accumulation

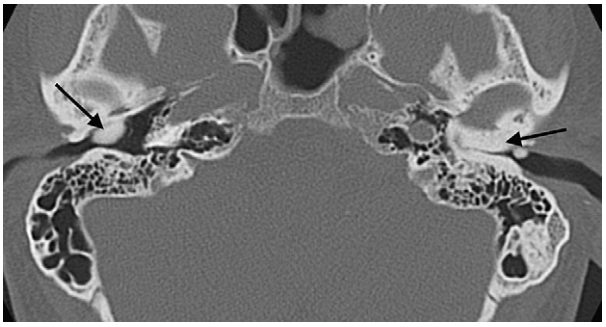

Figure 13 Axial HRCT showing bilateral, broad-based, irregular osseous lesions (black arrows) causing severe EAC narrowing, in keeping with exostoses. The patient was a keen kayaker, and the exostoses were treated surgically using canaloplasty.

of cholesterol crystals. Aural cholesterol granuloma usually occurs in the middle ear mucosa, although extension through the tympanic membrane into the EAC has been documented.[40] Cholesterol granuloma limited to the EAC has been reported, in which the lesion appeared clinically as a dark blue cystic mass.[41]

CT findings are non-specific, with a non-enhancing soft-tissue mass, isodense to brain, and with variable bony defects, mimicking a variety of conditions (including cholesteatoma). The cholesterol content means that cholesterol granuloma typically returns high signal on T1 and T2-weighted MRI sequences, relative to brain tissue.[40] There is no standardized approach to management, with conservative treatment, steroid therapy, and surgical removal and drainage all having been used.[40]

Bony growths

The differential diagnosis for EAC bony growths includes osteoma and exostosis. Histopathological analysis confirms the bony nature of the lesion but diagnosis may hinge on clinicoradiological features. HRCT is the first-line radiological investigation. A summary of differentiating features is shown in Table 1.

Osteomas (Fig 12) are slow-growing, benign lesions that affect both paediatric and adult patients. They occur in any part of the temporal bone, particularly the EAC.[42] They are usually asymptomatic and are incidental findings,[43] although conductive deafness and headache can occur.[44] HRCT helps exclude co-existing cholesteatoma — a rare but well-documented association — which occurs medial to the osteoma, presumed due to interruption of epithelial migration.[42,43]

EAC exostoses (Fig 13) are usually bilateral and multiple and arise as a result of repeated cold water exposures. They are also known as “surfer’s ear” due to their high prevalence amongst surfers (affecting 38% of surfers in one study[45]).

Surgery is indicated for bony growths if symptoms (including wax/water trapping, recurrent infection, and conductive deafness) are sufficiently severe. Osteomas are excised, whereas canaloplasty is used to widen the EAC in exostoses.

Acquired EAC stenosis

Acquired EAC stenosis is a rare entity with an estimated incidence of 0.6 in 10,000.[46] The most common cause is chronic otitis externa, particularly in hearing aid wearers.[47] Trauma (including surgery) and radiotherapy have also been implicated. A fibroproliferative inflammatory response causes granulation tissue to form, resulting in a narrow EAC.[47] Patients commonly present with conductive hearing loss.

Imaging is advisable, with HRCT being essential in post-traumatic stenosis to assess for the presence of cholesteatoma and to evaluate the middle ear cavity.[47] Treatment is with surgical canaloplasty, although recurrence is not uncommon.[47]

Conclusion

A heterogeneous range of disease entities can affect the EAC. Accordingly, it is important to include the EAC as a review area whenever temporal bone imaging is performed, and to avoid dismissing an apparent mass in the EAC as wax without careful evaluation. The presence of bony erosion and abnormal enhancement should be considered suspicious features, and there should be a low threshold for administering intravenous contrast media if symptoms such as otalgia are present.

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