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Computed tomography and magnetic resonance imaging of pathologic conditions of the middle ear

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Abstract

Computed tomography (CT) is an excellent technique for demonstrating even small abnormalities of the thin and complex bony structures of the middle ear. For this reason, it is the modality of choice in the study of conductive hearing loss (CHL). However, not every patient complaining of CHL requires a CT study. In fact, established indications encompass complex conditions, such as the complications of acute and chronic otomastoiditis, the postoperative ear in chronic otomastoiditis or in the localization of prosthetic devices, and the assessment of congenital or vascular anomalies. Particularly, the precise extent of bone erosion associated with cholesteatoma is correctly demonstrated by high resolution CT. Conversely, although fistulization through the tegmen tympani or the posterior wall of temporal bone is usually detectable by CT, the actual involvement of meninges and veins are better assessed by magnetic resonance (MR). MR is also indicated when complicated inflammatory lesions are suspected to extend into the inner ear or towards the sigmoid sinus or jugular vein. Neoplasms arising from or extending into the middle ear require the use of both techniques as their combined data provide essential information. Most important data for surgical planning concern the destruction of thin bony structures and the relationships of the lesion with the dura and surrounding vessels. DSA and interventional vascular techniques maintain an essential role in the presurgical work-up and embolization of paragangliomas extended into the middle ear. © 2001 Published by Elsevier Science Ireland Ltd.

Keywords: Computed tomography; Conductive hearing loss; Magnetic resonance

1. Introduction

Most middle ear lesions cause the impairment of one or more elements of the mechanical chain that transmits sounds—from the eardrum to the oval window. As computed tomography (CT) is an excellent technique for demonstrating even small abnormalities of the thin and complex bony structures of the middle ear, it is the modality of choice in the study of conductive hearing loss [1].

Mixed hearing loss—both conductive and sensorineural—requires a combination of CT and magnetic resonance (MR), as the latter is superior for the analysis of the inner ear content.

MR is also useful in some complex conditions affecting the middle ear, such as cholesteatomas and the postoperative ear, when the precise relationships with the dura and intracranial content have to be assessed.

Acute and life threatening complications of inflammatory lesions can be managed by both techniques, MR being more useful as it replaces digital subtraction angiography (DSA) in the evaluation of sinus and jugular vein involvement. DSA and interventional vascular techniques maintain an essential role in the presurgical work-up and embolization of paragangliomas extended into the middle ear.

2. Imaging technique

CT should be performed in both the axial and coronal planes. In the first case the patient lies in the supine position and scans are oriented parallel to the orbitomeatal line. Prone positioning with hyperextension of the neck is necessary for coronal acquisition. The imag-

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ing plane is oriented perpendicularly—as much as possible—to the orbitomeatal line. Contiguous slices (1mm thickness) are acquired using high-resolution bone algorithm. Iodine contrast administration is not necessary in the majority of cases as CT study focuses on bony structures and airspaces. In some circumstances (such as the assessment of complications of acute otomastoiditis or when vascularised lesions of the middle ear are suspected), injection of 100–120 ml contrast agent is recommended.

MR study requires the use of the head coil; images are oriented parallel (axial plane) or perpendicular (coronal plane) to the hard palate. Turbo T2 sequence (TR 4000, TE 102, matrix size 242 × 512, slice thickness 2–3 mm, acq. time 3 min) is performed in both the axial and coronal plane, followed by a T1 sequence (TR 500, TE 15, matrix size 192 × 512, slice thickness 2–3 mm, acq. time 3 min 15 s) on the axial plane. After the administration of Gd-DTPA (0.2 mmol/kg), T1 sequences are obtained on both the axial and the coronal plane. The sagittal plane may be occasionally indicated to assess the third portion of the facial nerve. An additional MRA sequence, obtained either by the TOF technique or with bolus administration of Gd-DTPA, can be acquired in particular situations (suspected sinus thrombosis, vascular anomaly or variant).

3. Acute otitis media (AOM) and mastoditis

AOM is most commonly caused by bacterial invasion of the middle ear through the Eustachian tube. This entity prevails in the pediatric population; *Streptococcus pneumoniae*, *Moraxella catarralis* and *Haemophilus influenzae* being the most frequent agents. In the majority of cases the diagnosis does not require imaging, and the lesion is cured with antibiotics [2–4].

Conversely, failure to control the mucoperiosteal lesion leads to a bony infection; the osseous framework of mastoid cells being frequently involved. This complication may potentially be of serious consequences.

In fact, although the infectious process is often limited to the temporal bone itself (**coalescent mastoiditis**, **labyrinthitis**, and **petrous apicitis**), it may spread outside (**subperiosteal abscess**) or extend intracranially (**dural sinus occlusive disease**, **meningitis** and **brain abscess**). As a consequence, in AOM and mastoiditis, imaging is indicated when clinical findings suggest a complication [5].

Clinical presentation of AOM is characterized by otalgia, fever and, in infants, sleeplessness and irritability [2,6]. Adenoid tonsil hypertrophy has been widely described in literature as a predisposing factor. Two main mechanisms have been depicted: obstruction of the Eustachian tube by the enlarged lymphatic tissue and direct spread of bacteria from the tonsil, itself a frequent focus of infection because it acts as a filter for inhaled agents [6–8].

Erythema and edema of the tympanic membrane are the earliest findings at otoscopy; they usually correspond to similar modifications of the mucosa of the middle ear.

When fluid collections (serous, mucoid or purulent) are retained within the middle ear cavity, additional otoscopic findings are the opacification and bulging of the eardrum, which may later drain externally, spontaneously.

In AOM, CT reveals that the middle ear cavity and mastoid cells are opacified, very often filled by fluid or debris with nonspecific characteristics; air fluid levels may be observed too. In uncomplicated AOM, the ossicular chain is preserved as well as the trabeculae and the cortical bone of the mastoid [9,10].

These bony structures are poorly demonstrated at MR. Consequently, in AOM and mastoiditis no more than middle ear fluid retention is correctly depicted by MR. Quite frequently this fluid shows variable signal intensities. Retained secretions usually appear hyperintense on T2 and hypointense on T1. However, the progressive dehydration of the fluid—and the relative increase in protein concentration—may lead to an increase of T1 signal combined with a decrease of T2 signal, particularly in chronic conditions. The absence of enhancement after Gd-DTPA administration rules out the presence of granulation tissue, which may be observed in chronic otomastoiditis.

Failure of antibiotic therapy to control the infection may lead to a progression from a mucoperiosteal disease to bone disease. Actually, this represents the turning point from a common AOM to a wide spectrum of complications. Although these complications all share the same starting point—bone infection—they may follow a more acute and aggressive course (for example coalescent mastoiditis) or a more subclinical progression (latent or masked non-suppurative mastoiditis) [11,12].

Coalescent mastoiditis is the progressive resorption and demineralization of the thin intercellular septa of the mastoid cells due to the mechanical compression of the edematous mucosa associated to local acidosis [7]. The result is destruction of the trabeculae and the development of an empyema. From a clinical perspective this complication is suspected in the presence of abundant ear discharge, pain and mastoid tenderness.

Further progression of the infection is characterized by the decalcification of the cortical bone of the mastoid. When this occurs on the outer cortical surface the result is the development of a **subperiosteal abscess** that may extend towards the external auditory canal, spreading along the root of the zygomatic bone, or have a postauricolar location.

When the mastoid is eroded at the level of its tip, adjacent to posterior digastric belly insertion, pus collections may spread downwards along the path of the sternocleidomastoid muscle, anteriorly limited by both the posterior cervical fascia and the posterior lining of the pharyngobasilar fascia [4,7]. This condition is described as **Bezold's abscess**. It is fortunately quite rare but potentially life-threatening because the collections may spread as far as the mediastinum and, therefore, dictate surgical drainage. Clinical diagnosis may be difficult because, differently from subperiosteal abscess, Bezold's abscess is deeply seated and, therefore, usually not palpable [7,13].

Coalescence and, when bone resorption occurs, **petrous apicitis** may also complicate the anterior spread of the intratemporal infection into a pneumatized petrous apex. The clinical presentation, first described by Gradenigo, consists of deep facial pain along the course of the trigeminal nerve, abducens palsy and purulent ear discharge. This classic triad resulting from inflammation of the Gasserian ganglion and of the VI nerve close to Dorello's canal is not constant.

The medial extent of the infection causes the erosion of the inner cortical surface of the mastoid, and opens a communication with the middle or posterior cranial fossa. An **epidural abscess** occurs when the collection lies between the dura and the skull bones. The invasion of the dura may cause **subdural abscess**, **meningitis** or **brain abscess**. The agents most commonly involved are *Proteus*, *Pseudomonas* or *Staphylococcus* [4,7].

An erosion of the sigmoid sinus plate may lead to **dural sigmoid** or **lateral sinus occlusive disease**, an ominous complication characterized by fever, severe headache and sixth nerve palsy [14]. Venous thrombosis, may also occur close to an epidural abscess.

A low-grade bone infection without pus formation is the hallmark of the **masked-latent non-suppurative mastoiditis** occurring after an apparently well treated AOM. In this particular condition, bone infection is characterised by proliferative rather than erosive changes. The eardrum is frequently normal. The incidence of complications is high [11,12].

Imaging plays a pre-eminent role in the diagnosis of all the aggressive and sub-acute complications of AOM. In the antibiotic era all these conditions are quite infrequent — intracranial complications are reported in only 0.15% of cases — [13]. Anyway, CT or MR should be performed in every patient presenting signs

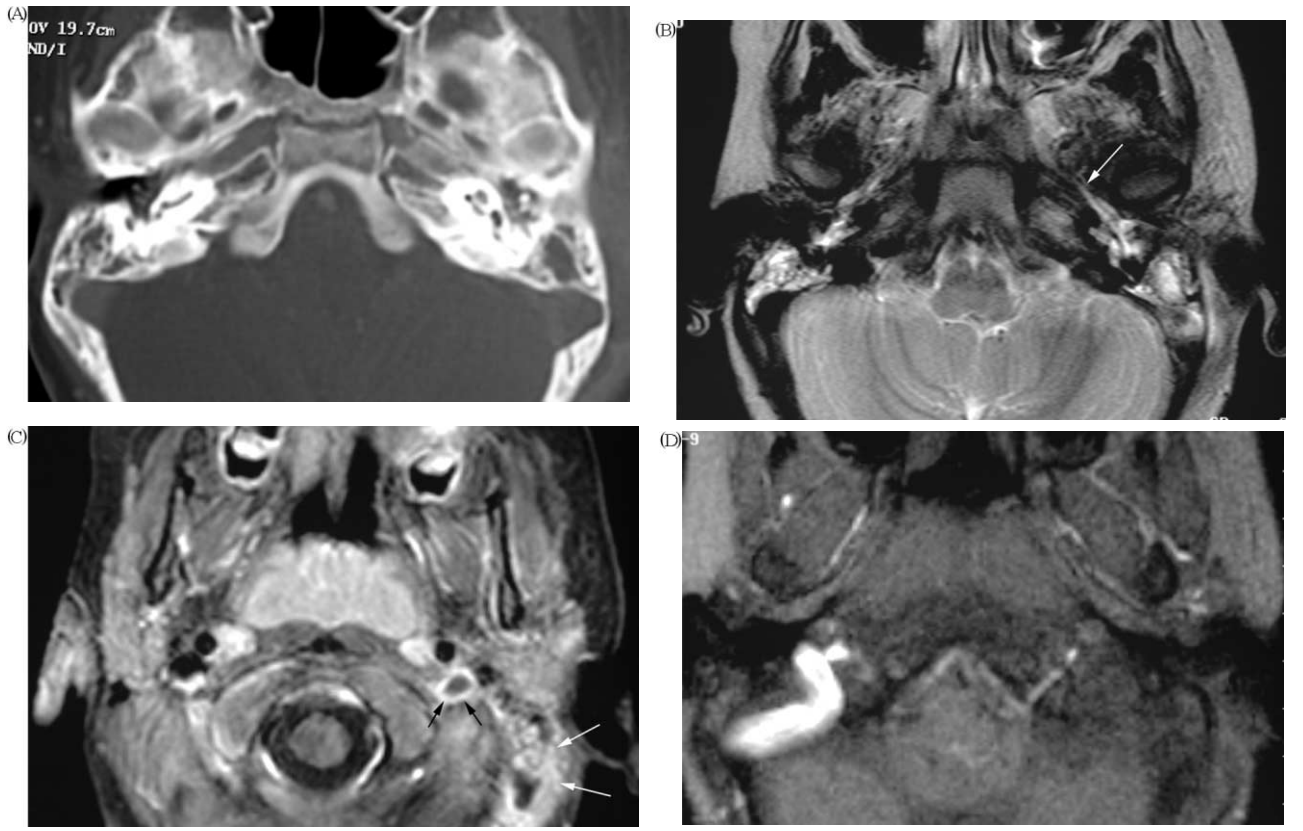

Fig. 1. (A–D) Complicated acute otomastoditis in a pediatric patient. CT shows bilateral opacification of the mastoid cells on the left side combined with resorption of intercellular walls and thickening of perimastoid soft tissues. MR T2 (B) demonstrates fluid retention within the mastoid, middle ear and along the course of the Eustachian tube (arrow), on both sides. A retroauricolar abscess (arrow) is detectable on Fat-Sat T1 after Gd-DTPA (C). Left jugular vein appears hypointense with enhancement of the vessel's wall (black arrow). MR venography with TOF technique (D) confirms the diagnosis of jugular vein thrombosis.

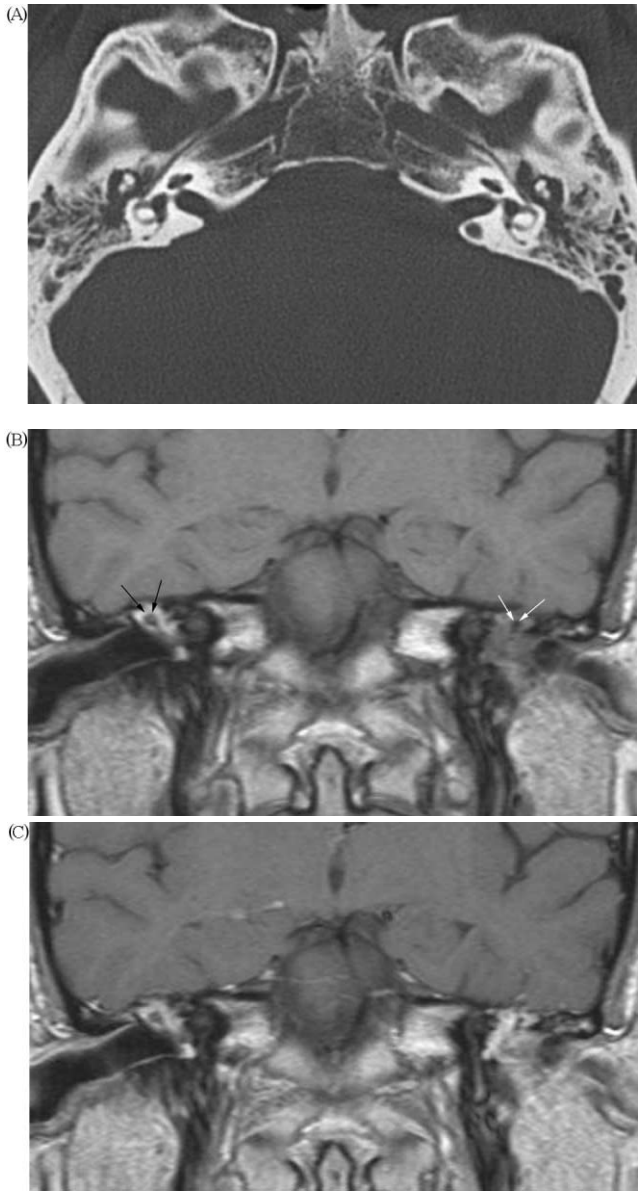

Fig. 2. (A–C) Wegener granulomatosis: anacusia on left side, conductive hearing loss on the right. CT (A) shows bilateral opacification within the middle ear and mastoid, no signs of bone erosion. MR T1 (B, before; C, after Gd-DTPA) shows enhancement of the retained material in the left middle ear: granulation tissue. On the right side an hyperintense signal fills the middle ear, consistent with dehydrated fluid collection. Part of the ossicular chain is demonstrated on both sides (arrows)

and symptoms of coalescence, during or after the apparent recovering of an AOM, to rule out major complications.

Coalescent mastoiditis is easily detected at CT. The diagnosis is obtained comparing the number, thickness and mineralization of mastoid intercellular trabeculae with the contralateral side, even though asimmetry is not uncommon [2].

CT and MR equally identify subperiosteal abscess (Fig. 1), Bezold's abscess and intracranial complications. Purulent collections are demonstrated by means of density or signal intensity. They are outlined by a thick, irregular and enhancing rim corresponding to the inflamed periosteum or dura (Fig. 1C).

The diagnosis of sigmoid or lateral sinus occlusive disease is sometimes a difficult task [15]. At CT, the absence of contrast enhancement within the sinus lumen is a sensitive but not highly specific sign. At MR the diagnosis is hampered by flow related phenomena: the presence of a signal void within the sinus excludes a thrombosis. On the other hand, slow venous flow may be responsible for a hyperintense T1 signal of the vessel, sometimes associated with Gd-DTPA enhancement. The final diagnosis requires an MRA study with TOF technique or after contrast administration [16] (Fig. 1D).

4. Chronic otomastoditis (COM)

It is generally due to an Eustachian tube dysfunction, even though a bacterial superinfection is possible. COM

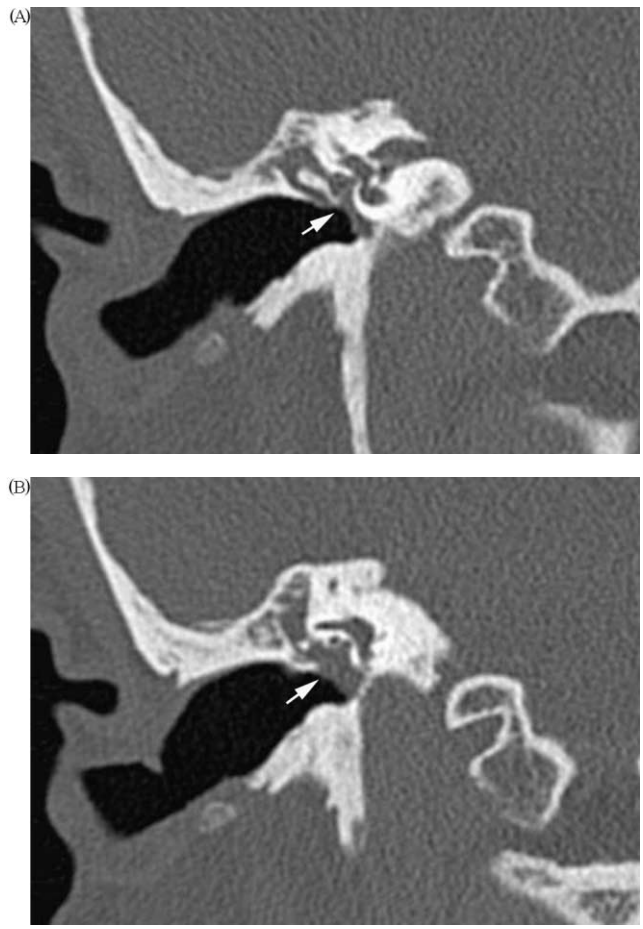

Fig. 3. (A and B) COM. Coronal CT demonstrates eardrum retraction (arrow) and opacification of both epi- and mesotympanum. The scutum and the ossicular chain are preserved: non-cholesteatomatous COM.

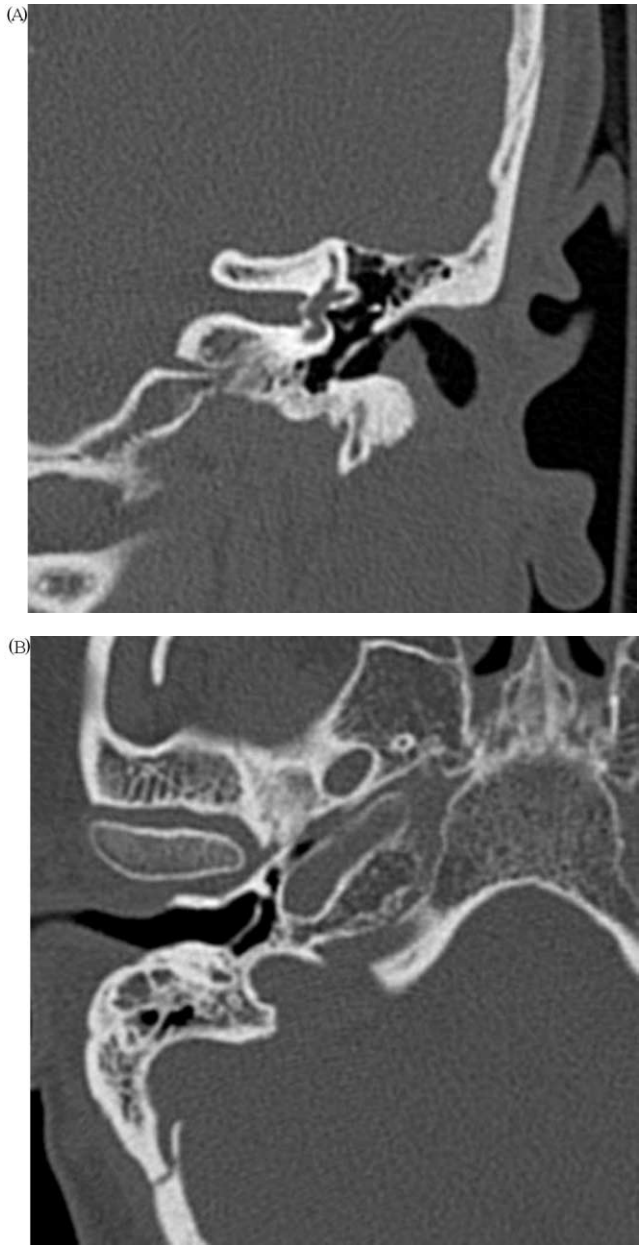

Fig. 4. (A and B) Tympanosclerosis, conductive hearing loss. Bilateral thickening of the tympanic membrane, it appears partially retracted. Irregular calcifications are observed on both sides.

encompasses a wide spectrum of conditions such as **middle ear effusion**, **accumulation of granulation tissue**, **cholesterol granuloma**, **tympanic membrane retraction**, **acquired cholesteatoma**, **ossicular erosion** or **fixation** [9,10].

Chronic otitis media is a rather common disease frequently associated with a limited pneumatization of the mastoid, even though the correlation between these two entities is not yet fully understood.

Two different patterns of disease are referred. An Eustachian tube dysfunction results in a COM mainly involving the mesotympanum (*tubotympanic form).* The *atticoantral form*, conversely, is limited to the epitympanum, being related to the chronic obstruction of the tympanic isthmi, i.e. the openings connecting attic and mesotympanum [17].

It has been suggested that the isolation of the attic and adjacent middle ear spaces results in negative pressure within these cavities. In this setting, retraction of the pars flaccida could occur, predisposing to the development of an acquired cholesteatoma [18].

Clinical presentation of COM includes recurrent episodes of AOM, otalgia with otorrhea and hearing loss. Vertigo and tinnitus are seldom referred. Various otoscopic findings are described, such as eardrum perforation or retraction, tympanosclerosis or middle ear mucosa proliferation. Audiologic examination may reveal conductive hearing loss.

The most common manifestations of COM are **effusion** and **accumulation of granulation tissue**, the latter being reported in up to 97% of cases [6]. Effusion is common as in AOM; anyway it also may be the presenting sign of a nasopharyngeal carcinoma. Careful inspection of the nasopharynx—normally included in the standard CT study of the temporal bone—is, therefore, recommended in every case of middle ear effusion, particularly when bilateral.

Cholesterol granuloma is a fluid collection containing cholesterol crystals, macrophages, red blood cells and debris, normally not associated with destructive changes of bone structures. It occurs in up to 14% of COM [6,19]. This lesion is more frequently detected within the petrous apex in patients with no history of recurrent otitis and abnormal mastoid pneumatization. It is referred to as **giant cholesterol cyst** and may cause

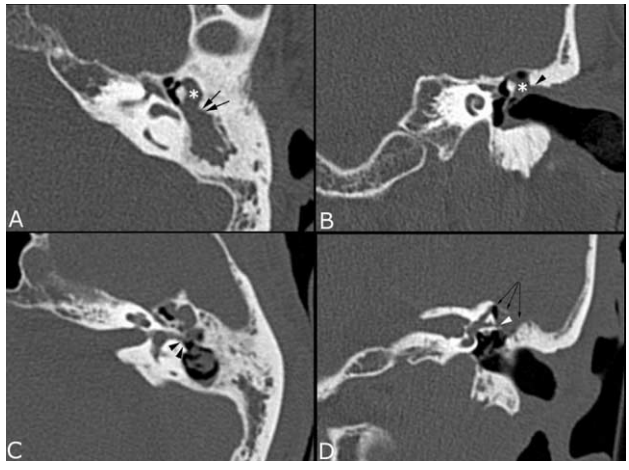

Fig. 5. (A–D) Cholesteatomatous COM. In the first case (A-B) a soft tissue mass extends from Prussak's space (asterisk) into the epitympanum and the mastoid through an enlarged aditus ad antrum (arrow). Erosion of the scutum (arrowhead) can be appreciated. The ossicular chain is medially displaced. In the second case (C and D) ossicular chain is undetectable, totally eroded. Lateral semicircular canal fistulization is detected on both coronal and axial plane (arrowheads). An interruption of the tegmen tympani is identified on coronal image (arrows).

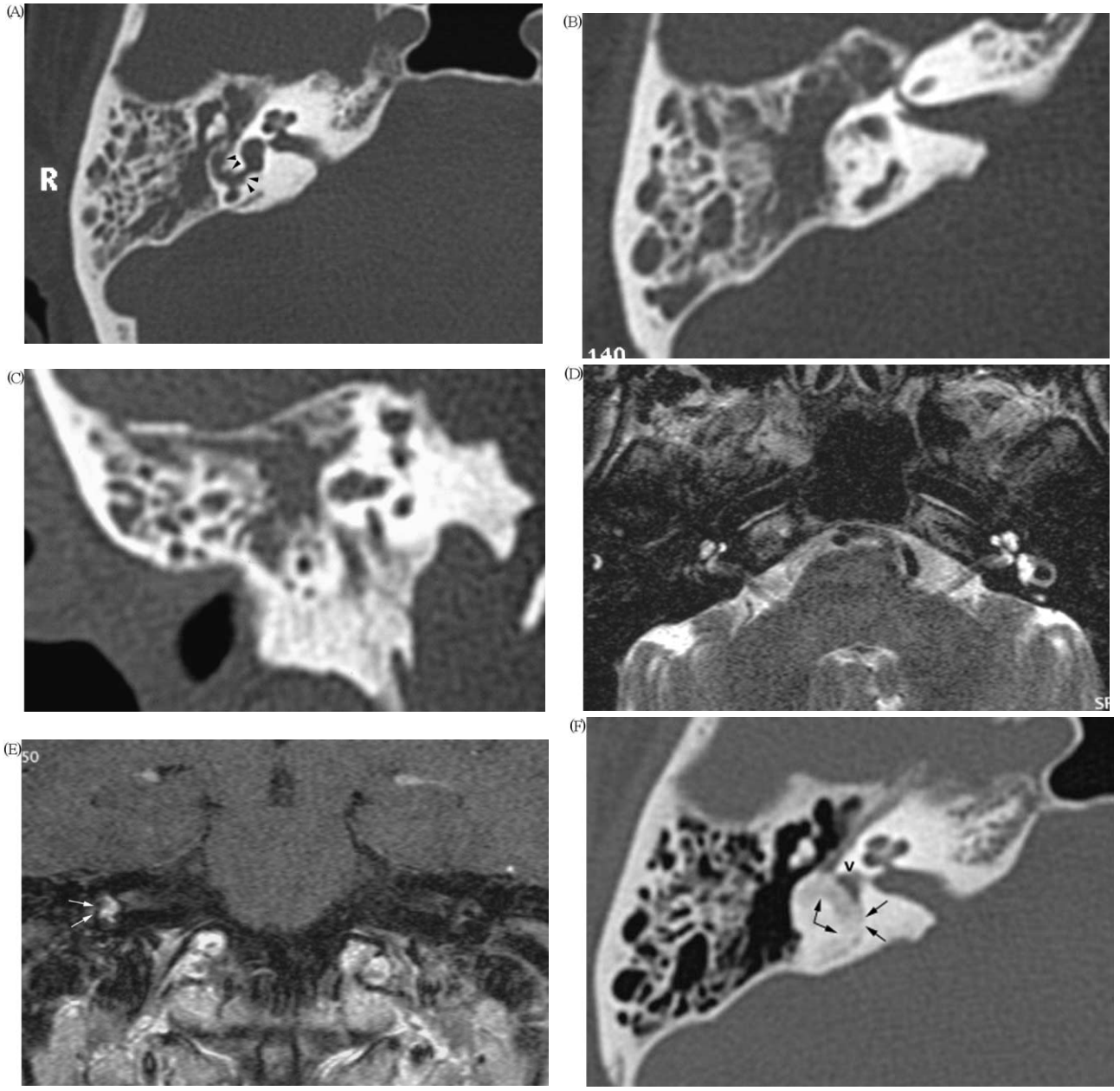

Fig. 6. (A–F) COM, right anacusia and vertigo. Three months before, the patient had an acute external and middle ear otomastoiditis treated with antibiotics. (A) CT shows opacification of right middle ear and mastoid with irregular resorption of the bone adjacent to the lateral semicircular canal (arrowheads). Its walls appear unsharp, the diameter is abnormally enlarged (A–C). Six months later MR demonstrates the abscence of the normal hyperintense signal on T2 (D) combined with enhancement (E) of the vestibule and semicircular canals on T1 (arrows). A CT examination performed in the same period (F) demonstrates marked enlargement of the semicircular canals, replaced by mineralized content (arrows): labyrinthine ossification. Part of the vestibule is still detectable (V).

bone erosions, particularly at the level of the carotid canal [20–24].

Tympanic membrane retractions may occur either in the pars tensa or in the pars flaccida. Retraction pockets in the pars flaccida should be carefully inspected, as they may be the sites of origin of acquired cholesteatomas. In the pars tensa different degrees of distortion are described, ranging from bare retraction to incus adhesion, middle ear atelectasis and adhesive otitis media.

Ossicular changes are found in up to 92% of cases [6]. **Ossicular fixation** may be the ending point of a COM. It is related either to deposition of fibrous tissue (chronic adhesive otitis media), hyalinization of collagen (tympanosclerosis) or new bone formation (fibro-osseous sclerosis). A conductive hearing loss is a result, depending on the degree of loss of the elastic properties of the eardrum, ankylosis of the malleus head or of the stapes, dysfunction of tensor tympani or stapedial muscles. Ossicular erosions are also found in the absence of

a cholesteatoma, particularly at the level of the long and lenticular processes of the incus.

Acquired cholesteatoma is a cystic lesion lined by a keratinizing squamous epithelium filled with desquamation debris arising from retraction pockets either in the pars flaccida or in the pars tensa of the eardrum, growing into the middle ear cleft [9,10,25,26]. Pars flaccida cholesteatoma arises in the Prussak's space (superior recess of the tympanic membrane), and has a typical pattern of growth consisting of early erosion of the scutum, medial displacement of the ossicular chain, and invasion of the mastoid through the aditus ad antrum [25]. Facial recess and sinus tympani are the most common sites of origin of pars tensa cholesteatoma. This lesion tends to grow either in the direction of the oval and round window—rarely causing cochlear fistulas—or towards the mastoid through the posterior tympanic isthmus and the posterior incudal space. Differently from Prussak's space lesions, pars tensa cholesteatoma generally displaces the ossicular chain laterally.

Cholesteatoma seldom arises from the anterior recess of the tympanic membrane (anterior epitympanic cholesteatoma). It may obliterate the supratubal recess and extend into the middle ear through the anterior tympanic isthmus. Most cholesteatomas with intracranial extension originally arise from this site [25].

Regardless of the sites of origin, all cholesteatomas share some common features, such as the erosion of the ossicular chain and/or scutum and/or mastoid bone, and the destruction of Körner's septum [9,10].

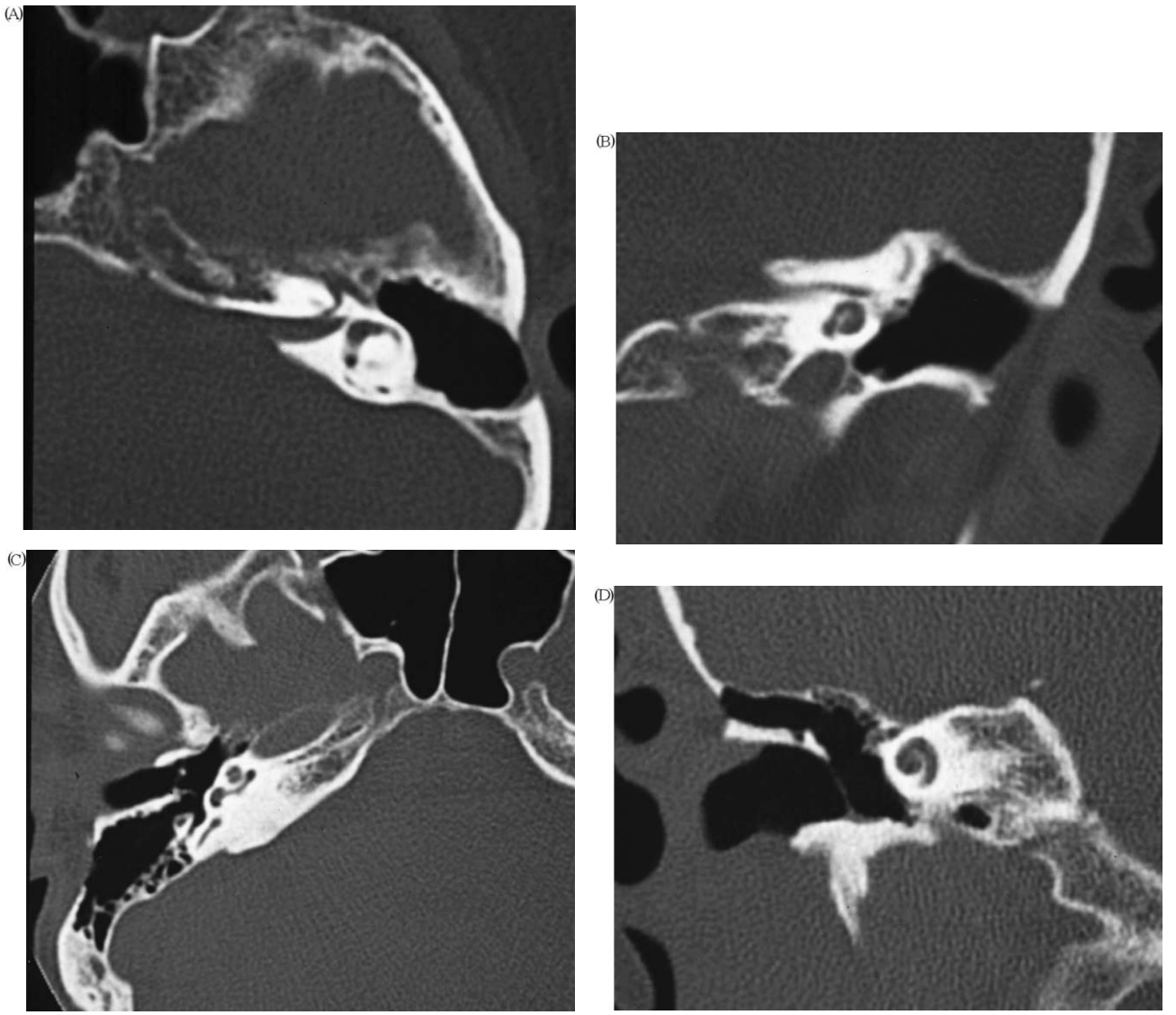

Fig. 7. Postoperative middle ear. (A and B) Radical mastoidectomy for COM. Normal CT findings encompass the absence of the ossicular chain, mastoid cells, lateral middle ear wall and eardrum. Normal air content within the postsurgical cavity is appreciated. (C and D). Closed tympanoplasty for COM. A defect on the outer aspect of the mastoid suggests partial mastoidectomy. The ossicular chain has not yet been reconstructed. Part of the residual malleus is detected on the axial plane (arrow). The tympanic membrane reaches the scutum showing a quite normal appearance. Normal air content within the middle ear cleft.

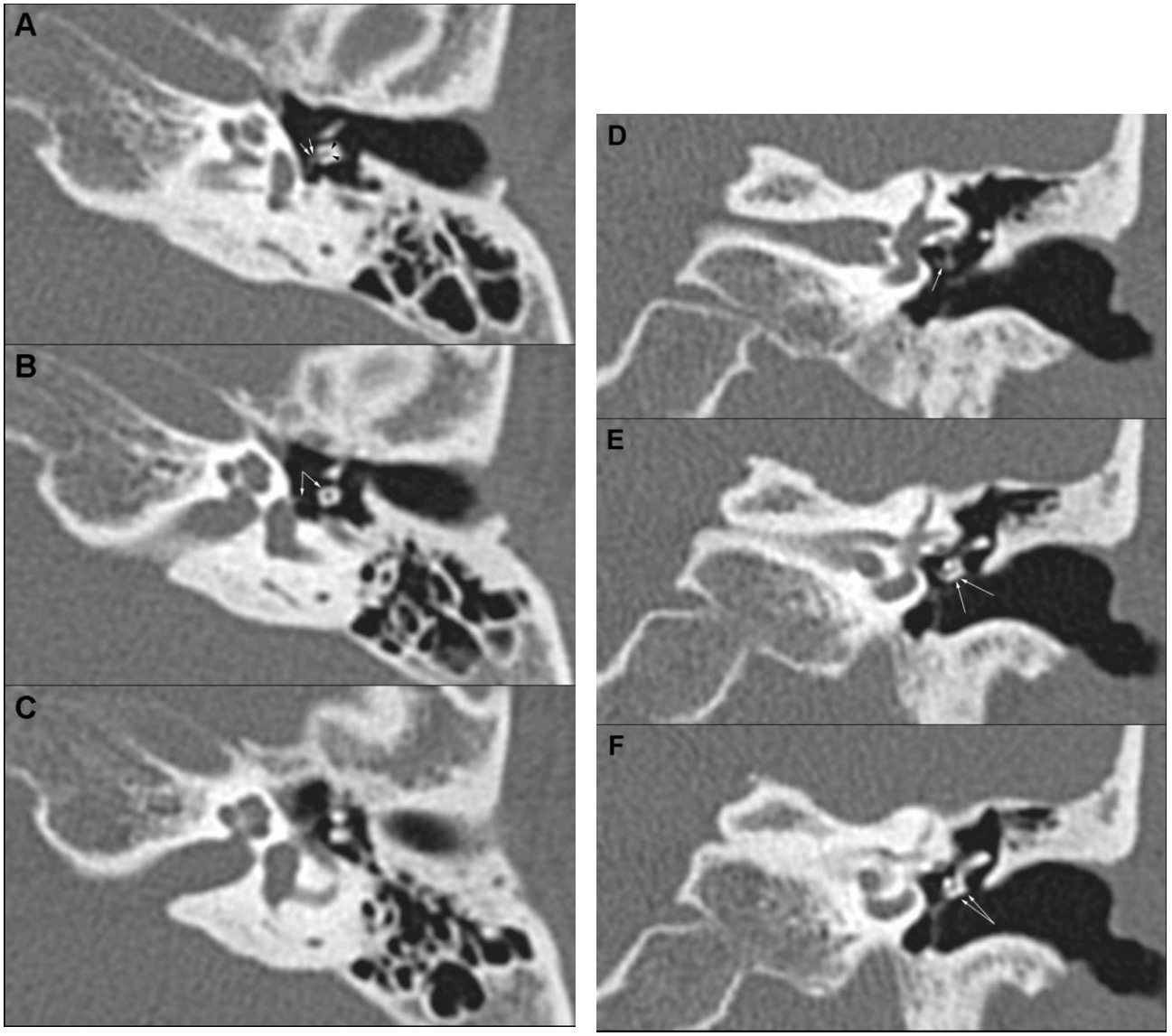

Fig. 8. Tympanoplasty for COM, replacement of the incudo–stapedial joint by the Applebaum prosthesis. (A–C) On the axial images the capitulum of the stapes (A) appears precisely located within the prosthetic notch (arrowheads). Crura of the stapes are identified (arrows). (B) The residual stapes and the upper portion of the prosthesis are demonstrated (arrows). (C) Body of the incus and head of malleus are shown. (D–F) On coronal images the stapes (arrow on A), the Applebaum prosthesis (arrows on B) and the incudo–prosthetic neo-joint (C) are detected.

The erosion of the medial wall of the middle ear leads to the onset of a fistula with the inner ear—the lateral semicircular canal being eroded in about 66% of cases—[27], and/or facial nerve palsy, particularly when the anterior epitympanic recess is involved. Progressive destruction of the attic results in intracranial extension when the tegmen tympani has been destructed.

Meningitis or dural sinus thrombosis represent major complications [19,24,28]. In some cases the cholesteatomatous mass may spontaneously drain externally, leaving just a thin membrane covering a wide cavity that resembles the postmastoidectomy appearance of the middle ear; hence this condition is referred to as automastoidectomy.

The diagnosis of cholesteatoma is usually accomplished by otoscopy. Nevertheless, imaging is mandatory to assess the size of the lesion, and its relationships with ossicular chain, tegmen tympani and facial nerve [28].

Imaging plays a major role in the evaluation of the various COM manifestations, CT being, in most of cases, the modality of choice [29].

The main reason is that MR lacks detail on thin bony structures, therefore its role is limited. However, the excellent contrast resolution of this technique provides valuable findings to differentiate a simple middle ear effusion from granulation tissue accumulation or cholesterol granuloma (Fig. 2). In fact, these entities are often indistinguishable at CT, whereas they all are

hyperintense on T2 sequences. Plain T1 allows us to discriminate effusion from cholesterol granuloma, as the latter appears spontaneously hyperintense (blood catabolites and lipidic content) [19]. After the administration of Gd-DTPA a bright enhancement is exhibited only by granulation tissue. Of course, a giant cholesterol cyst of the petrous apex can be suspected also at CT because of its location, but the MR pattern—similar to cholesterol granuloma—better addresses the diagnosis [24].

Tympanic membrane retractions are easily detected with CT (Fig. 3), particularly when the middle ear is free of debris. In these patients the eardrum is often thickened [30].

Postinflammatory ossicular chain fixation includes a wide spectrum of CT changes. Fibrous tissue fixation of the ossicular chain (chronic adhesive otitis media) is demonstrated when soft tissue debris encases one or more ossicles. The detection of multiple punctate calcifications within this tissue—associated with focal/diffuse thickening of the eardrum—suggests tympanosclerosis (Fig. 4). Additional findings are the presence of tendon calcifications, malleus manubrium fixation or new bone formation, mainly in the epitympanum [31].

Since tiny osseous structures—such as the lenticular process of the incus—are more frequently eroded, the absence of debris or fluid retention in the middle ear cavity improves the detection rate of ossicular abnormalities.

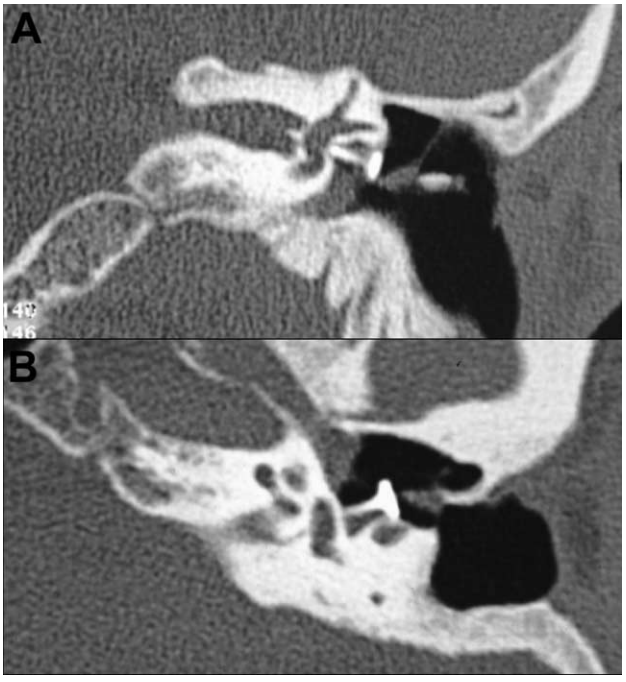

Fig. 9. Closed tympanoplasty with TORP (total ossicular reconstructive prosthesis) for a COM complicated by a cholesteatoma. Partial mastoidectomy with normal air content. A stainless steel piston results correctly placed within the oval window. Soft tissue lesions are located along the inner surface of the eardrum and within the hypotympanum suggesting a recurrent COM.

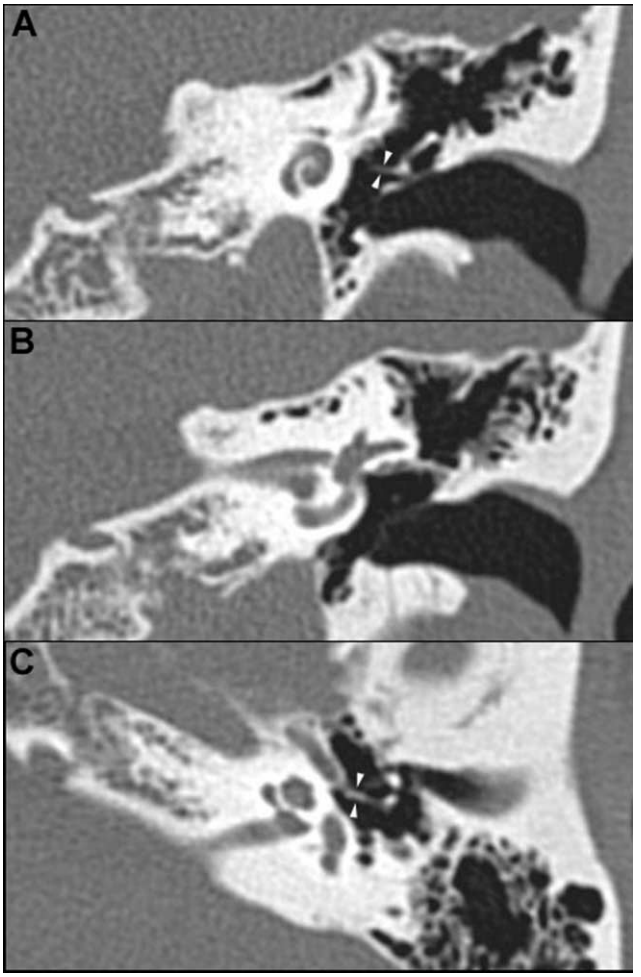

Fig. 10. Misplaced PORP. Relapsing conductive hearing loss after PORP (partial ossicular reconstructive prosthesis) for COM. The sinthetic PORP appears misplaced. On coronal (A) and axial (C) planes its inner tip (arrows) results anterior to the empty oval window (B).

CT is particularly effective in the evaluation of cholesteatoma: even though the appearance is quite nonspecific—a homogeneous, nondependent soft tissue mass with lobulated margins [24], some essential clues to the diagnosis may be observed. Scutum erosion, described in up to 90% of cases, destruction of the long process of incus (60–90%), enlargement of the aditus ad antrum with erosion of mastoid intercellular septa (30–50%) are the most common findings [19]. Labyrinthine fistula occurs in 4–25% of cases; it is correctly demonstrated by CT both in the axial and coronal planes [19] (Fig. 5).

MR study may be helpful when some complications of cholesteatoma are suspected at CT; for example an interruption of the tegmen tympani requires MR to rule out meningocele or meningoencephalocele. The superior contrast resolution of this technique, permits the

differentiation of cholesteatoma from dura and brain parenchyma. MR is also indicated when the patient complains of facial nerve impairment, vertigo or sensorineural hearing loss. Even though CT better demonstrates the bony walls of the facial nerve canal and of the labyrinth, only MR shows inflammatory changes of the nerve itself (thickening, enhancement) and/or enhancement of the membranous labyrinth content. CT better demonstrates the late ossific stage of labyrinthitis (Fig. 6). The extent of the cholesteatoma into the petrous apex is an additional indication for a MR study. A soft tissue mass in this location opens up a wide spectrum of differential diagnoses; the absence of Gd-DTPA enhancement is quite a typical finding of cholesteatoma [9,10,24].

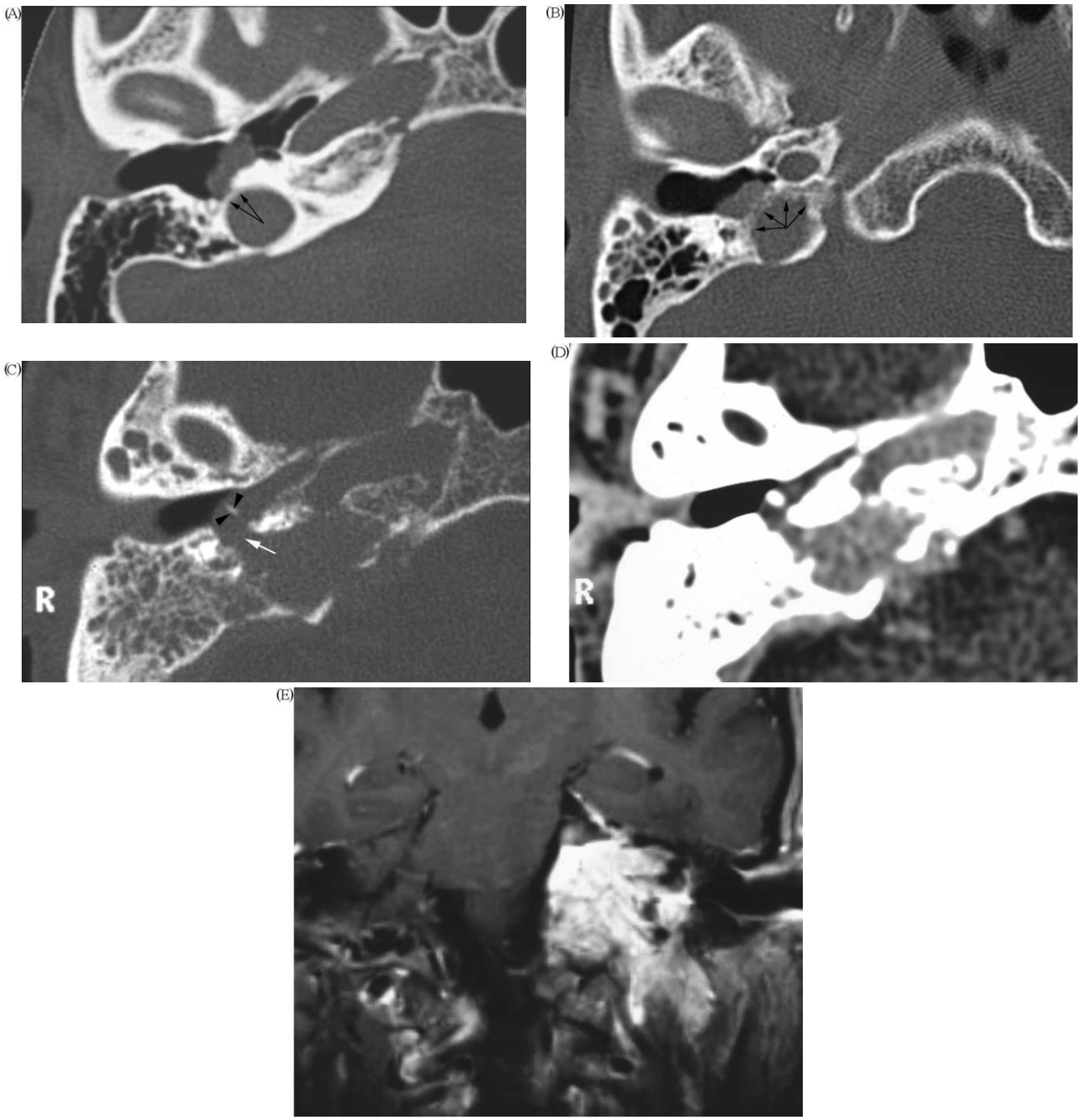

Fig. 11. (A–E). Paragangliomas. (A) CT shows a soft tissue mass arising from the inferior mesotympanum. The adjacent lateral bony plate of the jugular canal is normal (arrows), suggesting a tympanic paraganglioma. (B) In a different patient, a similar lesion is detected at the same level. In this case, the moth eaten erosion of the bony walls of the jugular canal (arrows) is consistent with a jugulo–tympanic paraganglioma. (C and D) A larger jugulo–tympanic paraganglioma destroys the jugular spine. Through the erosion of the antero-lateral wall of the jugular foramen, the lesion invades the middle ear, encasing the ossicular chain. After contrast administration intense enhancement is observed. (E) Jugulo–tympanic paraganglioma, MR T1 after Gd-DTPA. The lesion demonstrates marked enhancement.

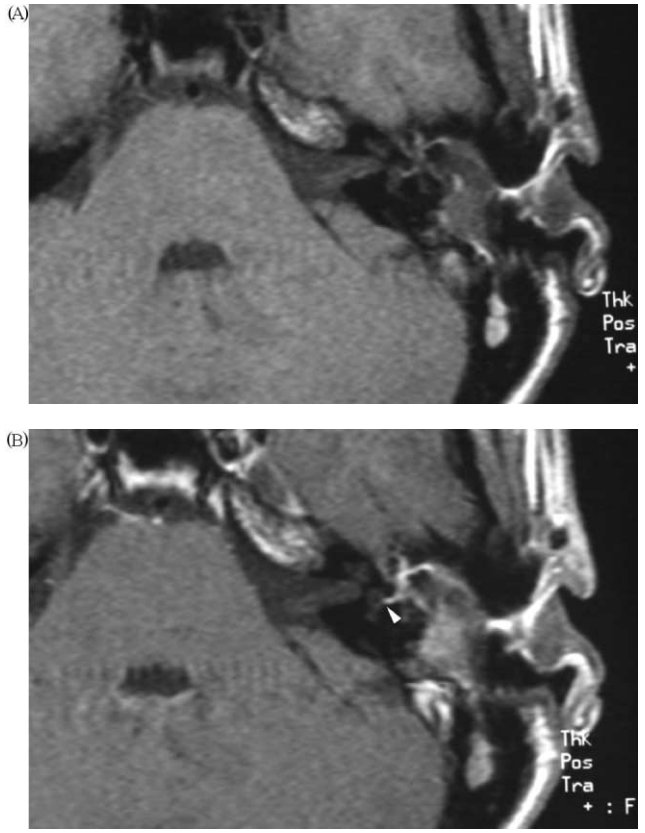

Fig. 12. (A and B) Middle ear carcinoid, MR T1 before (A) and after Gd-DTPA (B). A small, enhancing soft tissue mass fills the middle ear cleft. No enhancement is detected in the inner ear even if the inflamed mucosa reaches the oval window (arrowhead).

5. Postoperative middle ear

When surgery is performed for a chronic otomastoiditis the width of the resection is extremely variable according to the extent of the disease. Records of previous surgical operations are, therefore, indispensable to correctly assess the normal postoperative appearance. Generally, the rationale of surgery is to remove pathologic tissue while preserving the ossicular chain and the integrity of the external auditory canal [9]. Even though many different surgical approaches are described, a schematic classification may be summarized as follows:

1. *radical techniques*, (also indicated as open cavity procedures), namely radical mastoidectomy including the modified approach;
2. *conservative techniques* (closed cavity procedures), tympanoplasty (with different variants);
3. *reconstructive techniques*, ossiculoplasty;
4. *substitutive techniques*, stapedotomy and stapedectomy.

Signs and symptoms recommending a postoperative imaging study are related to the persistence of hearing loss (indicating functional failure), the onset of otorrhea (suggesting recurrence or insufficient ventilation of the cavity) and vertigo (suggesting the presence of a perilymphatic fistula).

Key points to assess by using CT are the surgical cavity (aeration, presence of residual debris, edges of the cavity), the ossicular chain, the bony structures of the middle ear and mastoid (to rule out defects of the tegmen, of the otic capsule or of the sigmoid plate) and the VII nerve canal (both tympanic and mastoid tracts) [32] (Fig. 7).

CT must be considered the technique of choice. MR is indicated only in selected cases, particularly when a meningo- or meningoencephalocele are suspected on a CT baseline study.

Ossicular chain destruction or disruption due to COM, cholesteatoma, or congenital malformations may be reconstructed by prosthetic devices. According to the extent of the damage, partial or total replacement of the chain is required. CT enables an excellent demonstration of the reconstructed conductive chain (Fig. 8)

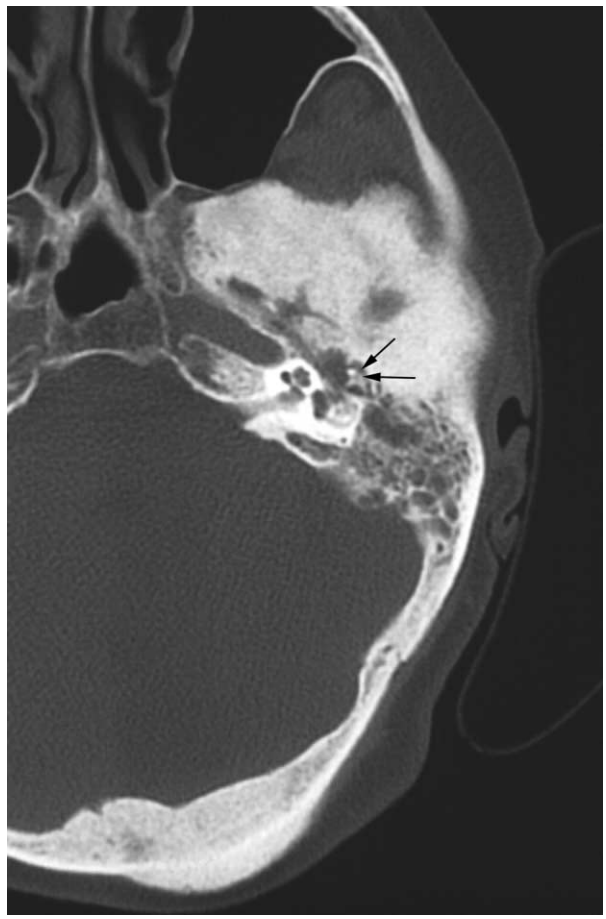

Fig. 13. Meningioma and fibrous dysplasia. Abnormal but quite regular enlargement of the occipital bone is consistent with fibrous dysplasia. Conversely, the petrous apex and the greater wing of the sphenoid bone show replacement of the normal shape and marked sclerosis due to a meningioma. Narrowing of the Eustachian tube and impairment of its function result in a COM. The ossicular chain is preserved (arrows)

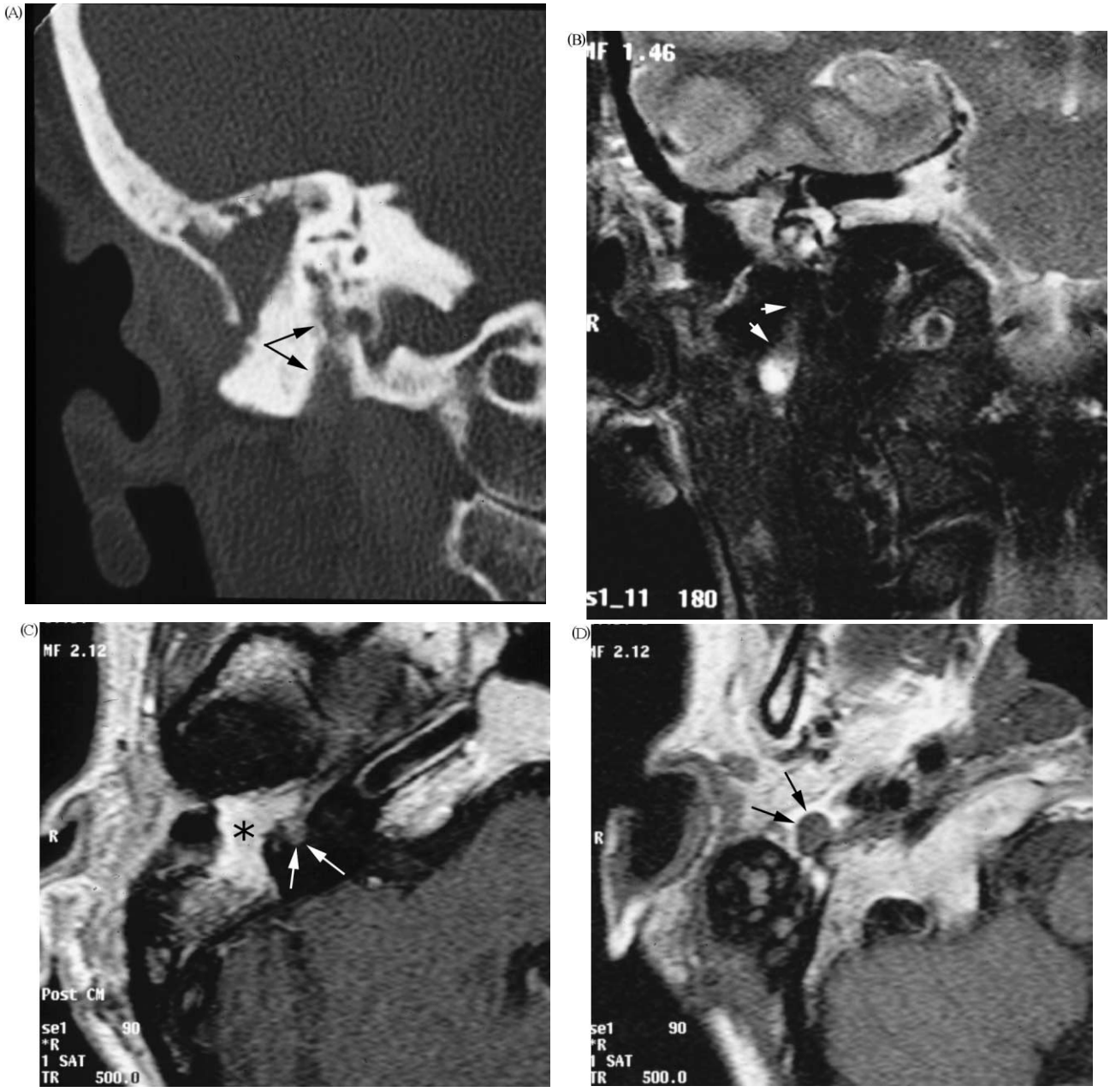

Fig. 14. (A–D) Adenoidcystic carcinoma of the parotid gland. The patient had been operated on for a misdiagnosed chronic otitis media a few months before. Enlargement of the stylomastoid foramen and of the third portion of facial nerve (arrows) with unsharp margins is demonstrated at CT (A). MR obtained 2 months after CT shows hyperintensity (B) and enhancement (C and D) of an enlarged facial nerve (arrows) suggesting perineural spread. Enhancing granulation tissue (asterisk) is demonstrated by MR (C) in the middle ear cleft.

[33]. Malfunctioning of the ossicular prosthesis results in a worsening of conductive hearing loss. Preoperative assessment by CT is indicated as revision surgery is associated with an increased risk of sensorineural hearing loss and an unsatisfactory rate of success [34]. Among the causes of prosthetic failures that can be demonstrated by CT are included recurrent COM and cholesteatoma, subluxation, dislocation, and extrusion of the device (Fig. 9 and Fig. 10). The development of granulation tissue or fibrosis can be suggested when soft tissue is detected at the oval window notch, within 4–6 months from surgery [35]. Complications, such as perilymphatic fistula with persistent or worsening vertigo, tinnitus, may be difficult to detect at CT, even though it may be suspected by the presence of pneumolabyrinth or the onset of middle ear effusion [35].

6. Congenital malformations—vascular anomalies

Congenital malformations may be observed in several syndromes such as Klippel–Feil syndrome, Wilder-

vanck syndrome, Madelung's osteocondrosis, Apert's syndrome, and achondroplasia. They are characterized by various classes of derangement of the normal anatomy of the ossicular chain that can be variably associated to external or inner ear anomalies [9]. Middle ear malformations can be summarized into five categories: (1) isolated stapedial ankylosis; (2) stapedial ankylosis with other ossicular anomalies; (3) ossicular anomalies with a mobile stapes; (4) oval/round dysplasia; and (5) incudostapedial disconnection. At clinical examination they are characterized by hearing loss (often bilateral) that can be conductive-most frequently—sensorineural or mixed. The goal of imaging in all these conditions is to demonstrate the anatomy of the middle ear, in particular the presence, morphology and position of the tympanic membrane and of the ossicular chain [9,10].

Three main vascular anomalies are described in the literature as possible causes of a pulsatile tinnitus combined with a tympanic 'vascular' membrane: persistent stapedial artery, aberrant internal carotid artery and jugular bulb anomalies.

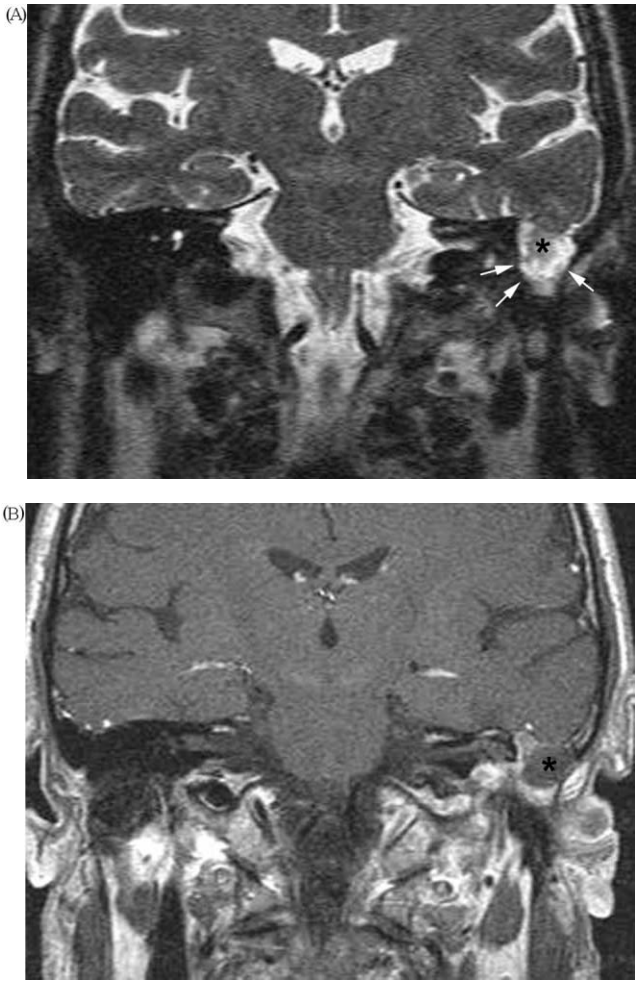

Fig. 15. (A and B) Meningoencephalocele. MR T2 (A) demonstrates the presence of a soft tissue mass (asterisk) in the epitympanum surrounded by a thin hyperintense layer (arrows), herniating through a defect of the tegmen. Both on T2 (A) and T1 (B) the signal of the mass is similar to brain parenchyma. Meningoencephalocele was confirmed at surgery.

CT findings of persistent stapedial artery [9,10,36,37] consist of the absence of the foramen spinosum (the middle meningeal artery arises from the stapedial artery itself) and the presence of a soft tissue mass located close to the proximal tympanic portion of the facial nerve. Actually, the persistent stapedial artery enters the hypotympanum and runs for a short segment along the Jacobson's and facial nerve canals.

An aberrant internal carotid artery (ICA) is due to the anastomosis between the (abnormally enlarged) carotico–tympanic and inferior tympanic arteries during the embriogenesis. Generally, the patient complains of a pulsatile tinnitus. Otoscopy shows a vascular retrotympanic mass mimicking a glomus tympanicum. MR angiography enables the diagnosis, although CT is generally exhaustive. The aberrant ICA is detected as a soft tissue mass entering the middle ear cleft just posterior to the vertical segment of the ICA, surrounding the promontory and joining the horizontal tract of the ICA [9,10].

Similar clinical and otoscopic findings may be observed in the presence of an asymmetrically large jugular bulb, high riding jugular bulb (extending upwards to the level of the floor of internal auditory canal) and jugular bulb diverticulum. CT precisely outlines the bony walls of the jugular foramen and the jugular spine, normally preserved by the asymmetrically large jugular bulb. A dehiscence, in particular at the level of the sigmoid plate, may be observed in the presence of a high riding bulb. At MR these variants may simulate a disease: they may show hyperintense T1 signal and enhancement due to flow related phenomena. MR venography, performed with TOF technique or contrast administration, rules out the differential diagnosis [9,10].

7. Neoplastic and pseudoneoplastic lesions

Primary neoplasms of the middle ear are, on the whole, rare. This region is more often involved by neoplasms arising elsewhere in the temporal bone. **Glomus tympanicum**, **congenital cholesteatoma**, **schwannoma** and, in the pediatric population, **rhabdomyosarcoma** are the most frequent lesions.

A variety of other lesions such as metastases, Langerhans cell histiocytosis, giant cell tumor and squamous cell carcinoma are only seldom observed. Meningocele and meningoencephalocele are typical pseudoneoplastic lesions.

Signs and symptoms of a neoplastic or pseudoneoplastic lesion of the middle ear are quite nonspecific; they include conductive (or mixed) hearing loss, (pul-

satile) tinnitus, vertigo and less commonly earache or discharge. From a clinical point of view the diagnostic task is represented by a mass detected by otoscopy beyond a preserved eardrum. The identification of a *vascular tympanic membrane* (blue or reddish coloration) or of a *glue ear* is the first clue to the possible presence of a vascular lesion. Pulsation of the mass is rarely appreciated.

Glomus tympanicum is the most common tumor of the middle ear and the most frequent cause of pulsatile tinnitus. It has a peak of incidence around the 5th and 6th decade with predominance for the female gender. Most commonly it arises from paraganglia remnants scattered along the course of Jacobson's nerve and, in the majority of cases, it is located at the level of the mucosa overlying the promontory. Much more frequent is the paraganglioma arising within the jugular foramen, from the paraganglia either surrounding the jugular bulb or located along Jacobson's or Arnold's nerves. Because of its common propensity to grow through the inferior wall of the tympanic cavity and to invade the mesotympanum it is referred to as **glomus jugulotympanicum**. Differential diagnosis between the two entities is impossible at otoscopy. Imaging is therefore mandatory for proper surgical planning [38].

CT findings of glomus tympanicum consist of a small enhancing mass centered at the level of the cochlear promontory and confined to the middle ear. Invasion of the anterior epitympanic recess, Eustachian tube or mastoid is possible, while ossicular destruction is not typical. At MR, the lesion appears hyperintense on T2, intermediate with bright contrast enhancement on T1. The characteristic salt and pepper pattern (due to the serpentine and punctate flow voids of vessels within the lesion) is not appreciated in small paragangliomas (less than 2 cm in diameter).

Much more aggressive is the pattern of growth of glomus jugulotympanicum: CT shows early enlargement of the jugular foramen with bone remodeling and erosion of the caroticojugular spine. The tumor spreads along paths of least resistance; therefore invasion of the hypo-, meso- and epitympanum frequently occurs, associated with ossicular destruction. The tympanic part of the facial nerve may be involved as well. Salt and pepper MR pattern is more frequently appreciated in these lesions [9,38] (Fig. 11).

Congenital or **primary cholesteatoma** is far less common than its acquired counterpart. It arises from ectopic epidermoid remnants in the middle ear [39,40]. Two typical sites of origin are described: the anterior epitympanum, in the proximity of the Eustachian tube opening, and the posterior mesotympanum, close to the incudostapedial joint. Progressive conductive hearing loss may be the patient's only complaint. In almost all patients, there is no history of chronic otitis media. The tympanic membrane is usually uninterrupted. Facial nerve impairment results from anterior epitympanum lesions, undetectable at otoscopy.

The CT density of the lesion is comparable to the acquired cholesteatoma; nevertheless some peculiar findings — related to a different history and pattern of growth — permit the differential diagnosis. In congenital lesions the eardrum is preserved, and mastoid pneumatization is normal. Though ossicular chain erosions may be observed at the incudostapedial joint, the inner ear is generally preserved. The MR signal pattern is similar to the acquired form, consisting of T2 hyperintensity, T1 intermediate signal and absence of enhancement.

Middle ear **schwannomas** may be primary lesions—arising from the facial nerve, the chorda tympani or Jacobson's nerve—or may originate from the VIII to XI cranial nerves extending into the tympanic cavity, bulging through the round window (VIII) or eroding the jugular foramen (IX–XI).

Facial nerve schwannoma is the most common primary neural neoplasm. Usually, the clinical presentation consists of a characteristic association of conductive hearing loss and facial nerve dysfunction. Taste abnormalities are described in lesions involving the chorda tympani.

CT appearance of schwannomas is nonspecific, and usually does not allow differential diagnosis with paraganglioma and cholesteatoma. Nevertheless, this technique may demonstrate ossicular erosion and the involvement of the lateral semicircular canal. Cochlear promontory erosion is more likely to be detected in Jacobson's nerve schwannoma [41,42].

MR better characterises the lesion. Gd-DTPA administration causes relevant enhancement (absent in cholesteatoma) although not as intense as in paragangliomas. The pattern of growth along the course of the facial nerve (less frequently the Jacobson's or chorda tympani nerves) further raises the probability of a schwannoma.

Rhabdomyosarcoma is a rare, highly aggressive mesenchymal neoplasm. Among the pediatric population it accounts for the second most common head and neck malignancy. Even though the nasopharynx and paranasal sinuses are typical sites of origin, the lesion may arise from muscular cells near the Eustachian tube opening. Its aggressive pattern of growth leads to marked bone destruction with early involvement of the facial nerve and potential intracranial spread. Imaging findings are nonspecific, generally the patient's age is the major clue to the diagnosis [9,43].

A wide variety of lesions are also described in the literature (Figs. 12–14) including **squamous cell carcinoma**, **metastasis** (from kidney, breast, prostate), **histiocytosis** (more commonly extending into rather than arising from the middle ear), **hemangiopericytoma** and **osteoma** [44–47]. Their occurrence is sporadic.

Meningocele or meningoencephalocele (Fig. 15) is a typical pseudoneoplastic lesion. It is generally due to cortical defects (congenital, iatrogenic or traumatic) of the tegmen tympani. A mass lesion detected at CT in the proximity of an interruption of the tegmen suggests the diagnosis, and mandates an MR study (T2 sequences are particularly useful) to identify the CSF signal or even brain tissue herniation [10,48].

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