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Review

Conductive hearing loss with a “dry middle ear cleft”—A comprehensive pictorial review with CT

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ABSTRACT

Conductive hearing loss (CHL) commonly results from middle ear fluid and inflammation (otitis media). Less commonly in patients with CHL, the middle ear cleft is well aerated or ‘dry’ with absence of soft tissue or fluid clinically and on imaging. There are numerous causes for this but they can be clinically challenging to diagnose. This pictorial review aims to illustrate and discuss the CT features of both common and less common causes of CHL in patients with a “dry middle ear cavity”.

1. Introduction

Conductive hearing loss (CHL) results from impaired acoustic transmission from the external auditory canal (EAC), through the ossicular chain in the middle ear cavity, to the oval window at the bony labyrinth. High resolution CT of the temporal bones (multidetector CT or cone beam CT) demonstrates the conductive hearing pathway anatomy well and hence is ideally suited to detect many of its abnormalities. CHL commonly results from middle ear fluid and inflammation (otitis media) and this is typically diagnosed clinically. There is usually accompanying otalgia and otorrhoea, with associated soft tissue opacification of the middle ear cleft on CT. Less commonly in patients with CHL, the middle ear cleft is well aerated or ‘dry’ with absence of soft tissue or fluid clinically and on imaging. There are a myriad of causes for this but they can be clinically challenging to diagnose; radiological diagnosis through CT is therefore vital. This pictorial review aims to illustrate and discuss the CT features of both common and less common causes of CHL in patients with a “dry middle ear cavity”.

2. Review

2.1. Tympanic membrane

Abnormalities of the tympanic membrane (TM) can cause CHL in a dry middle ear cleft. The TM is usually easily inspected on otoscopy and does not require CT evaluation, however, occasionally striking CT signs can be detected.

2.1.1. Myringosclerosis

Myringosclerosis is characterised by collagenous deposition, hyalinization and calcification of the TM resulting in limited mobility and impaired sound conduction [1,2]. It is usually post-inflammatory but can have a multifactorial causation including prior myringotomy or tympanostomy tube insertion [3–5]. Otoscopically, it appears as a white sclerotic plaque which may impede visualisation of the middle ear structures. On CT, the normal TM is a barely perceptible paper-thin structure, compared to myringosclerosis in which there is thickening and/or calcification of the TM (Fig. 1). A myringoplasty (with temporalis fascia/cartilage) can mimic myringosclerosis but should be evident from the surgical history (Fig. 1).

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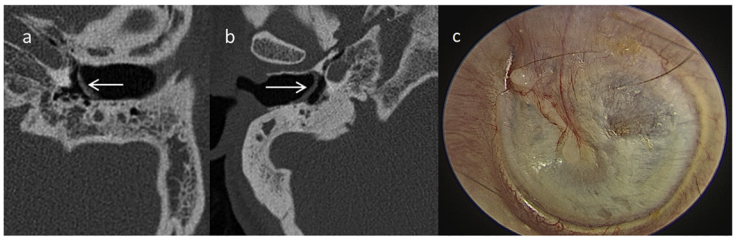

Fig. 1. Myringosclerosis. A. Axial CT of the petrous temporal bones demonstrates calcification and thickening of the TM. B. Myringoplasty can mimic myringosclerosis. C. Otoscopic view of myringosclerosis.

2.2. Ossicles and middle ear cavity

Defects in the ossicular chain from structural abnormality or lack of movement (fixation) can impede sound transmission and cause CHL loss in a dry middle ear cleft. High resolution CT depicts the ossicular chain well and is ideally suited to demonstrate many ossicular chain abnormalities.

2.2.1. Congenital ossicular malformations

Congenital middle ear malformations are often unilateral (70–90%), associated with external ear abnormalities, and usually diagnosed in early childhood [6]. However, if there is minimal external ear deformity and only unilateral hearing loss, the presentation and diagnosis may be delayed [6]. The imaging findings may be subtle, as such, a high index of suspicion and excellent knowledge of normal anatomy are essential.

Potential congenital abnormalities of the ossicular chain are wide ranging and occur in a myriad of combinations. These include: complete or partial hypoplasia (commonly the stapes or long incus process) (Figs. 2–4); ossicular conglomeration or fusion; and ossicular fixation (typically malleus onto the tympanic wall) (Figs. 4 and 5) [6]. Ossicular abnormalities are usually unilateral, and if bilateral then asymmetric; therefore, direct comparison to the contralateral side is invaluable. Use of multi-planar reformations to align both temporal bones exactly along the same plane will aid in detection of even subtle asymmetries (Fig. 2).

Hearing improvement following intervention correlates to the number of structures involved and several pre-surgical CT scoring systems have been devised to help select those that would benefit from surgical rather than conservative management [7,8]. These systems include assessment of the EAC, aeration and size of the mastoid and tympanic cavity, ossicular chain, oval and round window, facial nerve course and vascular anatomy [9,10].

2.2.2. Post-inflammatory ossicular fixation

Post-inflammatory ossicular fixation (PIOF) represents an abnormal fixation of the ossicles to the tympanic cavity as sequelae from prior middle ear inflammatory disease, with resultant impairment of sound conductivity. Three sub-types of post inflammatory ossicular fixation have been described: fibrous, tympanosclerosis, and fibro-osseous sclerosis [11–13].

The fibrous sub-type is characterised by fibrous tissue deposition and adhesions in the middle ear cavity. There is a predilection for oval window niche involvement with encasement of the stapes superstructure ("peri-stapedial tent", Fig. 6). It may also involve the malleus head or neck when found in the epitympanum. It is a difficult imaging diagnosis as the pathological soft tissue has a non-specific appearance akin to fluid or cholesteatomatous material. In the appropriate clinical setting of chronic middle ear disease, CHL, and peri-stapedial soft tissue in an otherwise dry middle ear, it should be suspected.

Tympanosclerosis is a similar pathologic entity to myringosclerosis with which it often co-exists. It occurs within the middle ear cavity proper where hyalinization/calcification can involve the tensor tympani (Fig. 7a/b) and stapedius muscles, ossicular ligaments (Fig. 7c), or onto the ossicles directly, including at the stapes footplate. Tympanosclerosis of the stapes crura may result in the stapes being "too well seen" with increased conspicuity of the normally thin stapes crura

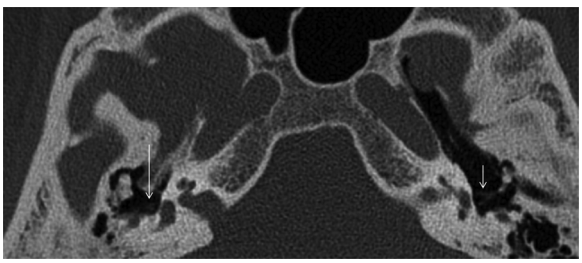

Fig. 2. Congenital stapes dysplasia. The right stapes is hypoplastic stapes (long arrow) in comparison to the normal left stapes (short arrow).

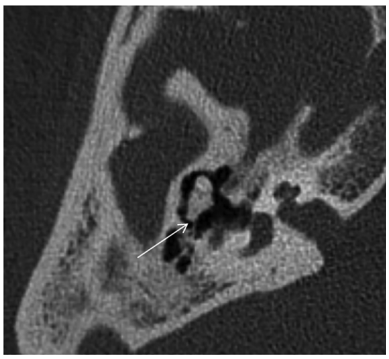

Fig. 3. Congenital incus dysplasia. There is subtle truncation of the short process of the incus (arrow). The patient has multiple other congenital middle and inner ear abnormalities (not shown).

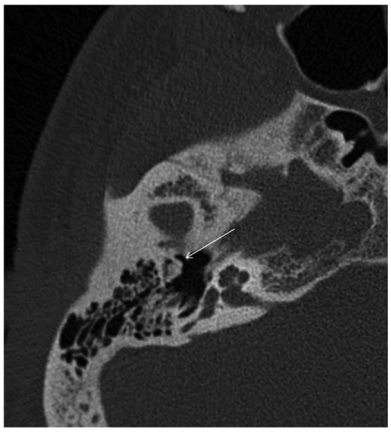

Fig. 4. Congenital malleus dysplasia and ossicular fixation. Axial CT demonstrates flattened and small malleus head consistent with hypoplasia. Compare Fig. 3 for normal appearance of the malleus head. There is also congenital fixation to the lateral epitympanic wall.

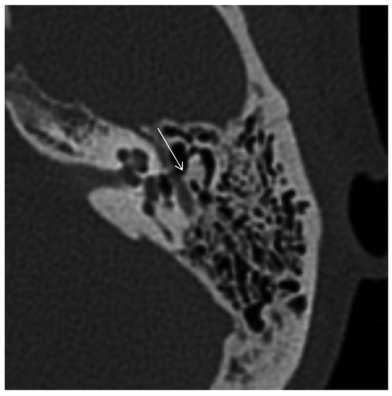

Fig. 5. Congenital ossicular fixation. The incus body is fused to the medial tympanic cavity wall (arrow) overlying the facial nerve canal.

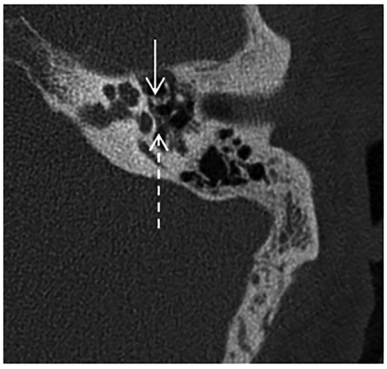

Fig. 6. Post-inflammatory ossicular fixation in a 33 year old female with prior middle ear disease. Subtle soft tissue opacity encasing the stapes (dashed arrow) compatible with fibrous ossicular fixation (peri-stapedial tent). Note also ectopic lamellar bone formation along the tensor tympani muscle (short arrow) consistent with fibro-osseous sclerosis.

(Fig. 8) [8].

The hallmark of fibro-osseous sclerosis is ectopic lamellar new bone formation (osteoneogenesis) within the middle ear cavity (Fig. 6). It is the least common sub-type but when present usually involves the epitympanum [11,12].

2.2.3. Long process of incus deficiency

Long process of incus (LPI) deficiency is being increasingly recognised on high resolution CT as a cause for ossicular discontinuity. The LPI is especially vulnerable to damage because of

its tenuous blood supply and exposure to the external milieu. It is classically eroded in inflammatory conditions of the middle ear such as chronic otitis media and cholesteatoma, but can also be congenital, associated with aging, or following trauma or surgery [11,14].

An air gap at the incudostapedial articulation is apparent when the incus long process is followed on sequential axial images to its articulation with the stapes capitulum (via the lenticular process). On axial CT, two “dots or parallel lines” should be visible in the mesotympanum – the anterior representing the malleus handle, and the posterior

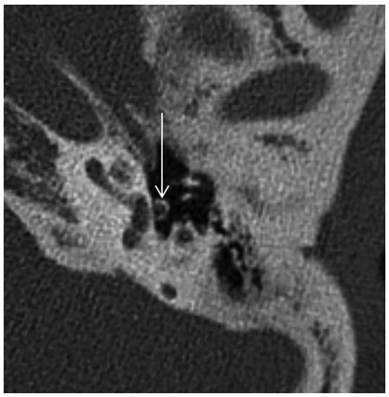

Fig. 8. Ossicular tympanosclerosis in a 63 year old female. Tympanosclerosis involving the stapes crura results in the stapes being “too well seen” (arrow).

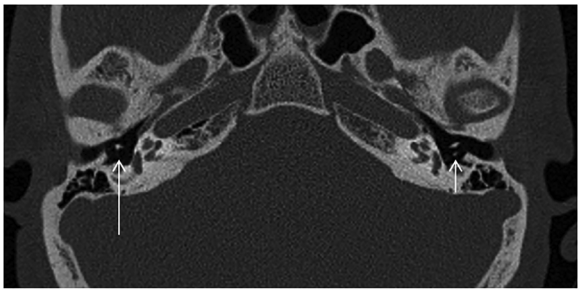

Fig. 9. Post-inflammatory long process of incus deficiency. LPI deficiency results in absence of the “two dots” in the mesotympanum on axial CT (long arrow on the right). Normal “two dots” on the left (short arrow), comprising the anterior malleus handle and posterior long process of incus.

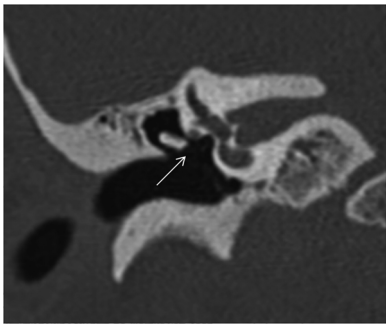

Fig. 10. Post-traumatic long process incus deficiency in an 18 year old female with CHL months after direct temporal bone trauma. Coronal CT demonstrates abnormal truncation of the LPI in a patient with prior trauma (arrow).

representing the long process of the incus. With long incus deficiency, the posterior “dot” will be absent (Fig. 9). Coronal reformations are also useful at demonstrating subtle discontinuities here (Fig. 10). Even suspected subtle abnormalities should be highlighted to the surgeon as this may aid in planning ossicular reconstruction [14]. Potentially, pre -operative measurement of the defect size may help prosthesis selection

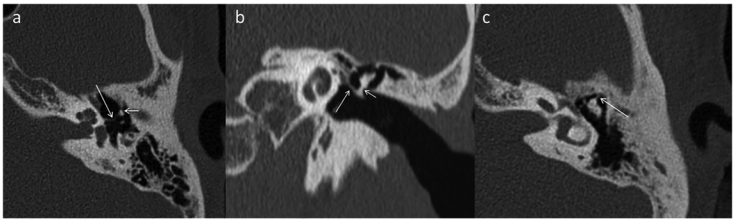

Fig. 7. Tympanosclerosis in two patients. A (axial) and B (coronal) demonstrates calcification of the tensor tympani muscle (long arrows) attaching to the malleus (short arrows). C. Axial CT demonstrates calcification of the anterior mallear ligament (arrow). Additional ectopic calcification more medially further supports tympanosclerosis. A normal ridge bony ridge can be seen at the attachment of the anterior mallear ligament and should not be confused for fixation.

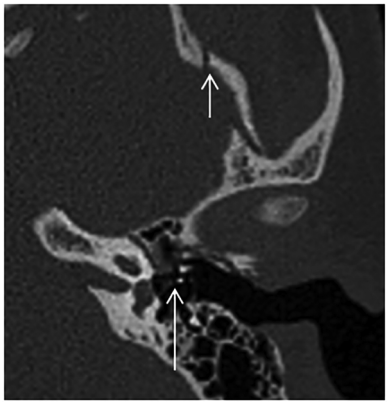

Fig. 11. Post-traumatic incudostapedial dislocation. Oblique axial CT of a patient with conductive hearing loss 1 month after trauma. There is an air gap between the lenticular process and stapes capitulum (long arrow). Note the fracture through the squamous temporal bone (short arrow).

and could possibly have an impact on future use of individualized 3D printed prostheses.

2.2.4. Ossicular dislocation

Ossicular dislocations are usually post-traumatic and readily apparent from the clinical history, however, the diagnosis may be missed or delayed, particularly if there are other distracting injuries such as intra-cranial haemorrhage. Less commonly, ossicular chain dislocations may be iatrogenic following surgery. There are 5 types of ossicular chain dislocation: incudomalleal; incudostapedial; complete incus dislocation; stapediovestibular; and malleoincudal complex dislocation [15–18].

The incus has minimal ligamentous support making it particularly vulnerable to disruption, with the incudostapedial joint being the most commonly affected [15,19,20]. As with assessment for LPI deficiency, both sequential axial and coronal images are useful to demonstrate subtle incongruities and separations at the incudostapedial articulation (Fig. 11). Malleoincudal dislocations appear as disruptions of the normal “ice-cream cone” appearance on axial CT (Fig. 12). Disruption of both incudal articulations results in a complete incus dislocation.

Stapediovestibular dislocations (dislocation of the stapes at the oval window) are rare injuries by virtue of the stapes’ deep location and strong fixation to the oval window by the annular stapedial ligament [15]. Stapediovestibular dislocations can be due to penetrating injuries through the EAC, in which the stapes is displaced medially with perforation through the oval window (internal dislocation). Alternatively, with severe traumatic force the stapes can be dislodged from the oval window into the middle ear (external dislocation) [15]. Malleoincudal complex dislocations (en-bloc dislocation of the malleus and incus) are uncommon injuries [15].

Ossicular disruptions are frequently associated with temporal bone fractures, some of which may involve the otic capsule and disrupt the facial nerve or bony labyrinth, the latter being a harbinger of irreversible hearing loss [21].

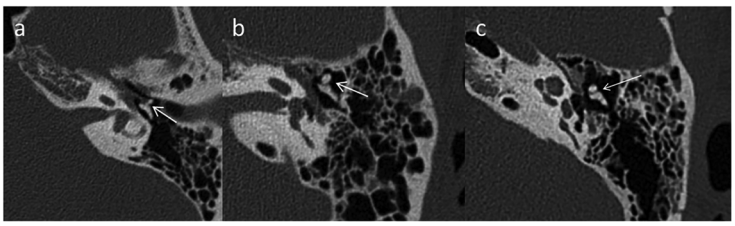

Fig. 12. Spectrum of post-traumatic malleoincudal injuries in three patients with CHL. A. Complete dislocation. The malleus has migrated lateral and cranial in relation to the incus body (arrow). B. Malleoincudal subluxation. Lateral subluxation of the malleus head (ice cream scoop) in relation to the incus body (ice cream cone). C. Subtle malleoincudal subluxation with a tiny air gap (arrow).

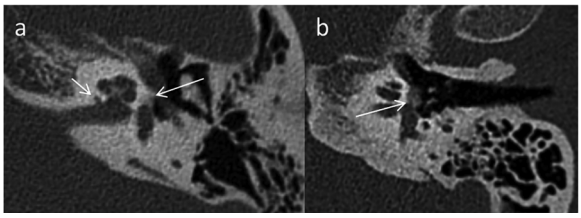

Fig. 13. Fenestral otospongiosis in two different adult patients with CHL. A. Subtle lucency at the fissula anti-fenestrum (long arrow). Note the well-defined lacuna adjacent the anterior IAC consistent with additional cavitatory otosclerosis (short arrow). B. Heaped up otosclerotic plaque encroaching and fixating the anterior stapes crura and footplate (long arrow).

2.3. Otosclerosis (otospongiosis)

Otosclerosis (otospongiosis) is a temporal bone dyscrasia primarily seen in adults, characterised initially by bone resorption, and subsequent deposition of immature spongiotic bone within the otic capsule [22–25]. The disorder is more common in Caucasians, is usually bilateral (80–85%) and displays a typical audiometric air-bone gap (Carhart’s notch) [22]. The imaging hallmark of otosclerosis is an ill-defined lucent focus within the normally dense otic capsule. During the healing phase, the spongiotic bone may re-calcify and become more solid, potentially becoming indistinguishable from normal bone, and occasionally forming protuberant plaques along the medial tympanic cavity wall.

Otosclerosis initially begins at the fissula antefenestrum (fenestral otosclerosis), a fibrocartilaginous cleft immediately anterior to the oval window (Fig. 13a). New bone deposition may encroach onto the adjacent annular ligament/footplate complex leading to fixation at the oval window and CHL (Fig. 13b). Later stages of the disease may involve the peri-cochlear and peri-labyrinthine structures (retro-fenestral or retro-cochlear otosclerosis) which rarely occurs without fenestral disease [22,26,27]. A distinctive appearance in retro-fenestral otosclerosis is the “4th ring of Valvassori” representing a continuous lytic focus around the cochlear (Fig. 14).

It is essential to describe the presence of retro-fenestral disease as these patients typically have mixed or sensorineural hearing loss (SNHL) and treatment may differ. Surgical treatment (stapedectomy/stapes prosthesis insertion) for purely fenestral disease tends to have better outcomes than retrofenestral disease due to the added sensorineural loss. Fluoride administration may have a role if there is evidence of progressive hearing loss as it is thought to delay the disease process. Cochlear implantation may be considered in patients with retro-fenestral disease and bilateral profound deficits [22]. Other potential sites of otospongiotic involvement include the round window (Fig. 15) and rarely, the ossicles (Fig. 16). Differentiation between partial and complete round window obliteration is of particular clinical significance, as the latter may limit the hearing outcomes [14].

There is increasing literature on a subtype of otosclerosis termed “cavitatory otosclerosis” [23,24,28,29]. As implied, there are discrete cavitatory foci or lacunae, which appear of lower density (approximating CSF attenuation) and better defined compared to regular otospongiotic plaques. These are usually adjacent the anteroinferior internal auditory canal (IAC) and may be more conspicuous than the

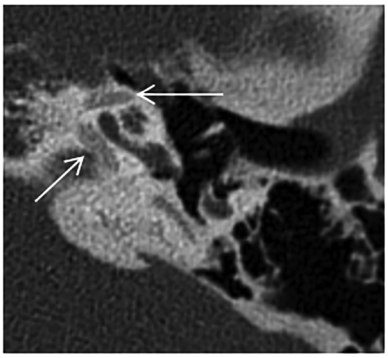

Fig. 14. Retrofenestral otosclerosis in a 74 year old male. Peri-cochlear lucency forming the “4th ring of Valvassori” (arrows).

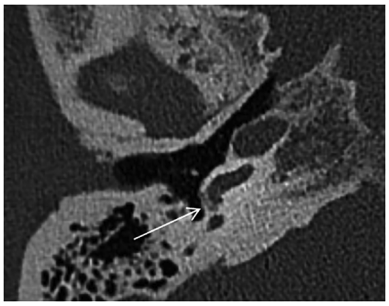

Fig. 15. Round window otosclerosis. 63 year old male with known otosclerosis and worsening CHL. Obliteration of the round window niche by otosclerotic plaque (arrow).

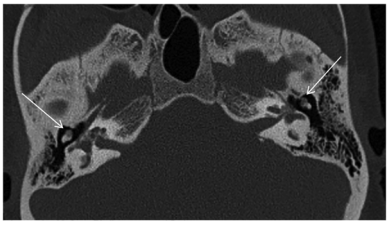

Fig. 16. Otosclerosis of the ossicular chain. Lucency of the mallear heads bilaterally consistent with ossicular otosclerosis (arrows). There is also bilateral fenestral and retrofenestral disease.

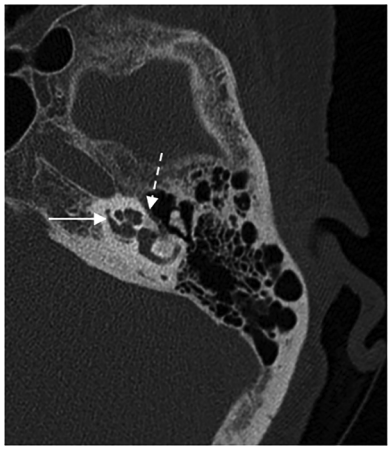

Fig. 17. Cavitatory otosclerosis. Well defined lacunae contacting the IAC (long arrow) consistent with cavitatory otosclerosis – this lesion is more extensive than Fig. 13a. There is also fenestral otosclerosis (dashed arrow).

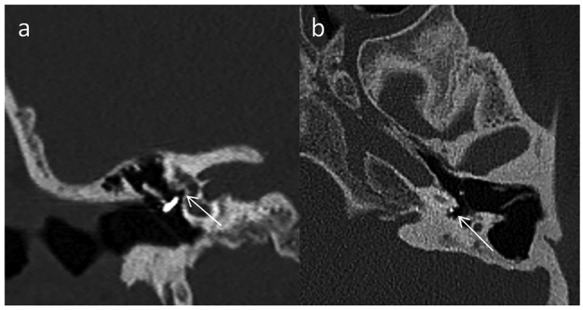

Fig. 18. Stapes prosthesis dislocation in a 68 year old female with recurrent CHL following surgery. A. Coronal CT demonstrates lateral dislocation of a stapes prosthesis from the oval window with an air gap visible (arrow). B. Axial CT demonstrates complete dislocation of a stapes prosthesis into the round window niche (arrow).

subtle fenestral disease (Figs. 13a and 17). The presence of a cavitatory focus along the anterior margin of the IAC should alert the radiologist to interrogate the fenestral region and otic capsule closely for signs of otosclerosis. The lacunae may communicate with the IAC cerebrospinal fluid (CSF) and rarely cavitate into the cochlear, creating a third window and potentially predisposing to surgical complications including CSF gush or cochlear implant lead mal-positioning [23,28,30].

2.4. Post-surgical ossicular chain reconstruction

Stapes prostheses are commonly inserted for the management of otosclerosis. CT temporal bone imaging may be requested for evaluation of persisting or recurrent conductive hearing loss after stapes surgery. The presence of oval or round window soft tissue opacity 4–6 weeks following surgery is abnormal and raises the possibility of post-surgical fibrosis which may impair sound conduction [31]. CHL may also result from new spongiotic bone fixing the prosthesis to the oval window, or impairing prosthesis function by occluding the round window.

Prosthetic subluxation or dislocation accounts for the majority of cases of post-operative CHL [32,33]. The prosthesis may be dislocated at the oval window (Fig. 18a), elsewhere into the middle ear cavity (Fig. 18b), or extruded from the tympanic cavity via a defect in the TM [31,34]. In 2% of patients, the stapes piston may penetrate the oval window into the vestibule (Fig. 19), potentially resulting in additional SNHL and vestibular symptoms if the penetration is excessive (otolithic impingement) [34,35]. Discontinuity at the prosthesis-incudal articulation can occur with a slack wire loop which migrates inferiorly under gravity (loose wire syndrome), or due to LPI necrosis which may be attributable to excessive piston length, ischemia, or foreign body reaction [31,36,37].

At imaging, metallic stapes prostheses are well demonstrated, whilst fluoroplastic prostheses may be more difficult to visualise [34]. The stapes prosthesis piston should sit centrally within the oval window and

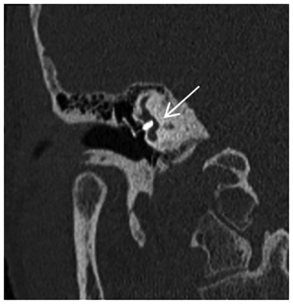

Fig. 19. Stapes prosthesis oval window penetration. Coronal CT demonstrates medial penetration of the stapes prosthesis into the vestibule (arrow).

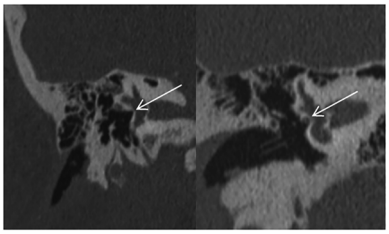

Fig. 20. Oval window atresia. Coronal CT in two patients. A. Narrowed oval window (arrow) with normal course of the facial nerve. The stapes is absent. B. Complete oval window atresia with aberrant facial nerve situated in the oval window niche (arrow).

there should be no air gap. On conventional helical CT, the medial depth of a metal stapes prosthesis is artefactually overestimated by up to 0.5 mm [34]. Allowing for this, medial penetration into the vestibule exceeding 2.2 mm or 50% of the vestibular width should raise the possibility of otolithic impingement secondary to disruption of the saccule and utricle [32,34].

2.5. Labyrinthine windows

The oval and round windows are essential for optimal sound transmission from the ossicular chain through the inner ear. Abnormality of the windows is an uncommon but recognised cause for CHL.

2.5.1. Oval window atresia

Oval window atresia is often associated with abnormalities of the external ear, but may be overlooked when the external ear is formed normally [6,38]. Children present with either conductive or mixed hearing loss; the sensorineural component related to associated inner ear abnormalities [39].

On imaging, an ossific web is seen over the oval window which may result in narrowing (hypoplasia, Fig. 20a) or complete atresia (Fig. 20b). The stapes may be malformed, absent, or mal-positioned. The long and lenticular processes of the incus may similarly be deficient or displaced. Associated facial nerve aberrancy is of critical significance; the nerve may be positioned inferomedially adjacent or over the oval window (Fig. 20b), potentially hindering or precluding surgical intervention.

2.5.2. Round window occlusion

Any process which obstructs the round window has the potential to impede sound conduction and result in CHL. Otosclerosis (Fig. 15), middle ear lesions, and inflammatory disease are all potential causes. A high riding jugular bulb (extending to the level of the IAC), with or without diverticulum, may also obstruct the round window (Fig. 21) and occasionally contact or displace the ossicular chain.

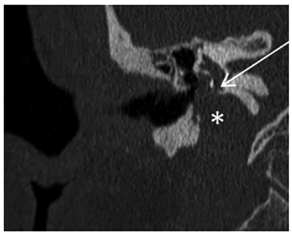

Fig. 21. Round window impingement in a 25 year old male with CHL. Coronal CT demonstrates a right sided jugular bulb (asterisk) with diverticulum occluding the round window (arrow). The diverticulum also encroaches on the oval window. No other cause for CHL was identified.

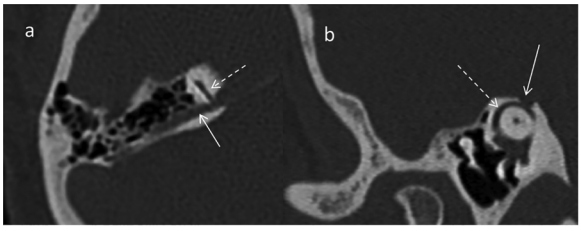

Fig. 22. Superior semi-circular canal dehiscence in a 49 year old female. A (axial) and B (Poschl view) CT demonstrate SSSCD into the superior petrosal sinus. Superior semi-circular canal (dashed arrows), superior petrosal sinus (arrows).

2.6. Third window phenomena

Third window phenomenon refers to defects in the bony inner ear integrity which allow dissipation of sound energy from the normally hydraulically sealed system [40]. The presence of a third window does not always result in hearing loss, thus the imaging findings should be correlated with clinical features, audiometry and vestibular testing. At audiometry there is a large air-bone gap, typically worse at lower frequencies [21].

Common causes of a third window include semi-circular canal dehiscence, enlarged vestibular aqueduct syndrome (EVAS), and bony dehiscence of the jugular or carotid vessels. Cavitatory otosclerosis may also result in similar third window phenomenon as previously described.

The aetiology of superior semi-circular canal dehiscence (SSCCD) (Fig. 22) is unknown, with an incidence of 2–11% on CT [21]. Patients present with hearing loss, vertigo, and pulsatile tinnitus; a classic finding is vertigo and nystagmus induced by loud noises (Tullio's phenomenon). On CT there is deficiency of the SSCC bony covering ; rarely, the SSCC may be dehiscent into the superior petrosal sinus (Fig. 22) [21]. Reformatted images perpendicular to the petrous temporal bone plane (Poschl view, Fig. 22a) are recommended to facilitate accurate measurement of the dehiscence, and to avoid partial volume artefact which may result in false positive cases. Posterior semi-circular canal dehiscence is less common and can present with similar findings to SSCCD.

EVAS is usually associated with other inner ear anomalies and a resultant complex mixed pattern of hearing loss. Third window dynamics are due to dissipation of acoustic energy through the enlarged aqueduct. The vestibular aqueduct should not measure ≥ 1 mm at its mid-point, or ≥ 2 mm at its operculum (Cincinatti criteria) [41].

2.7. Temporal bone dysplasias

Temporal bone dysplasias including Fibrous Dysplasia (FD), Pagets Disease, Osteogenesis Imperfecta, and Osteopetrosis can result in CHL via a variety of mechanisms. FD of the temporal bones can produce a CHL due to EAC stenosis, and may be further complicated by otitis media and cholesteatoma [42]. CHL secondary to Pagets disease occurs independent of canal stenosis and is secondary to diffuse softening of the otic capsule by replaced pagetic bone [14,43]. The altered bone density reduces acoustic impedance creating a so called "diffuse" or "distributed" third window (Fig. 23) [21].

3. Summary

Detecting the many varied causes of conductive hearing loss in a dry middle ear cleft on high resolution CT of the temporal bones is crucial as these conditions are often difficult to diagnose clinically. Accurate CT diagnosis is therefore essential to facilitate treatment of these conditions.

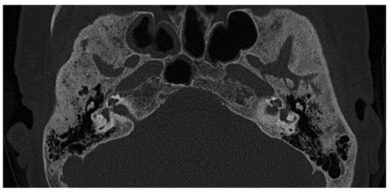

Fig. 23. Temporal bone Pagets disease in an adult patient with CHL on audiometry. Diffuse sclerosis/lucency and expansion of the temporal bones and skull base due to Pagets. The IAC and EAC are normal calibre (not shown).

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Conflicts of interest

None of the authors have any conflicts of interest to declare.

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