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INTRODUCTION

Post traumatic cholesteatoma is a rare but important late sequelae of trauma to the temporal bone. The dire consequences of this pathology require the astute clinician to have a high index of suspicion. The development of cholesteatoma following temporal bone trauma may occur from as early as 1 year and has been documented as late as 25 years.

A cholesteatoma is a cystic lesion formed from keratinising stratified squamous epithelium. Cholesteatoma may be either congenital or acquired in origin. Congenital cholesteatoma usually forms due to epithelial remnants being trapped in the temporal bone during embryogenesis. Acquired cholesteatoma may follow chronic otitis media, iatrogenic implantation of squamous epithelium into the middle ear or post trauma. Some of the complications that may develop from a cholesteatoma include facial nerve palsies, perilymphatic fistulae, intracranial sepsis, hearing loss and soft tissue sepsis.

Temporal bone fractures result from either blunt or penetrating injuries to the temporal bone. The fracture patterns that are seen may be described as either longitudinal, transverse or mixed in types. The longitudinal fractures run in the long axis of the petrous temporal bone and are less commonly associated with facial nerve paralysis. Transverse fractures run perpendicular to the long axis of the temporal bone and are more often associated with facial nerve paralysis and inner ear injuries. The sequelae of temporal bone fractures therefore include facial nerve paralysis, hearing loss, vertigo, vascular injury and cerebrospinal fluid leaks. A delayed complication is the development of cholesteatoma.

Temporal Bone Anatomy

The ear is divided into 3 parts – the outer, middle and inner ears.

The Outer Ear:

The outer ear consists of the pinna and the external auditory canal. The pinna is funnel shaped which serves as a collecting mechanism for sound waves. The external auditory canal is cartilaginous in its outer 1/3 and bony in its medial 2/3. It is lined by squamous epithelium. The canal contains sebaceous and ceruminous glands in its epithelium. The sebum and cerumen derived from these glands combine to form the substance known as wax.

The Middle Ear:

The middle ear extends from the tympanic membrane to the promontory and includes the mastoid air spaces which drain into it. The tympanic membrane is attached to an incomplete fibrous ring called the annulus. The supported inferior larger segment is called the pars tensa, and the unsupported superior segment the pars flaccida. The three ossicles in the middle ear transmit sound from the tympanic membrane to the oval window. The three ossicles consist of the malleus, incus and stapes. The middle ear also contains two muscles – the stapedius and tensor tympani which are responsible for protecting the ear from exposure to excessively loud sounds. The nerves contained in the middle ear consist of the facial nerve and its branch the chorda tympani, the auricular branch of the vagus and the tympanic branch of the glossopharyngeal nerve. The mastoid air spaces communicate via the aditus ad antrum into the epitympanic space of the middle ear cleft.

The Inner Ear:

The inner ear consists of the bony and membranous labyrinths. The bony labyrinth is filled with perilymph and the membranous labyrinth with endolymph. The 2 main structures of the inner ear are the cochlea anteriorly and the vestibule and semicircular canals posteriorly. The cochlea has 3 compartments – the scala vestibuli superiorly, the scala tympani inferiorly and the scala media inbetween. The cochlea subserves hearing. The vestibular apparatus consists of the utricle, saccule and the 3 semicircular canals. The utricle and saccule are receptors of linear acceleration. The superior, lateral and posterior semicircular canals are concerned with rotational acceleration.

LITERATURE REVIEW

1. Temporal Bone Fractures

Temporal bone fractures result from either blunt or penetrating trauma to the temporal bone. These fractures have been traditionally classified into transverse, longitudinal and mixed. Longitudinal fractures run parallel to the long axis of the petrous pyramid and comprise 70 – 90 % of all temporal bone fractures. Facial nerve palsy occurs in approximately 25 % of longitudinal fractures. Transverse fractures run perpendicular to the long axis of the petrous pyramid and comprise 10 – 30 % of all temporal bone fractures. Facial nerve paralysis occurs in approximately 50 % of these patients. Ishman et al, however, report that this system is obsolete and have reclassified temporal bone fractures into the petrous and non petrous involvement forms. They report that this new classification gives better correlation with facial nerve weakness, cerebrospinal fluid leakage and both conductive and sensorineural hearing loss.

Gunshot injuries to the temporal bone are often associated with other more serious life threatening injuries which often take precedence. Haberkamp et al reports 20 – 50 % of gunshot injuries to the head involve the temporal bone. They report that due to the density of the temporal bone, survival is possible even with massive injury. In their series, they report, the overall incidence of facial nerve involvement was 75 %, hearing loss 58 % and vascular injury 25 %. Vestibular dysfunction and persistent cerebrospinal fluid otorrhoea may also occur.

Facial nerve paralysis in temporal bone trauma may result from either direct injury or as a result of the fracture of the temporal bone. It has long been said that 90 % degeneration of the facial nerve function on electoneurography in the first 6 days is an indication for facial nerve decompression. However, Tuncay et al report that these patients often have associated life threatening injuries which take precedence, and is thus not always possible to evaluate these patients in the first 6 days. They tend to use electromyelograms (EMG), high resolution CT Scans and clinical judgement as their main monitoring tools. They report that as long as the nerve is decompressed within 3 months of onset of injury, the outcome is the same.

Vertigo following trauma to the temporal bone may have two possible aetiologies. Marzo et al describe the concussive syndrome where traumatic brain injury may be associated with the temporal bone trauma. 10 % of traumatic brain injuries are severe (classified as a Glasgow Coma Scale of 3 – 8), 10 % are moderate (Glasgow Coma scale 9 – 12) and 80 % are mild (Glasgow Coma Scale > 13). The second reported mechanism is a perilymphatic fistula. Their patient series with perilymphatic fistulae presented with persistent unilateral aural symptoms and sensorineural hearing loss. They also report that post traumatic Menieres Syndrome can occur, and may be managed medically with diuretics and salt restriction. Surgical decompression with shunt surgery has proven ineffective.

Cerebrospinal fluid leaks associated with temporal bone fractures are most often due to disruption of the tegmen tympani. This subjects the individual to the risk of meningitis. Fortunately, these are often self limiting. Those that persist will require surgical closure.

2. Cholesteatoma – Definition and Aetiology

A cholesteatoma is composed of a sac of squamous epithelium filled with keratin debris. Cholesteatoma may be either congenital or have an acquired aetiology. The acquired form usually results from chronic otitis media with active mucosal disease (Browning GG). The less common acquired aetiology is implantation of squamous epithelium into the middle ear and mastoid either at surgery or following trauma. Congenital cholesteatoma has an incidence of 0,12 per 100 000 as reported by Semaan et al. There are numerous theories to its development such as the persistence of the epithelial rest cells or alternately implantation of squamous epithelium into the middle ear due to microperforations in the tympanic membrane.

The theories of development of acquired cholesteatoma include:

1. Negative pressure theory – this involves the formation of a retraction pocket due to sustained or recurring negative middle ear pressure following eustachian tube dysfunction. This retraction pocket collects keratin and results in cholesteatoma formation.
2. Metaplastic theory – the middle ear mucosa undergoes squamous metaplasia due to chronic sepsis resulting in cholesteatoma formation.

3. Invasion theory – squamous epithelium migrates into the middle ear via a perforation in the tympanic membrane (usually pars flaccida) resulting in cholesteatoma formation.

One of the characteristics of cholesteatoma is its expansile and destructive nature. Semaan et al reported that one of the proposed mechanisms of bone destruction is pressure induced bone resorption. The other mechanisms involve the increased expression of Ki – 67, cytokeratins, calgranulins, cathepsins, cytokines and matrix metalloproteinases. The increased expression of these substances in childhood cholesteatoma also accounts for the aggressive nature of the disease in children.

Cholesteatoma as a disease entity may result in several complications. Smith and Danner reported that the incidence and complications of cholesteatoma are decreasing since the usage of antibiotics but the complications still occur and are potentially lethal.

One of the most disfiguring complications of a cholesteatoma is facial nerve palsy. Siddiq et al have shown that the prevalence of facial palsies has decreased from 1 – 2,3 % in the 1950's to 0,04 – 0,16% in 2006 in cholesteatoma patients. They report that facial nerve recovery can occur even if treatment is delayed for up to seven months. Other complications of cholesteatoma include hearing loss, vertigo and intracranial complications

3. Post Traumatic Cholesteatoma

McKenna et al report that most of the literature that deals with trauma to the temporal bone concentrates on the acute effects such as temporal bone fractures, sensorineural and conductive hearing loss, cerebrospinal fluid leakage and facial nerve paralysis. Little attention has been given to the less common and often delayed sequelae such as cholesteatoma formation.

Post traumatic cholesteatoma is not a frequently described condition. However, the otologist must be wary of it since the growth of this cholesteatoma, resulting from a temporal bone fracture, can remain undetected for years, allowing for invasive and extensive growth (McKenna et al).

Brookes described some of the theories of post traumatic cholesteatoma formation in accordance with Eckel's theories which include:

1. Central tympanic membrane perforation following blunt trauma and explosions resulting in deposition of squamous epithelium in the middle ear.
2. External meatal fractures and marginal tympanic membrane injuries resulting in invasion of squamous epithelium into the middle ear.
3. Soft tissue and bony defects of the mastoid and external meatus after gunshot wounds and traumatic meatal stenosis.

Brookes described that the latent period from time of injury to diagnosis of cholesteatoma may range from 1 – 25 years. One of the problems with diagnosing a post traumatic cholesteatoma is to establish a causal relationship between the fracture and the formation of the cholesteatoma. Bottrill however, reports that the intimacy between the fracture line and the cholesteatoma establishes this relationship.

The medico-legal implications of diagnosing a post traumatic cholesteatoma are tremendous. Brookes refers to specific conditions that must be fulfilled such as:

1. Clear proof of trauma
2. Trauma type known to predispose to cholesteatoma formation.
3. The tympanic membrane and external canal must have been normal prior to the trauma.
4. Site of trauma and cholesteatoma must correspond.
5. The clinical findings must be compatible with a traumatic cholesteatoma.

Post traumatic cholesteatoma may arise either from the middle ear and mastoid or the external auditory canal. Post traumatic cholesteatoma of the external auditory canal is rare. Brookes et al had documented only 8 cases in the literature up to 1984. He reports that most cases occur secondary to a post inflammatory acquired atresia of the ear canal. The external canal post traumatic cholesteatoma tends to occur earlier than its middle ear counterpart. It has been documented from 6 months to 4 years.

It is often difficult to examine a patient with a post traumatic cholesteatoma as the external ear canal may be obstructed by a polyp or may be stenotic. Heilbrun et al demonstrated the importance of computed tomography scanning in these instances. Some of the information that may be obtained from a computed tomography scan includes bony fragments within the mass, extension into the middle ear and mastoid, integrity of the facial canal, tegmen tympani dehiscence and the state of the ossicles of the middle ear. All of these factors influence surgical management.

A canal wall down mastoidectomy is advocated for removal of the disease and making the mastoid cavity amenable to inspection. Kveton suggests that obliteration of the mastoid and middle ear for severe trauma to the temporal bone is a very good option. He suggests that this prevents recurrence of cholesteatoma and chronic otomastoiditis. He concedes however, that the disadvantages include hearing loss, formation of a mucocele and the possibility of burying a cholesteatoma.

MATERIALS AND METHODS

The aim of the study is:

- To report on post traumatic cholesteatoma in our centre and the importance of recognising the disease in the South African context.
- To determine if surgical practice conforms to the practice reported in world literature.

Ethics approval has been obtained for the study from the Ethics Committee of the University of the Witwatersrand.

This study design is a retrospective review.

The study was conducted at the Johannesburg Hospital.

The study was conducted from November 2006 to March 2007.

The study population included patients that were admitted to the Otorhinolaryngology wards at the Johannesburg Hospital.

Data was collected from the hospital records at the Johannesburg Hospital. Parameters included were age, clinical history, clinical examination findings, audiometric findings, radiological findings, intra-operative findings and post operative sequelae.

All data is presented with Microsoft Word software programme.

CASE STUDY I

30 year old black male

History:

The patient gave a history of a gunshot wound to the left side of the head in 1998. He sustained an immediate left facial palsy and severe hearing loss for which he did not receive any treatment. He also reported an offensive left ear discharge since August 2002. Associated with this was a non pulsatile left sided tinnitus and aural fullness. There was no associated vertigo or otalgia.

Clinical Findings:

The patient had a complete left lower motor neuron facial nerve palsy.

The left ear contained a polypoidal mass occluding the external auditory canal with a mucopurulent offensive discharge.

Tuning fork tests revealed

- Rinne positive on the right ear (normal)
- Rinne negative on the left ear with Weber lateralised to the left

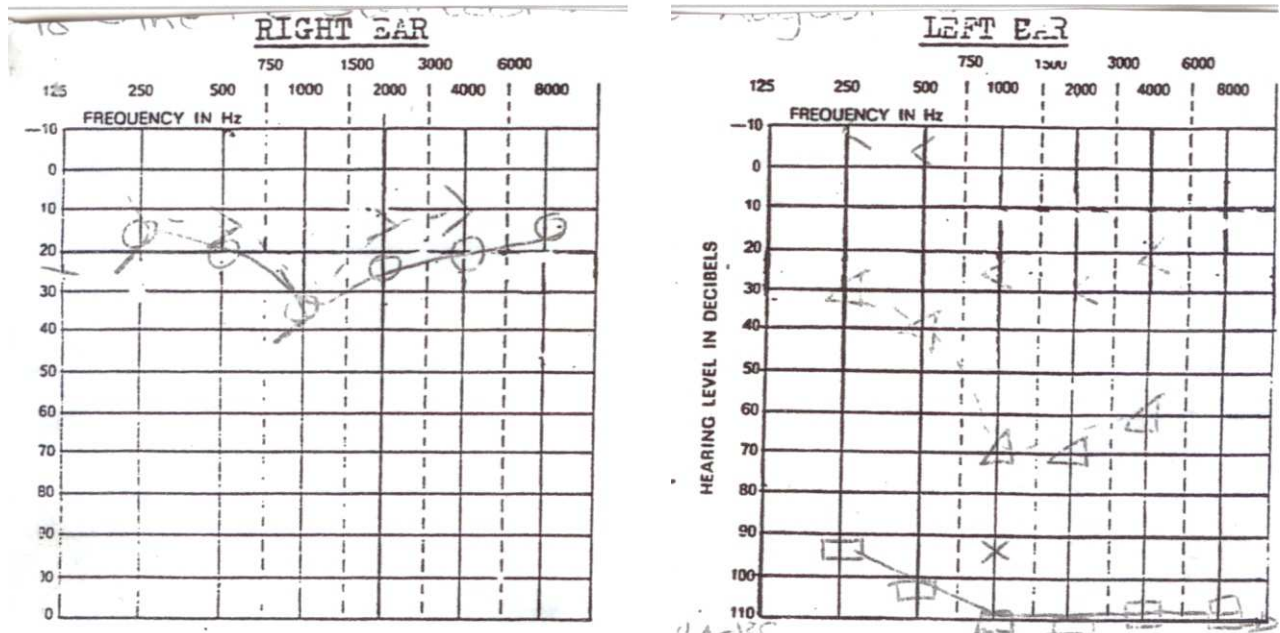
The left ear tuning fork tests revealed a conductive hearing loss compatible with middle ear disease.

The right ear was clinically normal as was the rest of the head and neck examination.

INVESTIGATIONS

1. Pure Tone Audiogram

Fig 1.



The hearing was within normal limits on the right ear.

The left ear demonstrated a profound mixed hearing loss with a conductive component of 60dB in the low frequencies and between 40 – 50dB in the mid and high frequencies.

The 60dB air bone gap in the audiometric assessment of the left ear indicated a maximal conductive deficit ie. the disease affects the tympanic membrane and ossicles.

2. CT Scan

Axial

Fig 2

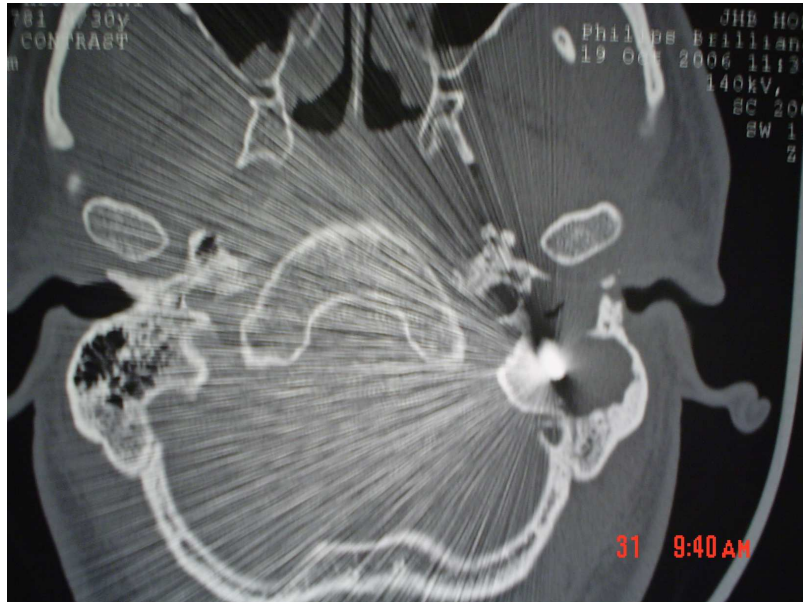


Fig 3



Coronal

Fig 4

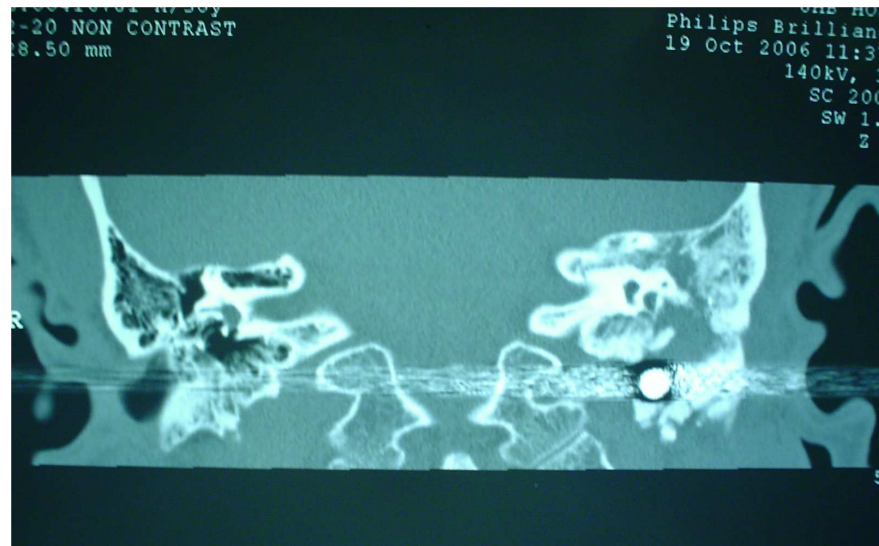
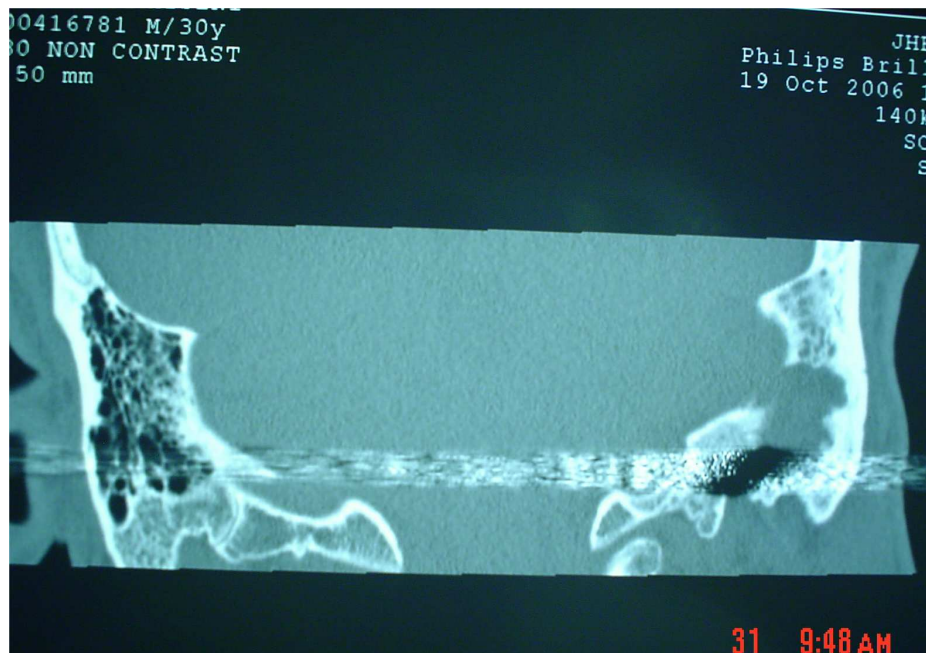


Fig 5



The CT scan demonstrates a soft tissue mass filling the entire mastoid cavity with extension into the middle ear. There is a bullet fragment in the mastoid antrum producing a significant scatter effect. The findings are those of a destructive disease process of the middle ear and mastoid thus necessitating surgical intervention.

Intraoperative Findings:

There was a transverse fracture line extending through the external auditory canal and mastoid bone. The tegmen tympani was dehiscient with exposed dura mater, however, there was no CSF leak noted. There was a large cholesteatoma sac involving the mastoid cavity and middle ear. The sac was closely adherent to the fracture line from which it appeared to take its origin. Ossicles were not identifiable. There was a bullet fragment lodged in the aditus ad antrum obstructing drainage of the mastoid into the middle ear.

Operative Procedure:

The approach was through a postauricular incision.

A canal wall down mastoidectomy (radical mastoidectomy) was performed. This involved removing the outer cortex of the mastoid bone and decorticating the outer air cells until the cholesteatoma sac was identified. The posterior canal wall was taken down. The sac was followed along the fracture line inferiorly, and the tegmen tympani superiorly into the middle ear cavity. The cholesteatoma sac was excised in its entirety. A posterior meatoplasty was performed and the entire cavity packed with ribbon gauze impregnated with BIPP (Bismuth Iodoform Paraffin Paste Packs). This is an antiseptic dressing used for long term management of an open wound.

Post Operative Management:

The patient was well postoperatively with no vertigo indicating that no perilymphatic fistula was present.

The BIPP pack was removed after approximately 1 month. The cavity was found to be clean and dry with no evidence of residual cholesteatoma or wound sepsis.

On review 3 months later, the cavity remained clean and dry with no residual disease. The complete left lower motor neuron facial nerve palsy remained.

CASE STUDY 2

A 13 year old Coloured male.

History:

The patient presented with a history of a gunshot injury to the left side of his face at the age of 3 years. He sustained severe injury to the left eye which was exenterated and replaced with an artificial eye. The injury also involved the left temporal bone resulting in a left facial paralysis which was not treated at the time. He complained of hearing loss since the incident with no associated tinnitus or vertigo. He presented now with a 10 month history of a discharging left ear.

Clinical Findings:

The patient had an artificial left eye. There was a complete left lower motor neuron facial nerve palsy.

The left ear canal was stenotic and the middle ear could not be visualised.

Tuning fork tests revealed: - Rinne was positive on the right ear (normal).
- He was unable to hear the tuning fork on the left ear and the Weber lateralised to the right ear.

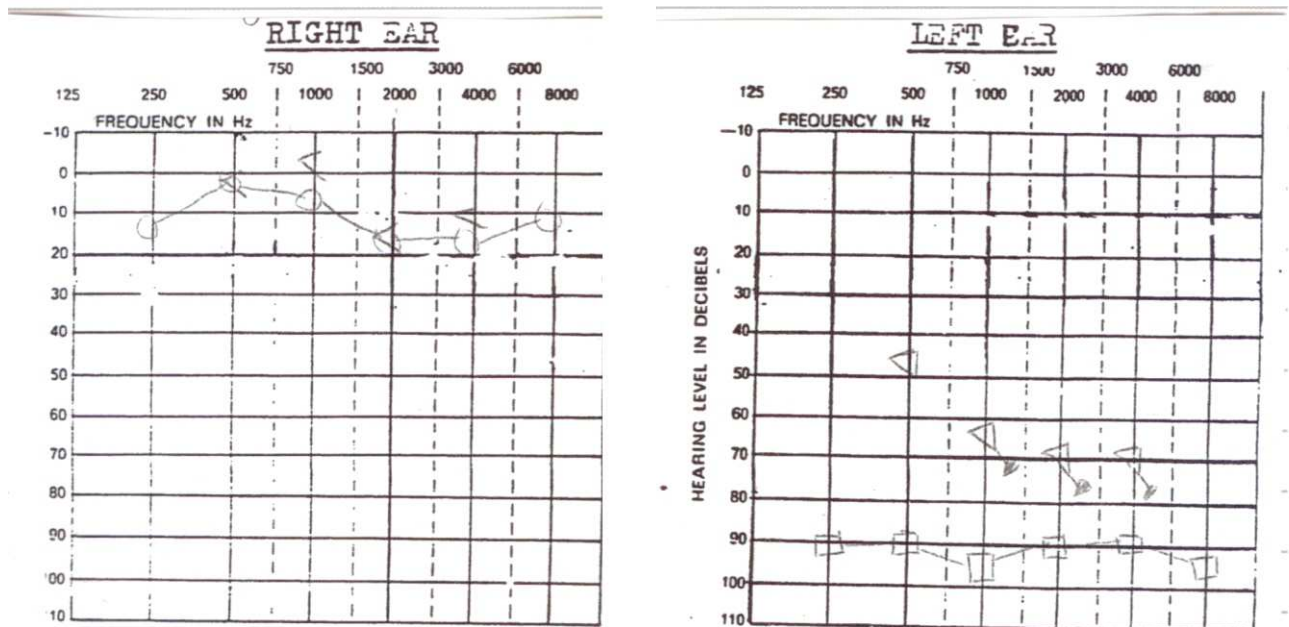
The tuning fork tests revealed a sensorineural hearing loss in the left ear compatible with cochlear or retrocochlear pathology.

The right ear was clinically normal, as was the rest of the head and neck examination.

INVESTIGATION

1. Pure Tone Audiogram

Fig 6



The hearing was within normal limits in the right ear.

The left ear demonstrated a profound sensorineural hearing loss in the mid and high frequencies. A conductive hearing loss of 40dB was present at 500 Hz.

The conductive component of the hearing loss may be attributed to a vibrotactile response. The sensorineural hearing loss is suggestive of a cochlear or retrocochlear insult. The possible causes are direct injury of the inner ear by the missile or a destructive disease process in the middle ear involving the inner ear structures.

2. CT Scan

Axial

Fig 7

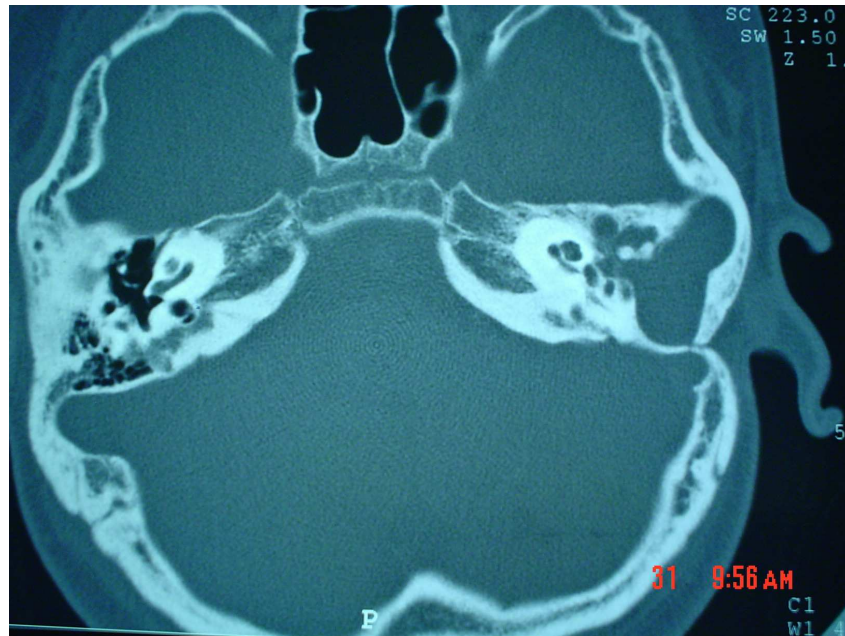
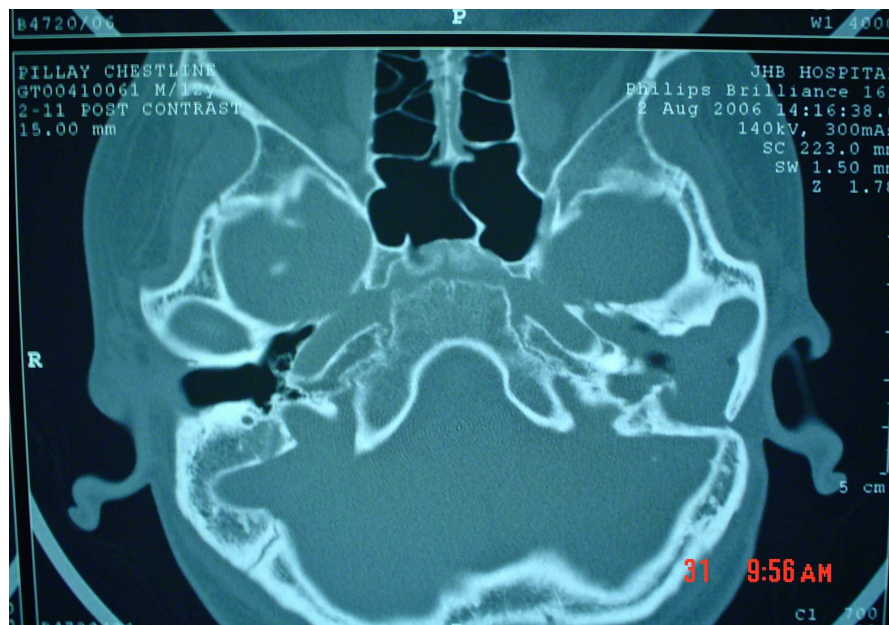


Fig 8



Coronal

Fig 9

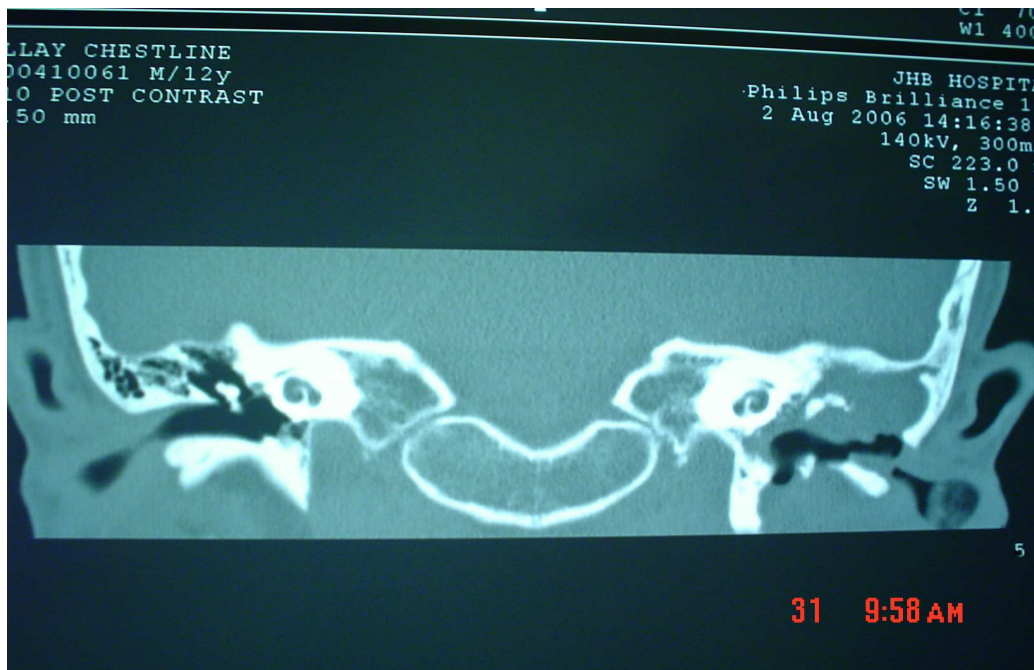
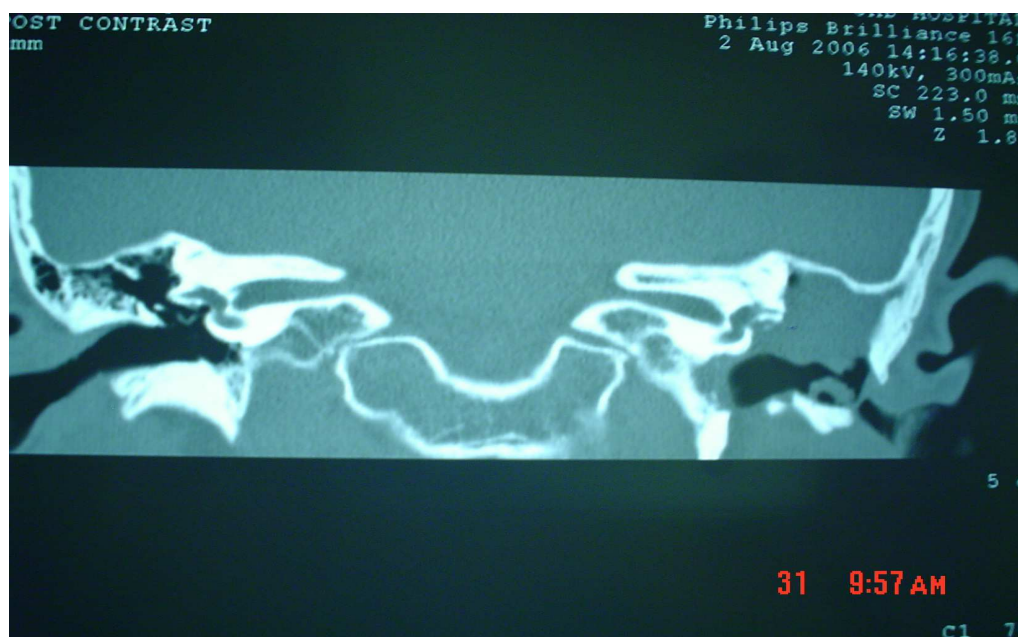


Fig 10



The CT scan demonstrates a soft tissue mass in the mastoid cavity extending into the middle ear. There is a soft tissue narrowing in the external auditory canal. There are bony fragments within the mass in the middle ear which may represent remnants of ossicles or fracture fragments. The inner ear is structurally normal.

Due to the radiological evidence of a destructive middle ear disease, surgical intervention was opted for.

Intraoperative Findings:

There was a longitudinal fracture of the temporal bone involving the external auditory canal, mastoid bone and middle ear. The tegmen tympani was dehiscient with no CSF leaks identified. There was a large cholesteatoma sac involving the mastoid and middle ear cleft. The sac was closely adherent to the fracture line. There was just a remnant of the malleus with no other ossicles identifiable. There was a stenosis of the external auditory canal behind which granulation tissue was identified.

Operative Procedure:

The approach was through a postauricular incision. A left canal wall down mastoidectomy (radical mastoidectomy) was performed. This involved removing the outer cortex of the mastoid bone, exenterating the cortical air cells which exposed the cholesteatoma sac. The posterior canal wall was then taken down to expose the entire cholesteatoma. The cholesteatoma was intimately attached to the fracture line. The cholesteatoma sac was dissected free of the fracture line and followed along the tegmen into the middle ear. It was then excised completely. A posterior meatoplasty was performed and the cavity

packed with ribbon gauze impregnated with Bismuth Iodoform Paraffin paste (an antiseptic dressing used for the management of long term open wounds).

Post Operative Management.

The patient was well postoperatively.

The BIPP pack was removed approximately 1 month later. The mastoid cavity was found to be healthy, and there was no wound sepsis.

On review 3 months later, the cavity remained clean and dry with no residual disease. The facial nerve palsy on the left side was still evident.

DISCUSSION

Gunshot injuries to the temporal bone are increasing in incidence. Due to the density of the temporal bone, survivors are also increasing in number. Thus, more patients are now presenting with the delayed sequelae of trauma to the temporal bone such as post traumatic cholesteatoma.

1. Site of Origin

One of the keys to diagnosing a post traumatic cholesteatoma is to establish a relationship between the cholesteatoma and the fracture. In Case Study 1, the cholesteatoma was seen arising from the transverse fracture line of the temporal bone.

In Case Study 2, the cholesteatoma had attachments to the longitudinal fracture line.

Bottrill reports the same finding where he noted that these cholesteatomas were seen to arise from the old fractures sites.

McKenna concurs with these findings where, in his patient series, also noted the intimacy between the cholesteatoma and the fracture lines.

2. Duration of Onset

Post traumatic cholesteatomas present many years following the initial trauma.

In the first case study, the patient presented 8 years after the initial trauma. He became symptomatic 4 years following the trauma. In the second case study, the time duration between the initial trauma and time of diagnosis was 10 years. The patient, however, became symptomatic after 9 years.

Bottrill reports that in his case study the patient presented 24 years after the initial trauma.

Brookes reports, in his series, that the duration from the initial trauma to cholesteatoma formation was 2½ months, 3 years and 10 months respectively.

McKenna reports that in his 3 patient series the time interval from trauma to cholesteatoma formation was 3 years, 6 years and 7 years respectively.

3. Theories of Pathogenesis.

Missile injuries to the temporal bone result in fractures, but also carry with them debris, bone and squamous epithelium. Some of the possible mechanisms that can be summarised from this case series are:

1. Implantation of squamous epithelium from the skin into the middle ear by the travelling missile.
2. Ingrowth of squamous epithelium of the external auditory canal into the middle ear and mastoid via the fracture defect in the temporal bone.
3. An obstructive phenomenon, resulting in inadequate drainage of the mastoid into the middle ear. This was evident by the bullet fragment in Case Study 1 which obstructed the aditus ad antrum and an external auditory canal stenosis in Case Study 2.

It is probably not a single factor, but a combination of these that contributes to the development of a post traumatic cholesteatoma.

Bottrill reports that epithelial migration through the fracture line and implantation of squamous epithelium are the most likely mechanisms

Brookes reports 3 theories:

1. Blast injuries resulting in tympanic membrane ruptures and implantation of squamous epithelium into the middle ear.
2. Fracture lines allowing for infection and epithelial ingrowth together with marginal tympanic membrane ruptures.
3. Traumatic meatal stenosis resulting in obstructed drainage of the ear.

McKenna theorised that traumatic implantation of skin into the middle ear and/or bony distortion allowing epithelial ingrowth were the most probable factors.

4. Clinical Features

The first patient presented with an offensive discharge, hearing loss, aural fullness, a left sided non pulsatile tinnitus and an aural polyp. The aural fullness may be attributed to the foreign body lodged in the mastoid, and the tinnitus to the middle ear and cochlear injury that was present.

The second patient presented with an offensive discharge, hearing loss and external canal stenosis. The offensive discharge was due to anaerobic sepsis that accompanies a cholesteatoma, and delivers the malodour typical of the disease.

Bottrill reported in his patient that a discharge from the ear, a white cystic mass in the attic of the mastoid and 20 dB conductive hearing loss were the presenting features.

Brookes reported external auditory canal stenosis, hearing loss and purulent otorrhoea as the main presenting symptoms.

McKenna noted purulent otorrhoea, otalgia, hearing loss and clinically evident cholesteatoma as the main features.

Thus all patients who present with purulent otorrhoea and hearing loss combined with a history of temporal bone trauma, should be considered as having a cholesteatoma until proven otherwise.

5. Imaging studies.

From this case series, the recommended imaging study of choice is a high resolution computed tomography scan of the temporal bones. It is not always easy to examine the middle ear, as the external canal may be obstructed due to a stenosis or a polyp. The computed tomography scan can assist in these difficult cases to establish a diagnosis of a post traumatic cholesteatoma. It can also provide information regarding the extent of the disease, integrity of the facial canal, dehiscence of the tegmen and state of the ossicles in the middle ear.

Bottrill utilised a plain mastoid X ray as his primary imaging study and reported that the defect seen in the mastoid bone was his indication for exploration.

Brooks advocated the use of plain X ray and tomograms of the temporal bones as adequate imaging studies.

McKenna utilised CT scans as his imaging study of choice.

6. Management

The prevention of formation of cholesteatoma following trauma may not always be possible. However, severe disruption of the external auditory canal may warrant debridement or widening of the external auditory canal (canaloplasty) in order to halt the disease process.

The principles of managing a post traumatic cholesteatoma are threefold:

Firstly, the disease must be eliminated. Secondly, the anatomy must be altered in order to facilitate the creation of a safe ear and thirdly, hearing should be restored (i.e. reconstruction of the hearing mechanism). It is therefore recommended that a canal wall down mastoidectomy be performed with complete removal of the cholesteatoma sac. The posterior canal wall is taken down to facilitate complete examination of the cavity at a later stage. A meatoplasty is done to further facilitate examination.

Hearing restoration need not be an immediate concern. It may be prudent to observe the patient for a suitable time period, of at least 1 year, to exclude recurrence of cholesteatoma prior to considering ossiculoplasty or other auditory rehabilitation techniques.

Patients with open mastoid cavities may develop a discharge. This can be managed initially with topical antibiotics and aural toilet. If the discharge is persistent, a temporalis muscle obliteration of the cavity can be performed. Kveton, however, recommends that cavity obliteration techniques should be performed in all severe temporal bone trauma. They report that this prevents cholesteatoma recurrence and

chronic mastoiditis. The disadvantages include hearing loss, formation of a mucocele and the possibility of burying cholesteatoma remnants.

Bottrill performed a modified radical mastoidectomy in the management of his case. He performed a canal wall down mastoidectomy but left the ossicles intact. This does result in better hearing, but the risk of cholesteatoma recurrence is higher.

Brookes performed a combined approach tympanoplasty in his first case as the first stage procedure. He planned an ossicular reconstruction as the second stage but was unable to perform it. In his second case, he performed a modified radical mastoidectomy with a homograft incus and temporalis fascia to reconstruct the hearing mechanism. In his third case, where the cholesteatoma involved the external ear canal only, a wide excision of the cholesteatoma was performed.

McKenna et al report their 3 case series as follows:

A radical mastoidectomy with no ossicular reconstruction was performed in the first case. In the second case, a modified radical mastoidectomy with incus interposition ossiculoplasty was performed. The canal wall was taken down but the stapes superstructure was left intact. The incus was used as a minor columella strut. In the third case, a modified radical mastoidectomy (canal wall down) and tympanoplasty was performed.

CONCLUSIONS

Post traumatic cholesteatoma is a rare but late complication of trauma to the temporal bone, but the incidence is increasing as the number of survivors are increasing.

The presentation of the cholesteatoma occurs many years after the initial insult. In this series the time interval was between 8 to 10 years. The main clinical features are hearing loss and an offensive discharge. The triad of previous temporal bone trauma, hearing loss and an offensive discharge points to the diagnosis of post traumatic cholesteatoma until proven otherwise.

The best method of imaging the disease process is a high resolution CT scan of the temporal bone. The information gleaned from a CT scan is far superior to that of plain X-rays.

The intimacy between a fracture line and the cholesteatoma sac is necessary in order to associate the development of cholesteatoma with the previous temporal bone trauma.

Optimal surgical management involves a canal wall down mastoidectomy, with complete excision of the cholesteatoma sac to produce a 'safe' ear. Reconstruction should be delayed until it is clear that there is no recurrence of disease.

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