Current trends in the management of the complications of chronic otitis media with cholesteatoma

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Purpose of review
Complications of cholesteatoma can be of a different nature from those of other otitis media. This review aims to undertake an analysis of current literature regarding management of the complications of cholesteatoma.

Recent findings
Despite a significant decline in the incidence of complications secondary to cholesteatoma in developed countries it is still a considerable problem in the developing countries. Among intratemporal complications, facial nerve paralysis and labrynthine fistula and among intracranial complications, meningitis, brain abscess and lateral sinus thrombosis are most common. In cases of facial nerve paralysis, decompression with complete disease eradication is considered to be the mainstay of treatment and usefulness of an epineural incision and the range of the decompression are still debatable. Labyrinthine fistula is commonly managed by a single staged matrix removal, followed by closure of the fistula. Partial labrynthectomy in difficult cases is gaining favor among surgeons today. Meningitis and brain abscesses are treated with antibiotics and steroid therapy followed by surgery when the patient is neurologically stable. In lateral sinus thrombosis, mastoidectomy and removal of infected tissue is the primary treatment. Sinus incision and thrombectomy does not seem to improve recanalization and anticoagulation is usually not necessary. Treatment of meningoencephalic herniations is based mainly on the diameter of the herniation.

Summary
There is considerable debate in the management of almost every complication of cholesteatoma. Multicentric studies to compare the efficacies of various treatment modalities are the need of the hour to come to definitive conclusions regarding the best treatment options.

Keywords
brain abscess, cholesteatomatous chronic otitis media, chronic otitis media, facial nerve paralysis, labyrinthine fistula, lateral sinus thrombosis, meningitis, meningoencephalitic herniation, otitis media

INTRODUCTION

Once established in the middle ear, mastoid or petrous bone, cholesteatoma is a destructive lesion that gradually expands and destroys adjacent structures, leading to complications. Approximately 5% of patients with cholesteatomatous chronic otitis media (CCOM) develop complications [1]. Although most studies in literature deal with overall and individual complications of otitis media, chronic otitis media (COM) or otorhinogenic causes, there are very few which deal with the complications of CCOM *per se*. In a review of articles published on complications of cholesteatomas in the last decade, only three original articles and one review article were found specifically addressing the limited issue of complications of CCOM [1–4]. This and the fact that the condition itself is quite

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KEY POINTS

- A classification of complications of CCOM has been identified.
- Labyrinthine fistula is popularly managed by one stage-total removal of cholesteatoma matrix on the fistula with high preservation rate of patient's hearing irrespective of fistula size.
- Sinus incision and thrombectomy does not seem to improve the prognosis in lateral sinus thrombosis.
- In case with brain abscess, single stage mastoidectomy with or without craniotomy should be performed with an attempt to first drain the abscess via the transmastoid route.
- Small meningoencephalic herniations can be treated by the transmastoid approach.

rare have resulted in debates regarding the optimal management of the complications of CCOM. Complications of otitis media/COM have been traditionally divided into intracranial and intratemporal (extracranial). But a literature search shows a variety of other complications associated due to the invasive nature of cholesteatoma that are not associated with other forms of COM and hence there is a need for proper classification of the complications. Here we review the classification and the recent opinions in the management of the complications of CCOM focusing mainly on facial nerve paralysis, labyrinthine fistula, meningitis, brain abscess, lateral sinus thrombosis (LST) and meningoencephalic herniation (MEH).

TEXT OF REVIEW

The advent of excellent antibiotics, better health infrastructure and advances in imaging have led to a significant decline in the incidence of complications secondary to CCOM in developed countries. But this is still a considerable problem in the developing countries from where large series are often reported [1–3,5–7].

Classification

Complications of otitis media, AOM and COM have been traditionally classified into intracranial and intratemporal (extracranial). But the complications of CCOM can be different and more deadly than those from other forms of otitis media due to the invasive nature of the disease. A literature search revealed that CCOM was capable of a variety of other complications apart from the routinely described intracranial and intratemporal complications, some of which are very morbid. They include intracranial invasion of cholesteatoma, distant abscesses like parapharyngeal, retropharyngeal or even lung abscess, jugular foramen syndrome, trigeminal neuralgia, secondary malignancies of the temporal bone and the facial nerve, osteomyelitis or fibrous dysplasia of the temporal bone or sometimes even frank destruction of temporal and/or occipital bones, and skull base. A proper description and classification of the complications of CCOM is necessary to standardize reporting in literature. The classification of complications of CCOM is shown in Table 1.

Facial nerve paralysis

Facial nerve paralysis caused by cholesteatoma is an uncommon complication of cholesteatoma (around 1–3%) [8–10] but it has a devastating effect on the patient. The onset is either sudden or gradual. Sudden onset is more common than gradual onset in cholesteatomas [8,10,11] and this could be caused due to devascularization, fibrosis or interruption of the facial nerve.

Surgery for the facial nerve damage secondary to cholesteatoma must be focused on two aspects; complete disease eradication and facial nerve decompression or repair. Four different types of damage to the facial nerve can be observed among patients; a compressed but normal segment, a reddish edematous segment, a fibrosed segment and an interrupted nerve [12▪]. Controversies exist regarding the range of surgical decompression for the fallopian canal, epineural incision, maintenance of the canal wall and simultaneous ossicular reconstruction [10]. Facial nerve decompression can be achieved with CWU, CWD or radical mastoidectomy depending on the site and extent of cholesteatoma, revision surgery or the surgeon's preference. Most authors prefer a CWD procedure while dealing with a facial nerve paralysis [8,10,13,14]. Some authors prefer to avoid incising the epineurium of the nerve that is considered to be a natural barrier to the spread of infection, and the opening of the canal is limited to the smallest area possible, that is, with facial nerve edema or redness [8,10,15]. Others prefer to decompress the entire length of the nerve from the geniculate ganglion to the stylomastoid foramen and incision of the epineurium [12▪,13]. Fibrosis, thinning, or interruption of the nerve is rarely found [8,13,16]. In such cases, when a nerve reconstruction is required, it is better to perform a CWD procedure with end to end anastomosis using nerve graft or nerve rerouting depending on degree of damage and length of the damaged nerve.

Table 1. Classification of complications of cholesteatomatous chronic otitis media

Complications of cholesteatoma	Mustafa *et al.* [1] ($n=91$)	Liang *et al.* [3] ($n=78$)
Intracranial		
Meningitis	16 (17.6%)	12 (15.38%)
Brain abscess	4 (4.4%)	9 (11.54%)
Epidural abscess		9 (11.54%)
Subdural abscess	1 (1.1%)	
Lateral sinus thrombosis/perisinus abscess	16 (17.6%)	6 (7.69%)
Otic hydrocephalus		
Encephalitis	2 (2.2%)	
Meningoencephalocele		
Intratemporal (extracranial)		
Temporal abscess (mastoid, zygomatic, Luc's Cetelli's Bezold's)	37 (40.7%)	16 (20.51%)
Postaural fistula		
Labrynthine fistula	9 (9.9%)	16 (20.51%)
Facial nerve paralysis	15 (16.5%)	24 (30.76%)
Petrous apicitis (Gradenigo's syndrome)	1 (1.1%)	
Combined	10 (11%)	
Other		
Distant abscesses (parapharyngeal, paravertebral zygomatic etc)		
Temporal bone osteomyelitis		
Jugular foramen syndrome		
Malignant transformation		
Septicemia	2 (2.2%)	

Facial nerve paralysis can also be a consequence of surgery for cholesteatoma when none existed preoperatively. Facial nerve dehiscence due to bony erosion is probably one of the primary contributing factors to intraoperative facial nerve injury in CCOM. Facial nerve dehiscence in CCOM is an underappreciated condition and it is especially important that it be noted because the presentation of a facial nerve paralysis in a patient with cholesteatoma is only around 1–3% but the overall incidence of facial nerve dehiscence found intraoperatively in cholesteatoma surgeries ranges from 19 to 33% which indicates that although the fallopian canal could be eroded by the disease, the facial nerve itself is fairly resistant to infiltration. The incidence of facial nerve dehiscence is very high in CCOM compared with COM without cholesteatoma. In CCOM, the incidence of dehiscence ranges from 30 to 33% in primary surgery and 30 to 42% in revision surgeries. The most common site of dehiscence is the tympanic segment (81–94%) followed by the mastoid (6–10%).

Labyrinthine fistula

Labyrinthine fistulas may affect either patients with CCOM or those with COM without cholesteatoma but are clearly more frequent in the former group. In his series of 16 patients with labyrinthine fistula, Magliulo *et al.* [17▪] reported that 15 (93.75%) were patients with CCOM. Early reports of labyrinthine fistula associated with cholesteatoma were by Tos [18] and Parisier *et al.*, [19] who reported a 4.8 and 9.6% incidence, respectively, of lateral semicircular canal fistula in CCOM. The incidence of labyrinthine fistula in CCOM in literature broadly varies from 4 to 15% and this has not changed in the recent decades [20,21,22▪▪,23,24,25▪,26].

The management of labyrinthine fistula has seen much debate and evolution from the time it was feared that removal of the cholesteatoma matrix would always lead to a dead ear [27]. However a review of hearing results in patients with labrynthine violation indicates that both of these techniques are equally likely to preserve hearing (84% with matrix removed and 83% with matrix preserved) [22▪▪]. In small fistulas, we prefer to remove the matrix in the first sitting (Fig. 1 [28▪▪], Table 2). We prefer a closed tympanoplasty even in cases of large fistulas wherein the matrix is left in place initially and is removed later during a preplanned second stage procedure 6 months after the first operation (Fig. 2) [28▪▪]. On the contrary when an open technique is performed we suggest that the

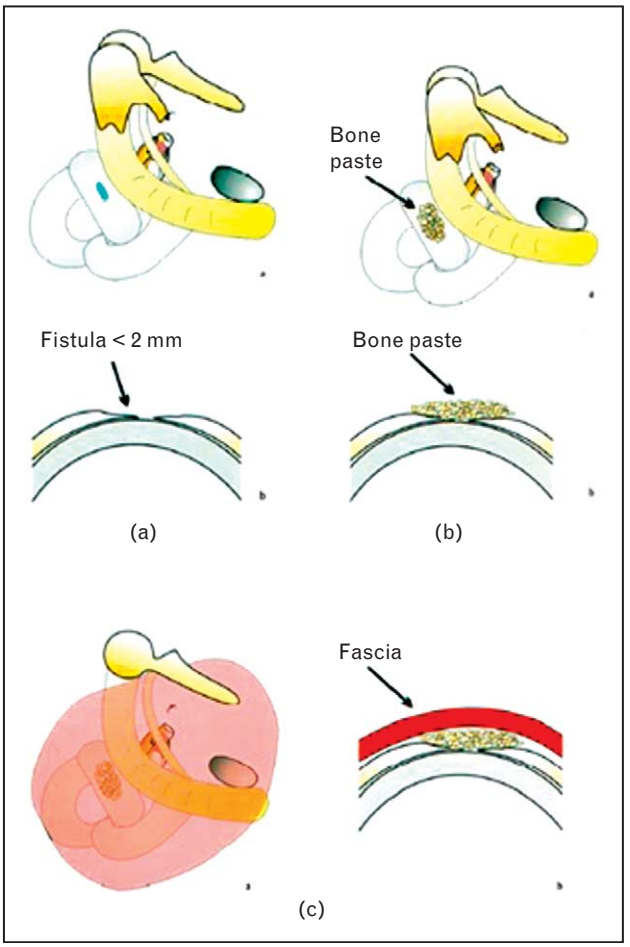

FIGURE 1. (a–c) Management of a small labyrinthine fistula in a single stage. View of the fistula after matrix removal. The fistula is immediately covered with bone pate. Fascia overlying the bone pate. Adapted from [28▪▪].

matrix be left over the fistula in all cases (Table 3). In cases with only hearing ear, with a fistula larger than 2 mm, an open technique is indicated. If the fistula is adherent to the membranous labyrinth or the fistula is larger than 1 mm, it is trimmed less than 1 mm larger than the margin of the fistula to interrupt its possible nutrient pathways and left in place and then the second stage is performed. It was seen in our series that when the ear was reinspected after 6 months, the cholesteatoma had completely disappeared in 67% of the cases. A residual cholesteatoma in the form of a small cyst that can be safely removed was seen in 33% of cases. Bony closure of the fistula occurred in 60% of the cases. In our series, there was no change in postoperative bone conduction in 97% of the cases (Table 4) [29]. Our technique has found favor among contemporary surgeons.

In 1995, Kobayashi *et al.* [30] proposed a hearing preservation technique that has certainly changed the attitude of many surgeons in the treatment of labyrinthine fistula. They developed a technique of drilling the SCC to facilitate eradication of cholesteatoma in a deep fistula and then to obliterate the two ends of the transected SCC without loss of postoperative hearing. Another study by the same group [22▪▪] states that the postoperative hearing results were closely related to the size of the labyrinthine fistula. Larger fistulas and multiple fistulas carried a higher risk of postoperative bone conduction hearing than smaller ones when the matrix was removed.

Meningitis

Meningitis due to secondary infection is one of the most common intracranial complications of COM with cholesteatoma [7,31–33]. Meningitis can develop via three routes; direct extension through bony erosion or preformed channels and hematogenous spread [34▪,35,36]. Connections between the hematopoietic bone marrow and middle ear are also suggested as a possible route for otogenic meningitis [37]. Clinical meningitis with features such as headache, neck stiffness, fever and photophobia is confirmed by CSF white blood cell count of greater than 300 per cm^3 or by organism identification via gram staining, culture or PCR. *Proteus mirabilis* and anerobes are the most commonly isolated organisms [1,38–41] followed by *Staphylococcus*, *Enterococcus* and *Pseudomonas aeruginosa*.

The broad spectrum antimicrobial therapy is primary treatment and intravenous corticosteroid therapy produces favorable prognosis [42]. Corticosteroids should be administered as early in the course as possible to maximize efficacy [6]. Modern

Table 2. Closed tympanoplasty: management of cholesteatoma matrix with regard to the size of the fistula

Size of the fistula	Matrix *in situ*	Matrix removed	Total
Small (<1 mm)	2/10 (20%)	8/10 (80%)	10 (100%)
Medium (1–2 mm)	5/26 (19.3%)	21/26 (80.3%)	26 (100%)
Large (>2 mm)	30/43 (69.8%)	13/43 (30.2%)	43 (100%)
Total	37/79	42/79	79

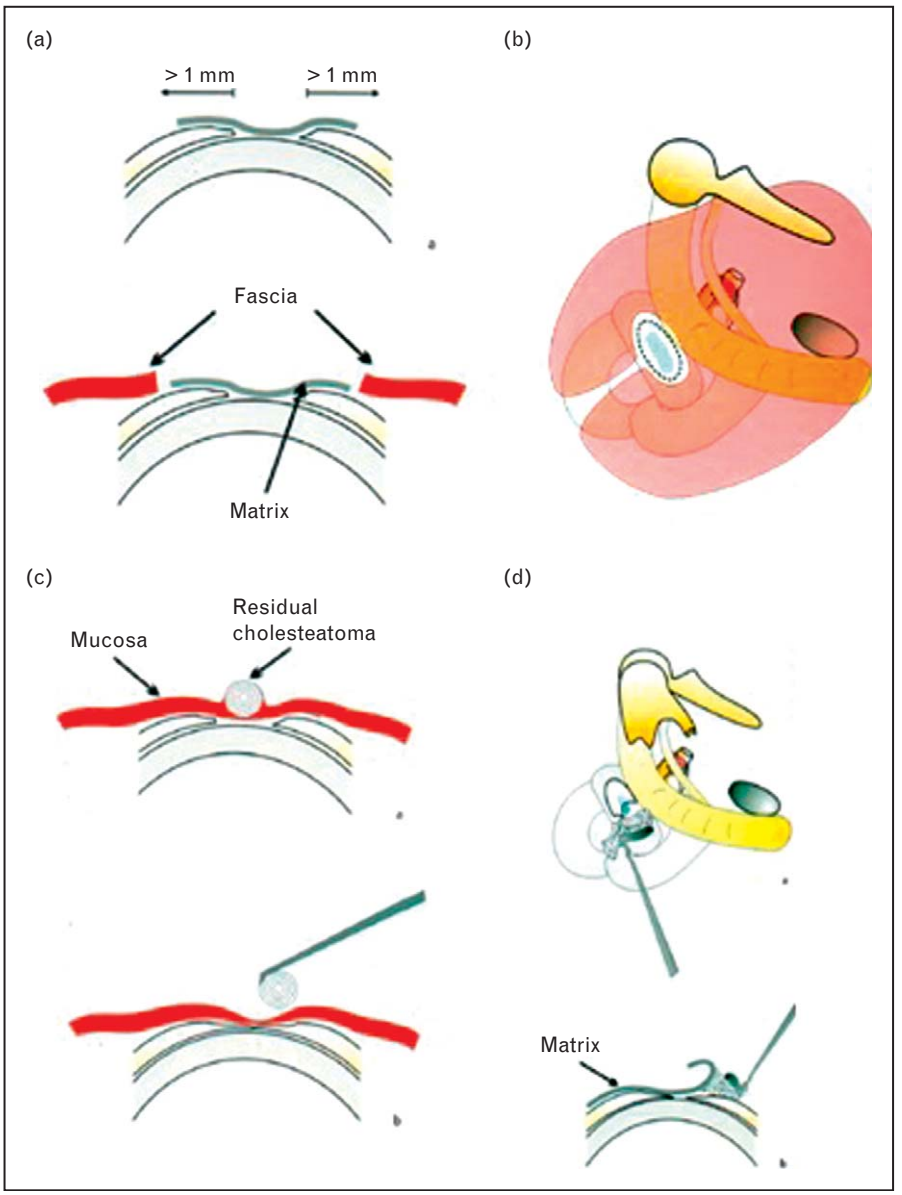

FIGURE 2. (a–d) Management of cholesteatoma matrix on the large labyrinthine fistula when it is left in place for a second staged surgery. (a) Matrix is left in place (b) Marix is exteriorized through the fascia. View in the end. Removal of cholesteatoma during second stage surgery. Dissecting the cholesteatoma matrix from the fistula. Adapted from [28▪▪].

antimicrobials have revolutionized the treatment of bacterial meningitis. The preantibiotic era practice of performing mastoidectomy within the first 24 h is now not recommended [40]. Emergency surgery is indicated only in patients with coalescent mastoiditis or with worsening infections and/or neurologic manifestations or neurologic failure 48 h after the initiation of drainage and high dose antimicrobial

Table 3. Management of large fistulas ($n = 117$)

Procedure	No	Matrix *in situ*	Matrix removed
Closed tympanoplasty	43 (36.8%)	30 (69.8%)	13 (30.2%)
Open tympanoplasty	23 (19.7%)	20 (87%)	3 (13%)
Classic/modified radical mastoidectomy	51 (43.5%)	44 (86.3%)	7 (13.7%)
Total	117 (100%)	94 (80.3%)	23 (19.7%)

Table 4. Hearing results in various surgeries for labrynthine fistulas

Results in open procedures ($n=79$)			
	Open tympanoplasty ($n=26$)	Classic or MRM (53, $n=37$)[a]	Total (79, $n=63$)[a]
Bone conduction level unchanged	25 (96%)	28 (75.7%)	53 (84.1%)
Bone conduction levels deteriorated	–	4 (10.8%)	4 (6.4%)
Dead ear	1 (4%)	5 (13.5%)[b]	6 (9.5%)
Results in closed tympanoplasty ($n=79$)			
	Matrix *in situ* (37, $n=35$)[c]	Matrix removed (42, $n=40$)[d]	Total (79, $n=75$)[d]
Bone conduction level unchanged	34 (97.1%)	36 (90%)	70 (93.4%)
Bone conduction levels deteriorated	1 (2.9%)	2 (5%)	3 (4%)
Dead ear	–	2 (5%)	(2.6%)

[a]16 patients with preoperative profound hearing loss are excluded.
[b]Three labrynthectomies.
[c]2 patients with cochlear fistula are excluded.
[d]2 patients with preoperative profound hearing loss are excluded.

therapy. A radical surgery for cholesteatoma removal must be performed as soon as the patient is neurologically stable. Improvement after the procedure is often drastic and immediate. It must be borne in mind that the mortality rate of bacterial meningitis in adults is high (5–18.75%) especially among older patients with pneumococcal meningitis [1,39,40,43].

Intracranial abscess

Brain abscess is the second most common intracranial complication due to otogenic infection [39,44]. However, some authors have reported high incidence of brain abscesses in their series over and above other complications [7,31,38,45]. It is also true that the commonest cause of abscesses in the temporal lobe and cerebellum is chronic ear infection [45,46].

A brain abscess begins when bacteria propagate in and around venous channels leading from the mastoid into the adjacent brain parenchyma. The first event after the arrival of bacteria into the cortex or white matter is the migration of polymorphs into local capillaries with endothelial swelling and focal cerebritis. At this stage, the disease can be successfully managed by intravenous antibiotics alone. With more time, the tissue becomes edematous, hemorrhagic, and necrotic and the abscess is formed. Brain abscesses may vary greatly in size, often have an irregular shape, and frequently are multilocular. At first, the capsule is poorly defined, but over time it becomes firmer and can easily be stripped from the underlying edematous brain [47].

Most authors agree on initial empiric broad spectrum antibiotic coverage (including gram positives, gram negatives and anerobes), switching to culture-specific treatment if and when sensitivities become available [31,48,49]. The antibiotics therapy is continued for at least 6 weeks with serial imaging follow-up [31,50]. There is a role for conservative management of small abscesses (<1 cm) [45] but surgery has to be done at the earliest opportunity for eradication of cholesteatoma. Once a decision has been taken to drain the abscess, it can be done via mastoidectomy [38,45], open evacuation via craniotomy, excision, aspiration through a burr hole or stereotactic aspiration. As a mastoidectomy has to be performed to eliminate cholesteatoma, it is logical to attempt a drainage via mastoidectomy first and if found inadequate, approach via craniotomy. Follow-up is by serial MRI every 2 weeks for 6 weeks. Mortality is between 0 and 31% [38,31, 44,45].

Lateral sinus thrombosis

LST usually results from perisinus abscess through mastoid bone erosion due to cholesteatoma. Pressure on the outer wall of the sinus by the abscess leads to necrosis or mural thrombus that becomes infected. If the infected clot propagates to the confluence of sinuses and superior sagittal sinus, otitic hydrocephalus can develop. Its extension to the internal jugular vein (IJV) can cause septic pulmonary emboli. The infected thrombus can go into the blood stream and give rise to septicemia and metastatic abscesses [6,47]. Clinical features include headache, fever, otalgia, otorrhoea, neck stiffness, dizziness, fever, postauricular pain, erythema and VII nerve palsy.

There is universal agreement that treatment of LST with a combination of antibiotics and surgery is required. A modified radical mastoidectomy has been used successfully in the treatment of CCOM presenting acutely with LST [51–54]. It has the advantage of providing definitive treatment for the patient while avoiding the need for a second procedure. The evidence from the literature illustrates that early surgery in these patients ensures a better prognosis [53,55]. Management of the lateral sinus has included observation, sinus decompression with simple mastoidectomy and removal of perisinus infective tissue, needle aspiration, venotomy with partial or complete evacuation of thrombus, IJV ligation and endovascular transvenous thrombectomy [56–62]. If free blood is aspirated then no further intervention is required. If there is no return of blood the diagnosis is confirmed. Most studies support incision of the sinus and evacuation of the clot [63,64].

The role of IJV ligation is controversial. Historically, the most common complication of LST was septic emboli with hip, ankle, knee and shoulder joint involvement [65]. In the preantibiotic era ligation of the IJV was commonly performed to prevent septic emboli [51]. Today the procedure is only indicated for specific reasons: when the clot extends beyond the mastoid area; persisting septicemia and pulmonary complications despite initial treatment with surgery and antibiotics; and infection or thrombosis of the IJV [51,63].

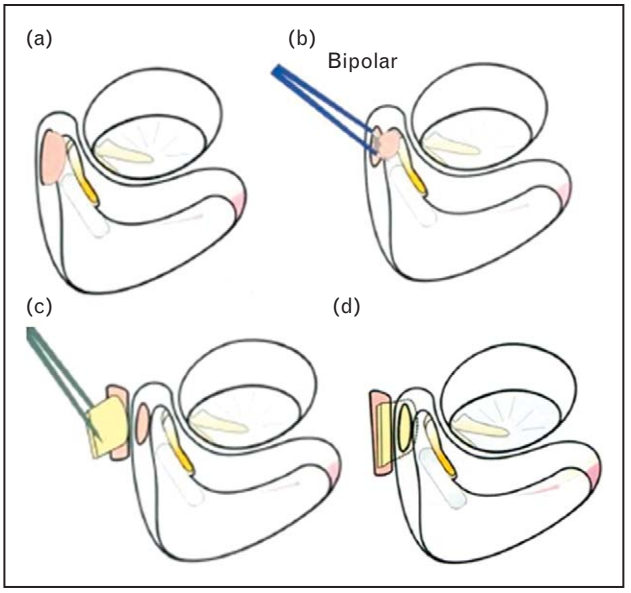

FIGURE 3. (a–d) Management of a meningoencephalic herniation. Menigoencephalocele herniation. Coagulation of the herniated tissue using bipolar cautery. Cartilage is used to reconstruct the defect after pushing back the herniated tissues. View after completion. Adapted from [28▪▪].

Meningoencephalic herniation

MEH of the temporal bone, also known as fungus cerebri, is a rare occurrence in clinical practice. It is a potentially life-threatening condition and is caused by tegmen erosion of cholesteatoma or iatrogenic tegmen defect due to previous cholesteatoma surgery [28▪▪,66].

In our experience, management of herniation depends on its size. A small MEH ($<1\,cm^2$) can be pushed back intracranially via transmastoid approach, and supported by a piece of cartilage, bone paste and fascia with layer by layer. Medium sized MEH ($1–2\,cm^2$) can be repaired by combined approach; after pushing back the herniation intracranially via transmastoid approach, a sufficiently large piece of autologous or homologous cartilage is inserted extradurally through a small craniotomy to ensure the reposition (Figs 3 and 4) [28▪▪]. The bony

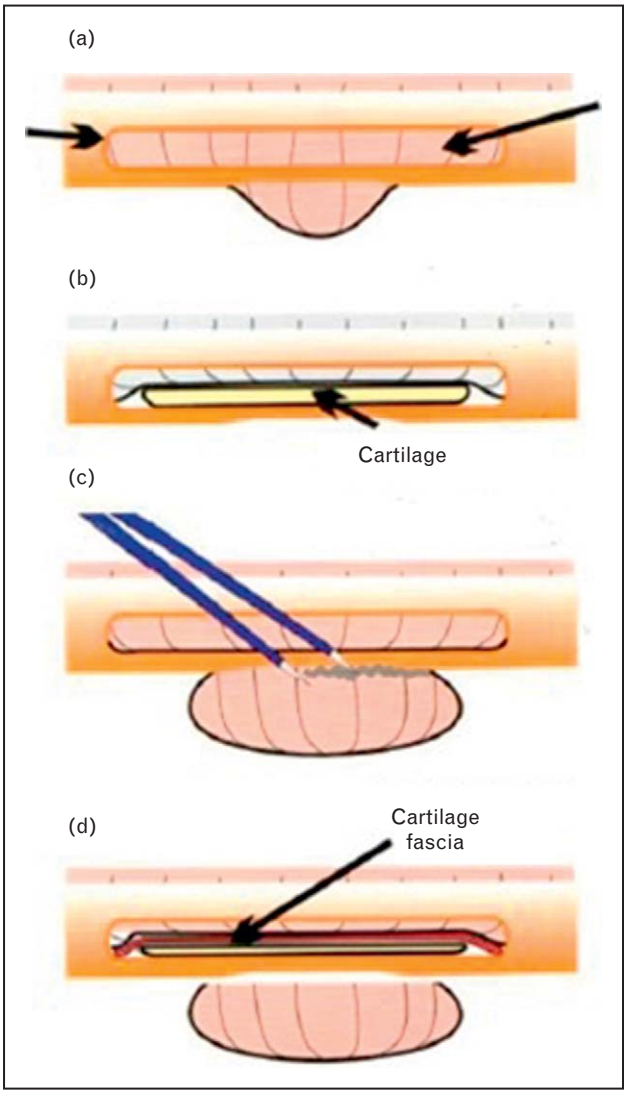

FIGURE 4. Repair of medium sized (a and b) and large (c and d) meningoencephalic herniation. Adapted from [28▪▪].

defect is further repaired by bone paste and fascia. In cases of large MEH (>2 cm^2), a middle cranial fossa approach is used. After resecting the herniated tissue by coagulation, a fascia is inserted between brain tissue and dura and the other fascia is extradurally placed. A cartilage is placed between bone defect and the dura for reinforcement [28▪▪,66,67].

CONCLUSION

Although many issues in the management of cholesteatoma complications are still debatable, it is clear that introduction of stronger antimicrobial agents and advancements in surgical techniques in the last couple of decades have produced better results than before. Early diagnosis and careful analysis is essential to decrease morbidity and mortality. There is considerable debate in the management of each complication resulting from chronic otitis media with cholesteatoma. Multicentric studies to compare the efficacies of various treatment modalities are the need of the hour to come to a definitive conclusion on the best treatment options.

Acknowledgements

None.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ▪ of special interest
- ▪▪ of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 509–510).

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