



Review

Myringitis: An update

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ABSTRACT

Myringitis can be acute or chronic. Though they commonly present with ear discharge with or without pain, the etiology and the management principles differ. Granular myringitis generally is an external ear pathology extending to tympanic membrane and present as painless otorrhea, whereas the bullous myringitis is commonly associated with acute inflammation of middle ear cleft and present with severe ear pain. This literature review discusses the clinical as well as the therapeutic aspects of these inflammatory conditions.

1. Introduction

The term myringitis is used to denote an inflammatory condition of the tympanic membrane (TM), involving its lateral surface with or without the involvement of the adjacent bony external auditory canal. The myringitis can be acute or chronic. Granular myringitis (GM) and eczematoid myringitis (EM) constitute the chronic inflammatory conditions of TM, whereas the acute conditions include bullous myringitis (BM), also known as bullous hemorrhagic myringitis, and fungal myringitis (FM). Though the symptomology of these conditions may overlap, the etiology and the management principles differ from each other. Many of these conditions can even have severe implications on long-term ear morbidity in the affected individuals (Drendel et al., 2012; Wild and Spraggs, 2003; Dawes, 1953). The aim of this narrative review is two discuss the etiopathology, clinical manifestations and the management of these inflammatory conditions of TM by segregating the updated literature on these topics.

2. Granular myringitis

GM is one of the commonly encountered conditions in outpatient departments. It is characterized by chronic painless otorrhea in the presence of granular areas over the TM (El-Seifi and Fouad, 2000; Blevins and Karmody, 2001), as depicted in Fig. 1. The other synonyms of GM include chronic myringitis, myringitis granulosa, granulomatous myringitis, granulating myringitis and granular external otitis (Bansal, 2017). The middle ear disease should be excluded before diagnosing the GM, and the duration of symptoms should be more than one month (Blevins and Karmody, 2001). Though the exact etiology is not known,

the trauma to TM surface due to repeated ear cleaning or previous surgery seems to be the forerunner for GM etiology. Localized infection of the deeper layers of the TM after the traumatic de-epithelization of the outer surface is the probable cause for granulation formation in GM. In all those studies which have cultured the discharge from GM, both methicillin sensitive and resistant *Staphylococcus aureus* and *Pseudomonas aeruginosa* are the two commonly isolated organisms (El-Seifi and Fouad, 2000; Levi et al., 2013; Kim, 2011). A history of the previous myringotomy and myringoplasty has been reported in nearly 80% of children with GM in a study (Levi et al., 2013). However, this disease is rare in children (Kim, 2011).

GM commonly affects females (Kim, 2011). Clinically, the affected individuals would have recurrent episodes of painless otorrhea, ear fullness or ear blockade with normal interim periods (Blevins and Karmody, 2001). Examination of the ear by otoscopy, otoendoscopy or microscopy would reveal the presence of granulation tissue over the TM, commonly in posterior-superior quadrant (El-Seifi and Fouad, 2000). Rarely there can be associated TM perforation, which is often transient (Blevins and Karmody, 2001) and would heal spontaneously (Wolf et al., 2006). Many authors have reported conductive hearing loss in GM (Blevins and Karmody, 2001; Levi et al., 2013; Fechner et al., 2002). In almost 20% of the patients, GM can affect both the ears (Wolf et al., 2006). The histopathological examination of these granular lesions has shown granulation tissue infiltrated by nonspecific chronic and acute inflammatory reaction (Kim, 2011; Wolf et al., 2006). If not treated adequately, long-standing GM can lead to inflammatory stenosis or atresia of the external auditory canal (Blevins and Karmody, 2001; Lavy and Fagan, 2000).

Various classifications of GM have been described in the literature.

Abbreviations: TM, Tympanic membrane; GM, Granular myringitis; BM, Bullous myringitis; EM, Eczematoid myringitis; FM, Fungal myringitis
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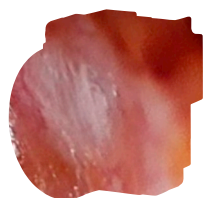


Fig. 1. Clinical photograph of granular myringitis showing diffuse granular depositions (black arrow) in the middle part of the tympanic membrane posteriorly (A and P - representing anterior and posterior respectively).



Fig. 2. Clinical photograph of bullous myringitis showing cyst like bulge (black arrow) in the posterior part of tympanic membrane (A and P - representing anterior and posterior respectively).

According to El Seifi and Fouad (2000), GM has three presenting forms, the focal, the diffuse and the segmental forms depending on the appearance on ear examination. Wolf et al. (2006) have classified the GM into four grades based on the extent of disease, in which the grade I is focal de-epithelization, grade II is focal polypoid granulations, grade III is diffuse polypoid formation over the entire TM and grade IV is when the granulations also involve EAC wall. Kim (2011) in his classification of GM integrates both the location as well as the appearance. He broadly categorizes the GM as either marginal or non-marginal, depending on the involvement of margin of TM by the granular lesions. Subsequently, depending on predominate appearance of the lesion, these lesions are sub-classified as polypoidal or ulcerative. Marginal ulcerative type is the most common variety seen in the series reported

by him. However, this classification has not been vastly used, probably because of multiple sub-categories involved in it as against the wolf's classification which is simple and yet practical for clinical care as well as research communications. Recently, an etiological classification was proposed by Bansal (2017) who divides GM into primary and secondary. However, by the definition of GM, many a condition included under the secondary GM in this classification are not supposed to be considered as GM, except maybe for the traumatic causes. In fact, the absence of middle ear infection is one of the criteria to diagnose GM (El-Seifi and Fouad, 2000; Blevins and Karmody, 2001). Moreover, though some of the non-infective pathologies like congenital cholesteatoma, middle ear effusion or grommet insertion are shown to be associated with the GM (Kim, 2011), the etiological role of these conditions in GM is not yet clear.

The diagnosis of GM is generally clinical. Whenever in doubt, a computer tomography of the temporal bone can be done to rule out the underlying otitis media with granulations. Some authors have discussed the utility of microscope-based optical coherence tomography in detecting the micro-anatomical changes of the tympanic membrane in GM (Guder et al., 2015). Recently, a hand held otoscope integrated with optical coherence tomography has also been shown to be a clinically useful tool to detect the changes in tympanic membrane (Park et al., 2018). However, the usage of optical coherence tomography in routine clinical practice for diagnosing the GM may not be cost effective. Traditionally the topical treatment with antibiotic ear drops has been the treatment of choice for many years (Blevins and Karmody, 2001). A diluted vinegar solution (Jung et al., 2002), diluted hydrogen peroxide (Van der Meer, 2010), 5 fluorouracil (Atef et al., 2010), castellani solution (Kim, 2011) are some of the topical agents being used for GM with variable success rates. Vinegar though useful, can cause canal irritation pain and dizziness (Jung et al., 2002). Diluted hydrogen peroxide and silver nitrate cautery are associated with increased risk of iatrogenic TM perforation (Van der Meer, 2010). Castellani solution comprises 4.5 g phenol, 10 g resorcinol, 0.3 g basic fuchsin, 5 ml acetone, 9.4 ml of 80% ethanol, and 85.6 ml of distilled water (Kim, 2011). The components of this solution have antifungal (carbol-fuchsin), antibacterial (ethanol and resorcinol), and acidic (acetone) properties. Also, it promotes re-epithelialization as well (Kim, 2011) and has no ototoxic effect (Gültekin et al., 2010). Nevertheless, the recurrence rate is high with conservative treatment using topical agents (El-Seifi and Fouad, 2000). Use of antibiotics and even steroid drops are generally associated with recurrence of the symptoms, and for the recurrent symptoms of GM, dilute vinegar seems to produce favorable outcomes (Neilson and Hussain, 2008).

Carbon dioxide laser ablation is an effective and minimally invasive office based method that can be of use in medically refractory GM cases (Fechner et al, 2002; Jang et al., 2006; Cheng and Shiao, 2008). One or 2-s exposure to the carbon dioxide laser with spot size 0.5–1 mm and a power setting of 5–10 Watts in continuous mode has been shown to be ideal for this purpose, with low rates of recurrence and a low incidence of complications (Jang et al., 2006). But, the follow-up duration of these studies is relatively short. It needs to re-iterated that laser therapy

Table 1
The differences between the Granular myringitis and Bullous Myringitis.

Granular Myringitis	Type of Myringitis	Bullous Myringitis
Chronic inflammation	Pathology	Acute inflammation
Trauma (External cause)	Etiology	Otitis media (Internal cause)
Staphylococcus aureus and Pseudomonas aeruginosa	Isolated organisms	Streptococcus pneumonia and Haemophilus influenza
Painless otorrhea	Symptoms	Severe pain, maybe otorrhea
Granular/ulcerative	Appearance of lesion	Blister/cystic
Normal or maybe conductive	Hearing loss	Conductive or Mixed
Symptoms tend to recur	Clinical course	Hearing loss may persist
Surgery > Topical drops	Treatment	Analgesics, decongestants
Laser ablation		Antibiotics

The etiological and the clinical characteristics are in bold.

can rarely lead to perforation of the TM (Fechner et al, 2002).

Surgical treatment with excision of involved part of TM and reconstruction using overlay or underlay myringoplasty is shown to be effective in cases with failed conservative treatment (El-Seifi and Fouad, 2000). The reported recurrence rate of GM with this approach is around 1% (El-Seifi and Fouad, 2000). The combined tympanic epithelial avulsion and overlay myringoplasty are especially suitable for type III and IV Wolf classification, and it takes around three months for the TM to heal completely and the air-bone gap to return to the pre-operative stage (Zhang et al., 2010). Canaloplasty can reasonably correct the conductive hearing loss due to external auditory canal stenosis or atresia in a long-standing case of GM (Lavy and Fagan, 2000).

A recent systematic review highlighted the non-existence of any randomized controlled trial in GM (Chung et al., 2018). By comparing all the treatment options, surgery seems to be the most effective treatment option (Chung et al., 2018). However, the topical agents form an essential line of therapy even now, having been used as an adjunct with either of laser therapy or surgery.

3. Bullous myringitis

The BM is relatively an uncommon condition. The reported incidence of BM in children of less than two years age is around 5.7% (Kotikoski et al., 2003a). In contrast to GM, the BM is commonly associated with the middle ear disease (Palmu et al., 2001), and there can be a transient inner ear dysfunction in more than half of the BM patients. Milligan (1926) in 1920s attributed the BM to viral influenza. Subsequently, etiopathological studies pointed towards the mycoplasma pneumonia also known as 'Eaton agent.' By inoculating this agent in normal young adults of 20–40 years age, Rifkind et al. (1962) demonstrated the development of BM in ¼ of the subjects most of whom had no prior demonstrable antibodies to Eaton agent. However, they failed to culture the mycoplasma from the fluid collected after its rupture from one of the affected patients. Only other study which has isolated mycoplasma from ear swab also has done so under questionable circumstances (Sobeslavsky and Abrahamovic, 1965; Mellick and Verma, 2010). As per a review by Roberts (1980), other 14 attempts to isolate the mycoplasma from the ear swab have not yielded. On the other hand, some authors have demonstrated four fold rise of the antibody titers in the paired sera of patients who were said to have extrapulmonary mycoplasma infection (Liong et al., 2015). However, the utility of the antibody titer in diagnosis of BM due to mycoplasma is yet to be established. Interestingly, Streptococcus pneumonia, Haemophilus influenza, and beta-hemolytic Streptococcus were successfully cultured from ear swab in these studies. The percentages of microorganisms seen in these studies were same as found in non-bullous acute otitis media (Roberts, 1980). Subsequent larger studies also have reported the same bacteriological profile in BM as that in acute otitis media (Palmu et al., 2001). These authors suggest that the BM should be treated as a subtype of acute otitis media with the elevated symptoms which is thought to be due to the heightened severity of the infection/inflammation in the middle ear cleft (Palmu et al., 2001; Roberts, 1980; Kotikoski et al., 2003b). Rarely, even a chemical irritation of the middle ear space in an otherwise intact TM can produce BM with acute otitis media (Minoda et al., 2011). Other conditions reported with BM include measles, chicken pox, infectious mononucleosis, unspecified viral infection of the upper respiratory tract (Roberts, 1980). Interestingly, in a comparative study between BM and acute otitis media, it was found out that the BM was not seen in ears with patent tympanostomy tubes (Kotikoski et al., 2003b).

As per the studies reported in the literature, BM seems to be common in women (Drendel et al., 2012). However, among the younger children, boys are affected more often (Kotikoski et al., 2003a). The children are prone to BM during the winter season for the apparent reason of dysfunctional Eustachian tube in cold climates. It can be bilateral in 16% (Drendel et al., 2012). Ear pain is the predominant

symptom in BM, which is generally not seen in other forms of myringitis (Kotikoski et al., 2003b; McCormick et al., 2003). The symptoms of BM are more ear specific compared to the symptoms of acute otitis media, even in infants (Kotikoski et al., 2003b). Ear examination might reveal clear fluid filled blister(s) or hemorrhagic blisters occupying the part or whole of the TM (Elzir and Saliba, 2013), as shown in Fig. 2. Due to the accompanying middle ear fluid, most of these patients would have conductive hearing loss, however, the sensorineural hearing would also be abnormal in more than half of the BM patients (Drendel et al., 2012; Hoffman and Shepsman, 1983; Hariri, 1990; Eliashar et al., 2004). The mechanism of reduced sensorineural thresholds in these patients is not clearly understood. Cochlear transduction and resulting increased intralabyrinthine pressure has been proposed by Milligan (1926) who suggested pilocarpine injections and lumbar puncture to reduce this pressure. Other proposed pathophysiological basis include virus-induced neural degeneration (Dawes, 1953) and toxic labyrinthitis through semipermeable round window (Goycoolea et al., 1980). Generally, the hearing loss in these patients would mainly be in high frequency range (Drendel et al., 2012), suggesting that the basal turn of the cochlea is the likely site to be insulted in these patients, irrespective of the actual cellular pathophysiological process.

The culture from the ear discharge may be done in selected cases with rupture of blebs, but its clinical relevance is still questionable. Serology can be of value in diagnosing mycoplasma infection (Liong et al., 2015; Al Busaidi et al., 2017) in BM, however, literature on this aspect is limited and needs to be explored by further clinical studies. Pure tone audiogram is necessary in BM cases to identify the type of hearing loss and also to quantify it. Tympanometry shows flat curve in a majority of the BM patients (Kotikoski et al., 2003b; McCormick et al., 2003). It can also demonstrate peaked curve with positive pressure, suggesting the acute nature of the fluid collected in the middle ear in BM (Kotikoski et al., 2003b). Apart from the hearing loss, the dysfunction of the entire vestibular system has also been objectively documented in the affected individuals of BM (Eliashar et al., 2004). Nevertheless, the hearing loss and the vestibular dysfunction are supposedly transient, and they recover after the relief of otitis media in most of these affected individuals (Eliashar et al., 2004). But some patients may have a persistent sensorineural hearing loss (Drendel et al., 2012; Hoffman and Shepsman, 1983; Hariri, 1990).

The treatment of BM comprises mainly analgesics, anti-inflammatory agents, and nasal decongestants. A combination of systemic and topical antibiotics with topical steroid is shown to be effective (Elzir and Saliba, 2013). The role of systemic steroids in BM concerning hearing recovery is not clear. Similar hearing recovery rates have been reported in patients who received only systemic antibiotics and in patients who received systemic antibiotics with oral steroids (Ciorba et al., 2011). Though some patients who received steroids had complete recovery of hearing, many had a partial recovery, on the other hand, the improvement was complete in some other patients who did not receive systemic steroids (Drendel et al., 2012). Surgery in the form of myringotomy may be required in refractory cases or those with impending complications due to associated acute otitis media. In 95% of the patients of BM, the pain subsides in 3 days, and otorrhea resolves in 5 days. Middle ear fluid may take 5 weeks for complete resolution (Kotikoski et al., 2003b).

4. Summary

Table 1 summaries the two commonly encountered inflammatory diseases of the tympanic membrane. Appropriate diagnosis of these conditions is essential to provide an accurate treatment and to enable symptom free recovery.

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