Journal of Otology 14 (2019) 149–154

Contents lists available at ScienceDirect

Journal of Otology

journal homepage: www.journals.elsevier.com/journal-of-otology/

Review

Post-inflammatory acquired atresia of the external auditory canal

Michel Kmeid [a, *], Jade Nehme [b]

[a] School of Medicine, Lebanese University, Beirut, Lebanon
[b] Lebanese University, Department of Otolaryngology, Head and Neck Surgery, Beirut, Lebanon

ARTICLE INFO

Article history:
Received 14 April 2019
Received in revised form
17 June 2019
Accepted 26 July 2019

Keywords:
Stenosing external otitis
Meatal atresia
External auditory canal
Conductive hearing loss
Canaloplasty

ABSTRACT

Acquired atresia of the external auditory canal (EAC) is a rare cause of conductive hearing loss. It has been traditionally classified into 4 categories: traumatic, post-operative, neoplastic and inflammatory.
Post-inflammatory acquired auditory canal atresia is thought to be the result of chronic and repetitive infectious bouts affecting the auditory canal. Nevertheless, the underlying pathophysiology of this disorder is yet to be fully elucidated. Current data fail to clearly state the impact that certain underlying systemic disorders may have on the EAC. The possible association to metabolic disturbances such as iron deficiency is also emphasized.
In the light of these findings, this analysis can be used to improve the classification of this entity thereby standardizing the assessment of therapeutic approaches.

Contents

1. Introduction

Acquired atresia of the external auditory canal is an unusual cause of conductive hearing loss. It has been classified by Tos et al. into 4 categories: post-traumatic, post-operative, neoplastic and post-inflammatory (Tos and Balle, 1986). The post-inflammatory group is a rare disorder with an estimated annual incidence of 0.6 cases per 100000 (Becker and Tos, 1998). It is defined as a chronic inflammatory process of the ear canal and the tympanic membrane that could lead to a replacement of the original epithelium with a dense fibrotic tissue. This may result in total obliteration of the auditory canal. Usually, the lateral part of the canal remains patent and takes the form of a blind ending pouch (Luong and Roland, 2005). Different terminology have been used to describe this entity including “chronic stenosing external otitis”, “medial meatal fibrosis”, “obliterative otitis externa”, and “acquired medial canal fibrosis” which is often a source of confusion.

There are many controversies regarding the underlying etiopathogenesis and management of this disease. The most common

* Corresponding author.
E-mail address: michelk.md@gmail.com (M. Kmeid).
Peer review under responsibility of PLA General Hospital Department of Otolaryngology Head and Neck Surgery.

https://doi.org/10.1016/j.joto.2019.07.002

cause as reported in the literature is chronic otitis externa and/or chronic suppurative otitis media following repeated infectious episodes. However, many reported series did not find any direct cause to the inflammatory process affecting the external auditory canal; they labeled these cases as “idiopathic”. Others suggested the possible link to systemic dermatologic conditions that may affect the skin of the auditory canal such as eczema or lichen planus. Although several local etiologies can lead to EAC inflammation and stenosis, there are no publications in the literature that clearly report the possibility of a systemic cause to this disorder. Herein, we present a review of the literature about the etiology, pathophysiology, clinical presentation and management of post-inflammatory acquired EAC atresia highlighting the possible role of an underlying systemic etiology in its pathogenesis.

2. Etiopathogenesis

Acquired atresia of the external auditory canal has been traditionally subdivided into 4 categories according to the Tos classification (Tos and Balle, 1986; Lavy and Fagan, 2000).

The post-traumatic group involves patients with extensive injury to the external auditory canal following blunt trauma (i.e. motor vehicle accidents), lacerations from a penetrating injury (i.e. gunshot wound), thermal or chemical burns, and otologic surgery. Fracture of the bony canal or a posteriorly displaced fracture of the mandibular head are often present (Tos and Balle, 1986; Becker and Tos, 1998). The end result is a circumferential loss of the epidermis covering the auditory canal resulting in the deposition of a granulation tissue, fibrosis and stenosis (Bajin et al., 2015). Acquired canal atresia may occur after otologic surgery when extensive dissection of the skin and the periosteum of the auditory canal is performed and the resultant tympanomeatal flap is inadequately reapplied to the bony canal. Additionally, lateral blunting may occur following an “overlay” myringoplasty (Bajin et al., 2015; Jacobsen and Mills, 2006). Although rarely reported, keloids and hypertrophic scars at the level of the skin incision in the EAC may also be a cause of post-operative canal atresia or stenosis (Bajin et al., 2015; Jacobsen and Mills, 2006).

Numerous neoplasms have been reported to originate at the EAC level causing narrowing of the canal with a resultant conductive hearing deficit. Cutaneous malignancies are the most common to be found (basocellular and squamous cell carcinoma) (White et al., 2012). Glandular benign and malignant tumors arising for the ceruminous glands of the EAC is also a possible although rare eventuality (White et al., 2012). Metastatic disease from primary breast, lung or renal carcinoma commonly involves the highly vascular petrous portion of the temporal bone. Usually the EAC is affected secondarily to temporal bone involvement; however, there are some reports of direct metastasis to the EAC with an intact temporal bone (Carson et al., 2005; Michaelson and Lowry, 2005). These tumors may present as a polypoid lesion of the EAC masquerading a chronic inflammatory process of the auditory canal.

The last category regarding acquired EAC atresia is the most controversial yet regarding its underlying pathophysiology and is often regarded as a distinct entity. Yet, there are no large series addressing its exact incidence. Additionally, the origin of the inflammatory process as well as the different steps that lead to narrowing of the canal and formation of the atretic plate are not fully understood. Traditionally, post-inflammatory medial meatal fibrosis is thought to be the result of repetitive infectious bouts affecting the conductive apparatus of the human ear. Becker and Tos described this entity as a fibroproliferative inflammatory process that progresses over years (Becker and Tos, 1998). This process originates either from the middle ear or the external ear. It usually begins as a loss of the squamous epithelial layer of the tympanic membrane and medial canal leading to inflammation and deposition of a granulation tissue (Lavy and Fagan, 2000; Ghani and Smith, 2013). A granular myringitis develops and this entity was first described by Toynbee as far back as 1860 (Toynbeen, 1860). This granulation tissue later on undergoes fibrosis and thickening. At the level of the narrow anterior tympanomeatal angle, the two adjacent granulomas of the tympanic membrane and medial canal meet triggering a process of epithelialization. The ensuing narrowing of the EAC predisposes to further infectious episodes perpetuating the whole process in a vicious cycle (Lavy and Fagan, 2000).

Few reports have described the effect of head and neck irradiation on the temporal bone and the external auditory canal and its link to EAC stenosis. In fact, multiple radiation-related changes can affect both the bony and soft tissue components of the auditory canal (Adler et al., 1985). Osteoradionecrosis of the bony canal with sequestration and lacunae formation is a common finding but it is important to note that ulceration and thickening of the skin of the canal, subepithelial fibrosis and atrophy of the ceruminous glands can also be prominent features (Adler et al., 1985; Tirelli et al., 2015). This often results in recalcitrant otitis externa, EAC stenosis and conductive hearing loss. Moreover, the risk of radiation-induced EAC stenosis and atresia increases significantly if surgery was previously performed around the EAC (i.e. parotid surgery) (Carls et al., 2002). Thus, radiotherapy is yet another local source of EAC inflammation, fibrosis and stenosis that merits recognition as a direct cause of chronic stenosing external otitis.

2.1. New insight

The above-discussed classification is limited by the fact that it only tackles local factors as a starting point for the development of acquired EAC atresia. Although the aforementioned underlying causes are clearly associated with chronic stenosing external otitis, the wide spectrum of this disorder should not be restricted to the above categories and other less conventional etiologies should be thought. It all started when some reports found in the literature failed to identify a specific etiologic factor in a number of affected patients (Hopsu and Pitkäranta, 2002, 2008; Slattery and Saadat, 1997). These patients did not have any history of trauma or prior ear surgery and had a healthy mastoid and middle ear cavities. Although an infectious event in the auditory canal was reported during the acute phase, it did not appear to be the direct causative agent of the relentless chronic inflammation in the auditory canal. As a result, the authors labeled these cases as “idiopathic”.

In fact, a few number of case reports and small case series have linked acquired atresia of the auditory canal to an underlying systemic disorder. Hopsu and Pitkaranta postulated the possibility of an associated auto-immune disorder that triggers the whole process (Hopsu and Pitkäranta, 2002, 2007). They presented 3 cases with “idiopathic” post-inflammatory medial meatal fibrosis who concomitantly suffered from a long-standing lichen planus of the skin and mucous membranes. Interestingly enough, one of these patients had also a history of rheumatoid arthritis and polymyalgia rheumatica and another suffered from insulin-dependent diabetes mellitus. Lichen planus is a chronic inflammatory disorder of unknown etiology that affects glabrous skin and mucous membranes. Given the fact that medial meatal fibrosis affects exclusively the glabrous skin of the medial auditory canal and tympanic membrane, the authors suggested the possibility of a link between the two disorders (Hopsu and Pitkäranta, 2007). That same association is also supported in a report by Martin et al. (1998). Suzukawa et al. reported a case of acquired EAC atresia secondary to graft-versus-host disease (GVHD) in a leukemic patient after bone marrow

transplantation further supporting the theory of an immunologic factor contributing to the pathogenesis of this disease (Suzukawa et al., 2007).

Falqueto et al. described a case of acquired EAC stenosis that manifested as a paraneoplastic syndrome secondary to renal cell carcinoma (Falqueto et al., 2017). Histopathological studies taken from the auditory canal were suggestive of pyoderma gangrenosum, a non-specific finding and a diagnosis of exclusion that could occur secondary to chronic systemic inflammatory diseases (ulcerative colitis, Crohn's disease, rheumatoid arthritis, vasculitic disorders) and myeloproliferative disorders. On the other hand, acquired stenosis and atresia of the EAC have also been reported secondary to amyloidosis and fibrous dysplasia of the temporal bone (Álvarez-Ruiz et al., 2007; Jethanamest and Roehm, 2011). Nonetheless, diffuse canal involvement occurred in these cases, in contrast to medial canal involvement that is classically reported in medial meatal fibrosis. The relevance of such observation is yet to be determined.

2.2. The possible link to iron deficiency

Two cases of acquired atresia of the auditory canal in patients with chronic iron deficiency anemia and features of Plummer Vinson syndrome have been recently reported (Kmeid and Nehme, 2018). The strong correlation between external ear stenosis and good response to a systemic iron replacement therapy lead the authors to the assumption of a possible link between chronic stenosing external otitis and iron deficiency states.

Three possible contributing factors may be evoked regarding the origin of this inflammatory process in this context (Fig. 1): ***1) Impairment of secretory cell function*** caused by the anaemic state: This should be supported by an ultrastructural study of the auditory canal secretory system. Main et al. studied the human auditory canal skin with electron microscopy: Two types of secretory glands were observed: modified apocrine ceruminous glands and sebaceous glands. These glands are implicated in the enzymatic and immunological defense system of the external auditory canal (Main and Lim, 1976). ***2) Keratinocytes in the medial EAC, which express markers of hyperproliferation***, could be the target of iron deficiency. A study published in 1996 documented that the differentiation of the epidermis in various parts of the human ear canal materializes according to specific cytokeratin expression patterns (Vennix et al., 1996). Immunostaining studies revealed that the cartilaginous part had a profile characteristic of normal skin type differentiation whereas the deep EAC skin, including the tympanic membrane showed a peculiar type of differentiation with the presence of hyperproliferative cytokeratins (Vennix et al., 1996). These cells could be specifically affected by the iron deficiency state. ***3) A small amount of smooth muscle cells*** are found in the EAC. They are usually located at the hair line of the canal (junction between the cartilaginous and osseous parts of the EAC). Under sideropenic conditions, the depletion of iron-containing mitochondrial electron transport enzymes and iron dependent nitric oxide synthase may alter the contractile function of these cells targeting a cascade of inflammatory reactions (Dallman, 1986).

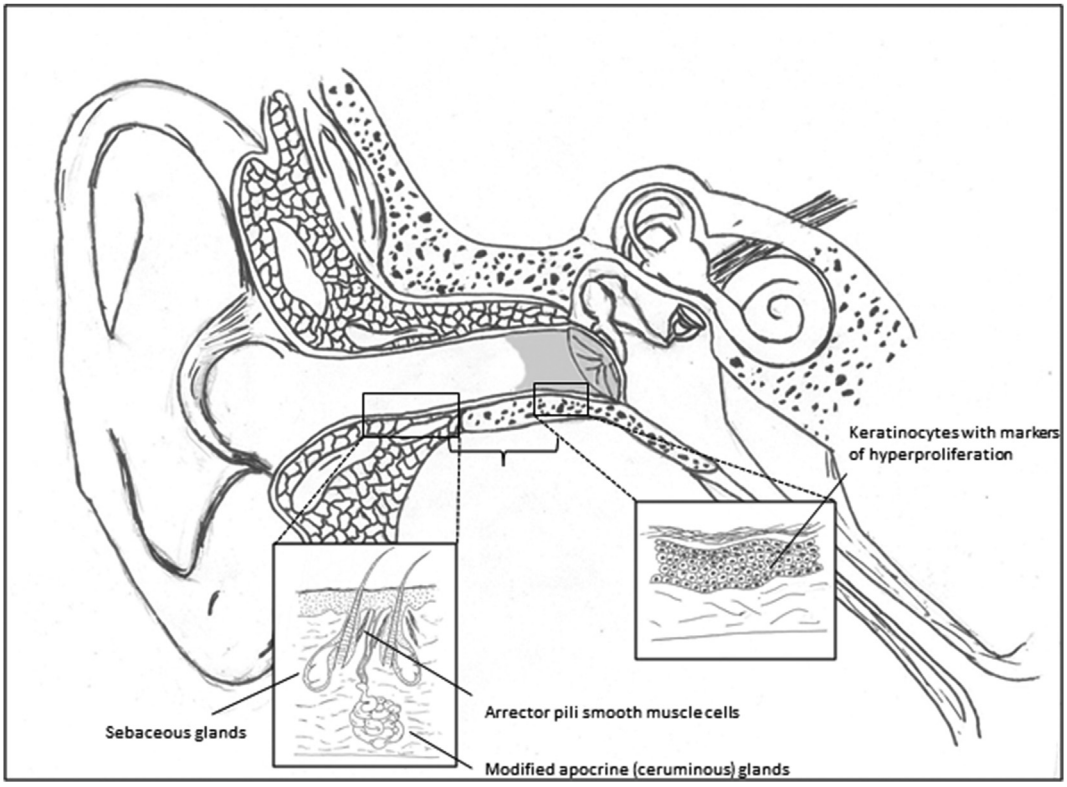

Fig. 1. Schematic representation of the anatomy of the external auditory canal with depiction of 3 potential targets for the inflammatory process. In medial meatal fibrosis, the medial canal is occupied by a dense fibrotic plug (grey shaded area) with the inflammatory process extending into the epithelial layer of the tympanic membrane. The skin of the lateral cartilaginous canal is thick with abundant *secretory glands* (ceruminous and sebaceous) and hair follicles. *Smooth muscles cells* can be found at this level mainly around hair follicles (arrector pili muscles) and are most abundant at the hair line (junction cartilaginous-bony canal). Secretory glands and smooth muscle cell can be susceptible to systemic immune-mediated or metabolic derangements. On the other hand, the skin of the bony canal is thin and glabrous and is devoid of submucosa and secretory glands. *Keratinocytes with markers of hyperproliferation* can be found at this level and may play an important role in the pathogenesis of post-inflammatory auditory canal atresia. At the transition zone, a key area in the formation of the atretic plate, ultrastructural features of both the medial and lateral canal can be found.

3. Clinical presentation

Two distinct stages of the disease can be identified (Lavy and Fagan, 2000). First, a wet phase develops where the ear canal is in a state of episodic inflammation that subsequently heals over with progressive fibrosis and stenosis. Patients usually complain of recurrent otorrhea and sensation of aural fullness. Bacterial and fungal cultures from the auditory canal usually yield the growth of non-specific micro-organisms that are commonly isolated in chronic otitis media and external otitis. The second phase, the dry phase, is characterised by a non-discharging stenotic ear with dense fibrosis. At this stage, affected patients mainly present with conductive hearing loss and stenotic yet dry auditory canals. Histological examination of the stenotic tissue usually reveals fibrosis with numerous blood vessels and a non-specific chronic inflammatory infiltrate (Lavy and Fagan, 2000). It is worthy to mention that EAC stenosis of any cause may lead and manifest similar to EAC cholesteatoma (White et al., 2012). This occurs secondary to inefficient evacuation of desquamated keratin debris.

4. Management

Management of acquired post-inflammatory atresia of the EAC is somehow controversial especially with regard to timing of surgery, the surgical technique used and the long-term surgical outcomes. Traditionally, when the patient is still in the wet phase, medical therapy with regular aural toilet and topical antibiotic/steroid ointments is believed to be the treatment of choice with surgery reserved for patients with non-discharging ears during the dry fibrotic stage. However, some authors believe that topical treatments are often inefficient with an inevitable progression to fibrosis and atresia (Becker and Tos, 1998; Slattery and Saadat, 1997; Keohane et al., 1993). In a study by Ghani et al., early surgical treatment during the wet, granular phase is advocated as it often alleviates from the repetitive and prolonged ear discharge (Ghani and Smith, 2013).

The surgical approach to acquired atresia of the EAC was first described by Paparella and Kurkjian in 1966 (Paparella and Kurkjian, 1966). Several minor modifications were applied later on to improve post-operative results but the basic principle remains the same: complete excision of the atretic plate along with the skin of the medial canal and the epithelial layer of the tympanic membrane leaving the fibrous lamina intact as possible, generous widening of the bony canal until the first mastoid cells are encountered, relining the bare bone of the canal using skin grafts and meticulous packing of the canal to ensure adequate graft reception (Lavy and Fagan, 2000). A retroauricular or an endoaural approach may be used, though, some authors believe that superior surgical results are achieved using a post-auricular technique (Jacobsen and Mills, 2006). Packing can be done using silastic sheets, gelfoam, bismuth iodine paraffin paste or antibiotic soaked gauze (Ghani and Smith, 2013). Additional meatoplasty is recommended to facilitate the lateral migration of cerumen and desquamated keratin debris (Bajin et al., 2015).

There is some debate regarding what should be used as a graft to cover the bony canal. Split thickness skin grafts are most commonly used; nonetheless, some prefer using full thickness grafts as they are less likely to contract minimizing the risk of restenosis (Moore et al., 1984). Adkins and Osguthorpe used transposition flaps to cover the canal in 8 cases with no recurrences (Adkins and Osguthorpe, 1981). Some authors prefer using preauricular or retroauricular pedicled skin flaps (Bell, 1988; Nagaoka et al., 2016; Stucker and Shaw, 1991). Stucker and Shaw advocate for the use of posteriorly based pedicled flaps as they are well vascularized and they exert a posteriorly directed traction vector if they contract maintaining the patency of the canal during the healing process (Stucker and Shaw, 1991). The most important step during surgery is a complete visualization of the anterior fibrous annulus with exposure of the anterior tympanomeatal angle (Becker and Tos, 1998). This angle is described by Tos and Balle as a key area to access in order to completely excise the fibrous plug and prevent restenosis (Tos and Balle, 1986).

There are few publications that tackle the benefit of surgery and long-term surgical results. Clearly, a satisfactory post-operative outcome involves resolution of symptoms and closure of the pre-operative air-bone gap (ABG). The largest study in this regard was published by Becker et al., in 1998. Although they report an overall satisfactory long-term benefit from surgery, the recurrence rate was 11% and the percentage of patients with a post-operative ABG <20 dB diminished from 90% at primary follow-up to 61% at 5 years (Becker and Tos, 1998). Another study by Ghani et al. concluded that the long-term results of surgery are generally poor especially when performed during the dry phase; they suggested seeking other alternatives such as osteo-integrated implants for restoration of hearing (Ghani and Smith, 2013). A recent meta-analysis by Keller et al. also corroborates this conjecture stating that although current data supports the efficacy of surgery on the short-term, there is a statistically significant decline in hearing gain over time questioning the long-term prospects of surgical treatment (Keller et al., 2017).

The less than optimal surgical outcomes are mainly related to the most frequently reported postoperative complication: restenosis (Ghani and Smith, 2013). Multiple factors are hypothesised as possible causes for recurrent atresia and hearing deterioration with time. Early restenosis is usually the result of a faulty surgical technique with incomplete removal of the fibrous plug or inadequate coverage of the bare bone (Becker and Tos, 1998; Slattery and Saadat, 1997; El-Sayed, 1998). In fact, exposed bone will granulate leading to fibrosis and stenosis (Slattery and Saadat, 1997; El-Sayed, 1998). Additionally, applied skin grafts harvested from the upper arm or retroauricular area lack the physiological quality of the auditory canal skin with absent sebaceous and ceruminous apocrine glands (Jacobsen and Mills, 2006). Allowing these free grafts to heal by secondary intention may result in scarring and contractures (Stucker and Shaw, 1991). Late restenosis have been reported to occur as far as 9 years post-op (Slattery and Saadat, 1997) and is usually the result of the ongoing inflammation in the auditory canal (Keller et al., 2017). Less common surgical complications include persistent otorrhea and tympanic membrane perforations (Ghani and Smith, 2013). Ghani et al. attributes the persistence of continuous ear discharges and wet canals post-operatively to probable chronic dermatologic conditions that interferes with a normal skin healing (Ghani and Smith, 2013).

Interestingly enough, none of the publications found in the literature addresses the possibility of an underlying systemic etiology when managing this condition. Acquired atresia of the EAC is often regarded as a loco-regional disorder and it is managed as such. Yet, when we consider the possibility of an underlying systemic cause, the rationale supporting current therapeutics would change, aiming to correct, when present, culprit underlying general conditions such as immune mediated diseases or nutritional deficiencies (i.e. iron deficiency). Obviously, no extensive workup is warranted when the etiology is clear on clinical basis (traumatic, post-operative, recurrent otitis). Appropriate radiological and pathological studies are recommended when a neoplasia is suspected. In cases where the cause remains unknown, we suggest performing a complete metabolic panel as well as an ANA screen. A biopsy from the EAC prior to surgical management is warranted if initial lab tests are unyielding. Nevertheless, Future studies are necessary indeed in order to clarify this association and test the

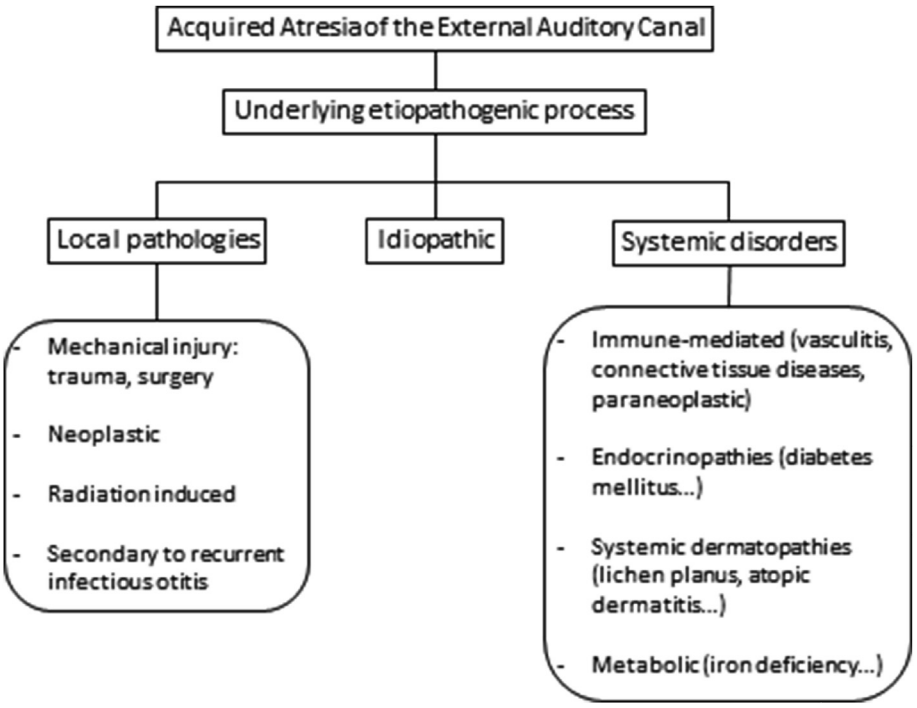

Fig. 2. Acquired atresia of the external auditory canal can be classified into 3 categories according to the underlying etiologic process. (1) Local pathologies affecting solely the EAC include mechanical injury (trauma or post-surgical), neoplastic (temporal bone and EAC benign and malignant tumors), radiation induced or local inflammation and fibrosis secondary to recurrent bouts of infectious otitis. (2) Systemic disorders may also affect the EAC though the exact underlying pathophysiologic mechanism is not fully understood and this includes immune mediated and collagen vascular diseases, endocrinopathies, systemic dermatopathies and metabolic disorders. (3) Some forms, however, remain idiopathic.

efficacy and long-term benefits of systemic treatment modalities as an adjunct to local therapies.

5. Conclusion

Post-inflammatory acquired atresia of the external auditory canal is a rare and unfathomable disorder. Numerous points warrant clarification apropos of its etiopathogenesis and the necessity to distinguish it from acquired canal stenosis. In the absence of large experimental studies and clinical trials and given the rarity of this relatively unfamiliar entity, these findings and correlations remain largely hypothetical. Nevertheless with this review, we propose adding to the traditional classification the possible association of this disorder to systemic immune-mediated or metabolic disorders (Fig. 2). With further supporting evidence, this extension of the well-known Tos classification may affect our understanding of otologic disorders in general and on the management of seemingly isolated external ear diseases.

Ethical considerations

All data collected are subjected to prior ethical approval ensuring the safety, rights and anonymity of all participants in this review. All procedures were followed in accordance with the responsible committee on human experimentation and with the Helsinki Declaration of 1975 and subsequent revisions.

Financial disclosure and conflict of interest

The authors whose names are listed above certify that they have NO affiliations with or involvement in any organization or entity with any financial or non-financial interest in the subject matter or materials discussed in this manuscript.

References

Adkins, W.Y., Osguthorpe, J.D., 1981. Management of canal stenosis with a transposition flap. Laryngoscope 91, 1267–1269.
Adler, M., Hawke, M., Berger, G., et al., 1985. Radiation effects on the external auditory canal. J. Otolaryngol. 14, 226–232.
Álvarez-Ruiz, S.B., Pérez-Gala, S., Aragüés, M., Fraga, J., García-Díez, A., 2007. Unusual clinical presentation of amyloidosis: bilateral stenosis of the external auditory canal, hoarseness and a rapid course of cutaneous lesions. Int. J. Dermatol. 46, 503–504.
Bajin, M.D., Yilmaz, T., Günaydin, R.Ö., Kuşçu, O., Sözen, T., Jafarov, S., 2015. Management of acquired atresia of the external auditory canal. J. Int. Adv. Otol. 11 (2), 147–150.
Becker, B.C., Tos, M., 1998. Postinflammatory acquired atresia of the external auditory canal: treatment and results of Surgery over 27 Years. Laryngoscope 108, 903–907.
Bell, D.R., 1988. External auditory canal stenosis and atresia: dual flap surgery. J. Otolaryngol. 17, 19–21.
Carls, J.L., Mendenhall, W.M., Morris, C.G., Antonelli, P.J., 2002. External auditory canal stenosis after radiation therapy. Laryngoscope 112, 1975–1978.
Carson, H.J., Krivit, J.S., Eilers, S.G., 2005. Metastasis of colonic adenocarcinoma to the external ear canal: an unusual case with a complex pattern of disease progression. ENT: Ear Nose Throat J. 85, 36–38.
Dallman, P.R., 1986. Biochemical basis for the manifestations of iron deficiency. Annu. Rev. Nutr. 6, 13–40.
El-Sayed, Y., 1998. Acquired medial canal fibrosis. J. Laryngol. Otol. 112, 145–149.
Falqueto, L.E., Kaddoum, M.L., De Miranda, M.M.L., Ramos, H.F., 2017. Acquired stenosis of external auditory canal secondary to paraneoplastic manifestation of renal cancer. Braz. J. Otorhinolaryngol. 84 (2), 249–251.
Ghani, A., Smith, M.C.F., 2013. Postinflammatory medial meatal fibrosis: early and late surgical outcomes. J. Laryngol. Otol. 127, 1160–1168.
Hopsu, E., Pitkäranta, A., 2002. Idiopathic inflammatory medial meatal fibrotizing otitis. Arch. Otolaryngol. Head Neck Surg. 128, 1313–1316.
Hopsu, E., Pitkäranta, A., 2007. Idiopathic, inflammatory, medial meatal, fibrotising otitis presenting with lichen planus. J. Laryngol. Otol. 121, 796–799.
Hopsu, E., Pitkäranta, A., 2008. Idiopathic inflammatory medial meatal fibrotizing otitis presenting in children. Otol. Neurotol. 29, 350–352.
Jacobsen, N., Mills, R., 2006. Management of stenosis and acquired atresia of the external auditory meatus. J. Laryngol. Otol. 120, 266–271.
Jethanamest, D., Roehm, P., 2011. Fibrous dysplasia of the temporal bone with complete canal stenosis and cholesteatoma. Otol. Neurotol. 32 (7), e52–e53.
Keller, R.G., Ong, A.A., Nguyen, S.A., O'Connell, B.P., Lambert, P.R., 2017.

Postinflammatory medial canal fibrosis: an institutional review and meta-Analysis of short- and long-term outcomes. Laryngoscope 127, 488–495.
Keohane, J.D., Ruby, R.R.F., Janzen, V.D., MacRae, D.L., Parnes, L.S., 1993. Medial meatal fibrosis: the university of western ontario experience. Am. J. Otol. 2, 172–175.
Kmeid, M., Nehme, J., 2018. Bilateral obliterative external otitis in the context of chronic iron deficiency – report of two cases. Oncol. Hematol. Rev. 14 (1), 42–44.
Lavy, J., Fagan, P., 2000. Chronic stenosing external otitis/postinflammatory acquired atresia: a review. Clin. Otolaryngol. 25, 435–439.
Luong, A., Roland, P.S., 2005. Acquired external auditory canal stenosis: assessment and management. Curr. Opin. Otolaryngol. Head Neck Surg. 13 (5), 273–276.
Main, T., Lim, D., 1976. The human external auditory canal, secretory system–an ultrastructural study. Laryngoscope 86 (8), 1164–1176.
Martin, L., Moriniere, S., Machet, M.C., Robier, A., Vaillant, L., 1998. Bilateral conductive deafness related to erosive lichen planus. J. Laryngol. Otol. 112, 365–366.
Michaelson, P.G., Lowry, T.R., 2005. Metastatic renal cell carcinoma presenting in the external auditory canal. Otolaryngol. Head Neck Surg. 133, 979–980.
Moore, G., Moore, I., Yonkers, A., et al., 1984. Use of full thickness skin grafts in canalplasty. Laryngoscope 94, 1117–1118.
Nagaoka, M., Noguchi, Y., Kawashima, Y., Ito, T., Koda, H., Kitamura, K., 2016. Long-term result of meatoplasty using inferiorly based retroauricular island pedicle flap for external auditory canal stenosis. Auris Nasus Larynx 43, 382–386.
Paparella, M.M., Kurkjian, J.M., 1966. Surgical treatment for chronic stenosing external otitis. Laryngoscope 76, 231–245.
Slattery 3rd, W.H., Saadat, P., 1997. Postinflammatory medial canal fibrosis. Am. J. Otol. 18, 294–297.
Stucker, F.J., Shaw, G.Y., 1991. Revision meatoplasty: management emphasizing de-epithelialized postauricular flaps. Otolaryngol. Head Neck Surg. 105, 433–439.
Suzukawa, K., Karino, S., Yamasoba, T., 2007. Surgical treatment of medial meatal fibrosis report of four cases. Auris Nasus Larynx 34, 365–368.
Tirelli, G., Nicastro, L., Gatto, A., Nata, F.B., 2015. Stretching stenosis of the external auditory canal: a report of four cases and brief review of the literature. Acta Otorhinolaryngol. Ital. 35, 34–38.
Tos, M., Balle, B., 1986. Postinflammatory acquired atresia of the external auditory canal: late results of surgery. Am. J. Otol. 7, 365–370.
Toynbeen, J., 1860. The Diseases of the Ear: Their Nature, Diagnosis and Treatment. Blancard and Lea, Philadelphia.
Vennix, P.P., Kuijpers, W., Peters, T.A., Tonnaer, E.L., Ramaekers, F.C., 1996. Epidermal differentiation in the human external auditory meatus. Laryngoscope 106 (4), 470–475.
White, R.D., Ananthakrishnan, G., McKean, S.A., Brunton, J.N., Hussain, S.S.M., Sudarshan, T.A., 2012. Masses and disease entities of the external auditory canal: radiological and clinical correlation. Clin. Radiol. 67, 172–181.