Acquired Stenosis of the External Ear Canal

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KEYWORDS

• Acquired stenosis external ear canal • Canalplasty • Meatoplasty • Skin grafts • Stents

KEY POINTS

- Acquired external ear canal stenosis can result from multiple causes such as infections, inflammation, accidental or iatrogenic trauma, benign or malignant tumors, and radiation therapy.
- Clinically, patients present with recurrent otorrhea, conductive hearing loss, cerumen impaction and even canal cholesteatoma.
- Surgery is the mainstay for treatment.
- Long term outcomes are unpredictable and range from successful recanalization with hearing improvement to restenosis with worsening conductive hearing loss.

INTRODUCTION

Acquired stenosis of the external ear canal (ASEEC) can result from many causes which initiate a cascade of inflammatory tissue reactions resulting in stenosis of the ear canal.[1] Stenosis or narrowing of the external ear canal (EAC) occurs lateral to the tympanic membrane resulting in a skin lined blind canal.[1] It typically presents as recurrent otorrhea, conductive hearing loss, often with significant cerumen impaction and ear canal cholesteatoma. Different etiologies such as chronic otitis externa, dermatologic diseases, iatrogenic (previous ear surgery or radiotherapy) or accidental trauma, tumor and inflammation have been implicated in its causation. ASEEC involve either the outer ear canal (eg. after trauma) or medial ear canal (eg post-inflammation) or both and can cause conductive hearing loss. History, clinical examination, audiometry, and Computed tomography (CT) scan help to confirm the diagnosis, extent of the stenosis and middle ear status. Diffusion weighted magnetic resonance imaging (DWI MRI) may be required to determine the presence and extent of cholesteatoma. Although medical treatment can be attempted, surgery is the primary modality for

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treatment. Several techniques excising scar tissue completely or partially, with or without skin grafting and with and without stenting of the ear canal have been described in literature. Recurrence with restenosis is the most common complication. Stable, long-term outcomes can be challenging in some patients.[2] This article reviews the historical background, prevalence, etiopathogenesis, medical and surgical management of this condition and discusses future trends.

HISTORY/BACKGROUND

- The earliest clinical description of postinflammatory ASEEC ("atresia) was published by Novick in 1939.[3]
- John Conley described "atresia" of the external auditory canal in 10 military personnel after gunshot wounds and other traumas in 1946.[4] He classified the stenosis as web type, solid fibrous healed type, solid fibrous and bony healed type and advocated an endaural approach, excision of scar tissue, application of a thin, split, free skin graft and packing the ear canal with rubber foam impregnated with sulfonamide or penicillin compound.[4]
- Proud in 1955, recommended the terminology "stricture" (Latin "stringere" meaning to "draw tight") for acquired ear canal closure, to avoid confusing the nomenclature of congenital ear canal agenesis or aplasia with the acquired abnormality[5] He also reported successful surgical corrections of acquired ear canal stenosis without prosthetic devices, using endaural approach, radical mastoidectomy, wide meatoplasty and split thickness grafts from the thigh to line the cavity.[5]
- In 1960, Gundersen and in 1965, Eichel and Simonton published case reports describing the treatment of ASEEC caused by chronic otitis externa.[6,7]
- In 1966, Paparella suggested removal of all diseased skin, widening the bony canal maximally with a dental bur, excision of skin with conchal cartilage and replacement with split thickness skin graft using microsurgical tympanoplasty techniques.[8] His report provided the first detailed description of the histology of the tissue excised from the stenosed ear canal. He reported that it showed subepidermal infiltrate of chronic inflammatory cells, predominantly histiocytes. A narrow band of condensed fibrous connective tissue separated the epidermis and the inflammatory cells. The overlying epidermis showed cellular edema and the dermal tissues appeared fibrotic and abnormally hyalinized.

PREVALENCE/INCIDENCE

ASEEC (**Fig. 1**) is not a common condition. However, most otolaryngologists encounter it in their practice. In the largest series of patients treated for acquired ear canal stenosis, an incidence of 0.6 cases per 100000 was reported.[9] Most studies report a female preponderance.[2,10–13] Another common finding is the presence of bilateral disease in patients with ASEEC.[10] The average age at presentation is in the fifth decade although 2 cases have been reported in children.[14,15] The incidence of ear canal cholesteatoma in ASEEC varies from 6% to 9%.[2,9,16] Chronic infection was observed to be the leading cause of ASEEC, followed by postsurgical trauma and accidental trauma.[2,16]

NOMENCLATURE

Acquired stenosis of the external ear canal has been described variously by different authors as "EAC atresia"[9,17–19], "medial meatal fibrosis"[20], acquired medial canal

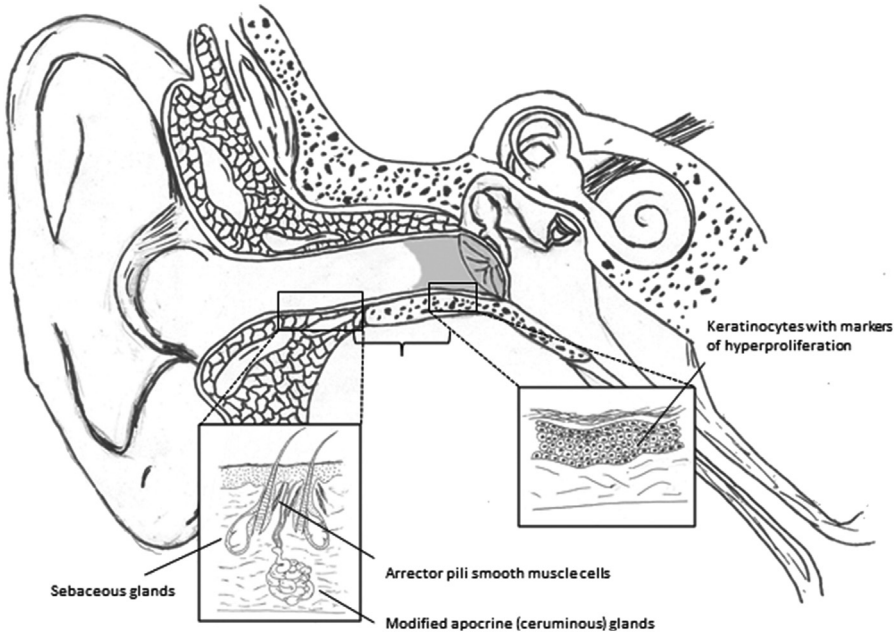

Fig. 1. Acquired stenosis of external ear canal (ASEEC). (*From*: Kmeid M, Nehme J. Post-inflammatory acquired atresia of the external auditory canal. Journal of Otology 2019;14(4): 149–154, see Fig. 1.)

fibrosis," "EAC stenosis"[21] and obliterative otitis externa[22,23] representing the different conditions that are caused in this disease process.

ETIOLOGIES AND PATHOGENESIS

In 1943, Conley described 10 cases of posttraumatic ear canal stenosis and classified them into 4 groups: 1. Web type; 2. Solid fibrous healed type; 3. solid fibrous and bony healed type and 4. infected fibrous and bony type.[4]

Tos and Balle classified ASEEC in 1986 and Lavy and colleagues reported it in 2000.[12,15] (Table 1). Subsequently various authors sub-classified the condition based on the etiology, as idiopathic, postinflammatory, posttraumatic, iatrogenic (postsurgery or postradiation), neoplastic or dermatologic.[12,20,24–30] (Table 2).

Postinflammatory ASEEC accounts for more than 50% of the cases.[2,16] It is a sequelae of chronic otitis media or externa or inflammatory dermatological conditions.[10]

Post-traumatic ASEEC: Accidental blunt trauma such as motor vehicle accidents, penetrating injuries (gunshot wounds) with ear canal lacerations, chemical or thermal burns, fractures involving bony ear canal or posterior displacement of fractures of the head of the mandible can lead to loss of the epidermal lining of the ear canal.[9,12] Circumferential loss of the epidermis heals with granulation tissue formation, followed by fibrosis and subsequent stenosis of the ear canal(Fig. 2).[31] Hearing aid molds can cause chronic irritation of the outer ear canal skin, interfere with the normal lateral epithelial migratory pattern of cerumen clearance.[1] The chronic inflammation and lead to chronic otitis externa and eventually lead to stenosis.[32] ASEEC can occur after ear surgery if ear canal skin has been extensively dissected and the periosteum is inadequately covered with the tympanomeatal flap.[33] Overlay myringoplasty with

Table 1
ASEEC classification after Tos et al and Lavy et al[12,15]

1	Post-traumatic atresia	Severe direct trauma, ear contusion, fractures of the anterior wall of the external auditory meatus with the displacement of the mandibular neck
2	Postoperative atresia	Failed meatoplasty postcanal wall down mastoidectomy, severe blunting of the tympanic membrance after myringoplaty
3	Neoplastic atresia	Occlusion of the ear canal by malignant tumor
4	Postinflammatory atresia	Fibrous obliteration of the ear canal following infection, chronic otitis media with otorrhea or external otitis associated with use of hearing aids

lateral blunting has also been reported to be associated with acquired ear canal stenosis.[31,34] Keloids and hypertrophic scars at the site of the ear canal incision are rare causes of *post-ear* surgery canal stenosis.[31,34]

Surgery for benign tumors of the ear canal such as osteomas and exostoses or malignant tumors of the ear canal can cause acquired ear canal stenosis. Most cancers of the external ear canal are primary cancers and metastatic tumors are rare. Cutaneous malignancies such as basal cell carcinoma, squamous cell carcinoma and melanoma are found in the ear canal, although tumors of the ceruminous ear canal glands can also occur.[35] Metastatic tumors from primaries in the lung, breast and kidneys involve the temporal bone due to its vascularity but direct metastasis to the ear canal have also been reported.[35–37]

Surgical treatment of ear canal tumors with circumferential dissection can result in stenosis of the ear canal. Carls and colleagues[30] reported that high dose external beam radiation alone does not predispose patients to external ear canal stenosis. However, there is an increased risk of ear canal stenosis after combined high dose external beam radiation therapy and surgery around the ear canal.[30,38] Other authors have described radiation related osteoradionecrosis of the bony ear canal, sequestration, inflammation of the soft tissue, fibrosis with resulting external otitis and stenosis.[33,39]

Case reports describe external ear canal stenosis resulting from dermatological conditions such as lichen planus, and epidermolysis bullosa affecting the ear canal.[26,29,40] Hopsu and colleagues, postulated an association between auto-immune

Table 2
Modified sub-classification of ASEEC based on etiology[9,15,20]

1	Idiopathic	
2	Postinflammatory	Chronic external otitis due to chronic otorrhea, hearing aid usage
3	Postaccidental trauma	Blunt or penetrating trauma, posterior displaced mandibular head fractures
4	Postiatrogenic trauma	Postear canal surgery for canal wall down mastoidectomy, blunting after overlay myringoplaty, circumferential ear canal surgery for osteoma or exostoses removal, keloids or hypertrophic scars at site of ear canal incisions, radiation of ear canal for parotid, ear canal tumors
5	Neoplastic	Ear canal cutaneous malignancies such as melanoma, basal cell or squamous cell cancers
7	Dermatologic	Lichen Planus, Epidermolysis bullosa

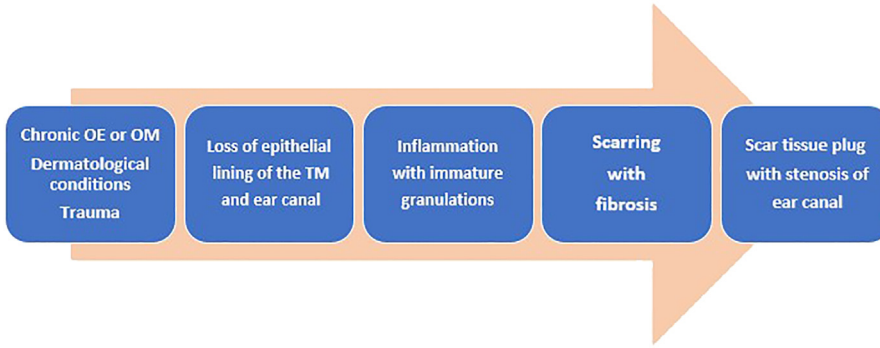

Fig. 2. Pathogenesis of ASEEC -inflammatory cascade.

disorders and medial meatal fibrosis as both lichen planus and ear canal stenosis affect the glabrous skin of the auditory canal and tympanic membrane.[24] Kmeid and colleagues reported acquired ear canal stenosis in two patients with chronic iron deficiency anemia and features of Plummer Vinson syndrome.[41] As the patients responded to systemic iron replacement treatment with improvement in their stenosing external otitis, they suggested a link between iron deficiency anemia and acquired ear canal stenosis.[33]

Most authors *agree that in medial ear canal stenosis* an initial inflammatory process, irrespective of the etiology, triggers loss of the squamous epithelial lining of the medial ear canal. Granular myringitis with the de-epithelialization of the lateral surface of the TM and exposure of the fibrous layer has also been proposed to trigger the inflammatory cascade.[15] Inflammation leads to the production of immature granulation tissue which ultimately matures and forms a firm fibrous plug. The earliest histological description of the tissue was by Paparella.[8] In the absence of inflammation, for example, in post-traumatic cases, the initial trigger is lateral to the tympanic membrane, and heals with the formation of a fibrous, atretic plate, with the entrapment of squamous epithelium and subsequent formation of ear canal cholesteatoma.[42] Bonding and Tos[13] proposed that granulation tissue is formed due to localized inflammation in an isolated area of the ear canal. Epithelialization on either side of the granulation tissue results in membranous atresia and if left untreated, can progress to the formation of a fibrous plug.[13] Kmeid and colleagues have proposed a possible link to iron deficiency anemia and have reported a good response to systemic iron replacement therapy.[33,41]

Lavy and colleagues[15] described 2 stages in the pathogenesis of ASEEC.

- *Primary or Wet stage*: characterized by episodic inflammation, granulations, and recurrent drainage. Organisms such as Pseudomonas and Proteus have been cultured from the drainage. Eventually the granulations heal with fibrosis and stenosis.
- *Secondary or Dry stage* characterized by a thick fibrous plug, with stenosis of external ear canal.

CLINICAL PRESENTATION AND ASSESSMENT

- Hearing loss
- Aural fullness
- Chronic otitis externa

Patients typically present with recurrent otorrhea and progressively worsening hearing loss. There may be discharge with breakdown of the central part.[15] Culture of the discharge shows Pseudomonas and Proteus which are not specific as they are often present in otitis media and chronic otitis media.[14]

A common finding in outer ear canal stenosis is a narrowing of the external meatus, while in medial ear canal stenosis, there may be a solid plug of fibrous tissue occluding view of the tympanic membrane.

Although the condition is typically unilateral, 10-67% of the cases can be bilateral[14,43]

Ear canal cholesteatoma with bone resorption and widening of the floor of the ear canal wall medial to the outer ear canal stenosis has been reported by several authors.[9,17]

Pure tone audiometry shows conductive hearing loss of approximately 30-40dB air bone gap, flat tympanogram and absent stapedial reflexes.[15] Bonding and Tos reported progression of the hearing loss from 15dB to 40dB over 8 years.[13]

IMAGING

High Resolution Computerized tomography (HRCT) scan is most informative for the assessment of the bony ear canal.[44] It helps to evaluate ASEEC, keratosis obturans, cholesteatoma, status of middle ear cavity and ossicles. Typically, the scan will show (**Figs. 3** and **4**) aerated middle ear space, with soft tissue occupying and narrowing the external auditory canal. Traditional and diffusion weighted magnetic resonance imaging is useful to detect concomitant ear canal cholesteatoma.

TREATMENT

Medical management has been supported by Stoney and colleagues,[45] however most of the authors agree that the surgical treatment of ASEEC forms the mainstay of managing the condition.[1,8,12,15]

MEDICAL TREATMENT

Medical management has a limited role, and the goal is to control infection, and prevent progression of granulation tissue to the mature stage. Medical treatment includes aural toilet, topical antibiotic drops, and stenting of the ear canal.

- Although frequent aural toilet is recommended, repeated trauma to the EAC can worsen the condition. Therefore, Luong and colleagues suggested a “no-touch” technique” during aural toilet.[1]

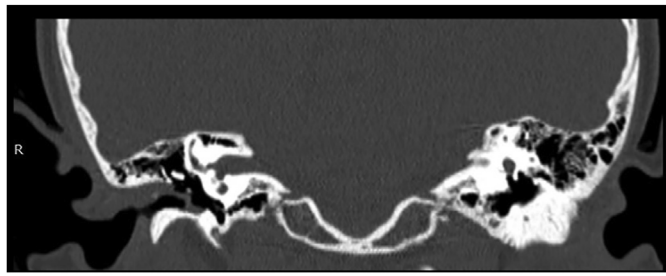

Fig. 3. Coronal CT showing right ear with ASEEC and normal middle ear space.

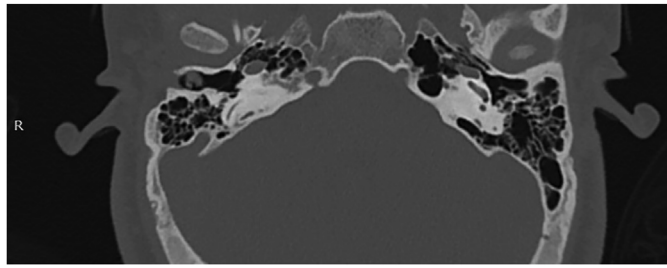

Fig. 4. Axial CT scan showing ASEEC Right ear with cerumen (*white arrow*) plug medial to the stenosis.

- Topical antibiotic drops or powders can be useful to control infection. Luong and colleagues support the use of powders as they adhere to wet surfaces and deliver more stable drug levels.[1]
- Stenting the ear canal with a non-expanding gauze or expanding cellulose wick is typically performed. Miller suggested the use of a soft, expanding cellulose wick to create steady pressure and dilate the ear canal.[46] However, Luong and colleagues observed that a wick has the potential of worsening the inflammatory process due to constant friction with the inflamed EAC.[1]
- Caffier and colleagues proposed using an ear wick with 0.1% tacrolimus ointment every 2nd or 3rd day as an alternative to steroids for the treatment of therapy-resistant otitis externa.[47] They reported clear improvement in 85% of patients short term and complete healing in 46% of their patients at follow-up of 10 -22 months. Magliulo observed that tacrolimus prevented progression of persistent chronic inflammation to mature fibrotic stenosis in his small group of patients with ASEEC.[2]

SURGICAL TREATMENT

Most authors agree that surgical management is necessary to address ASEEC.[8,9,11,15,34,48] Ghani and colleagues recommend surgery in the early wet, granular stage to prevent recurrent ear drainage and disease progression.[49]

Indications for surgery are.

- Failed medical management.
- Progression of the condition to conductive hearing loss

Steps of surgical treatment[15] are.

- Excision of the fibrous plug with the preservation of the fibrous layer of the TM
- Circumferential widening of the ear canal
- Covering the bare bone of the EAC with skin flaps
- Addressing the external ear canal cholesteatoma concomitantly if present.
- Packing of the EAC to maintain patency.

SURGICAL TECHNIQUES

One of the earliest descriptions of surgery for ASEEC was by Conley in 1946. He reported surgical removal of the ear canal plug with recanalization, split thickness skin grafting and sponge packing in 10 young combat soldiers without previous history of ear disease.[4] Paparella and colleagues, in 1966, described postaural approach, canalplasty with circumferential widening of the bony canal using an electric drill

followed by a split thickness skin graft.[8] Surgical principles described by Conley[4] and later by Paparella and colleagues[8] have survived the test of time and continue to be used even today, albeit with variations.

Both endaural[31] as well as postaural[31] approaches have been described, although Jacobsen and colleagues[34] reported improved outcomes with the postaural approach. A wedge resection of the fibrous plug was proposed by Soliman and colleagues[50] to prevent postoperative restenosis. However complete excision of the fibrous plug is recommended by most surgeons.[9,13,17,20,43] The fibrous plug is dissected lateral to medially up to the level of the tympanic membrane (TM).[10] The epithelial layer of the TM is denuded, leaving the fibrous layer intact. Generous widening of the bony canal (canalplasty) is performed, meticulously avoiding entering the mastoid air cells or damaging the facial nerve and the temporomandibular joint.[21] Ideally, canalplasty should allow unobstructed view of the tympanomeatal angle and this angle should be at least 90 degrees to prevent scarring.[12] Meatoplasty can be performed at this time to widen the cartilaginous ear canal.

There is consensus about lining the bare ear canal bone, however there is lack of consensus about the best graft to be used. Surgeons have covered the bare ear canal with meatal flaps[51] or regional flaps[51–53] or split (STSG)[34] or full thickness (FTSG)[54] skin grafts. Although FTSG is durable and contains glandular elements,[54] STSGs are preferred by most authors as they are less bulky and have a higher acceptance rate.[4,17,43,55] Some surgeons prefer to harvest the grafts from the arm while others prefer harvesting from the retroauricular area. Depending on the location of the ear canal stenosis, McCary and colleagues[55] used island, segmental or circumferential split thickness skin grafts with excellent long-term results. Adkins and Osguthorpe[52] used transposition flaps in eight cases and reported no recurrences. Retroauricular or preauricular pedicled skin flaps are preferred by some authors.[53,56,57] A well vascularized pedicled flap is associated with a higher rate of graft uptake according to Dhooge and Vermeersch.[58]

Finally, the ear canal is packed to maintain patency. Depending on the surgeon's preference various material have been used for packing and these include gelfoam,[10] or a "Swiss roll" (silastic sheeting with bismuth iodoform soaked ribbon gauze),[34] backing strips, expandable wicks, tracheostomy tube,[59] Foley's catheter,[50] dental impression material,[60] acrylic stents,[61–63] silicon tubes,[19] and drug eluting stents.[64] Matin-Mann et al have proposed a 3D printed, individualized, drug-eluting (dexamethasone, ciprofloxacin, TNF-alpha) external ear canal implant (EECI) to prevent restenosis.[64] Most surgeons remove the packing material after one week, although Soliman and colleagues[50] advocated removal after 6 weeks. The packing material helps to keep the skin graft in place but can completely occlude the external ear canal with resultant hearing loss and can also be a nidus for infections. After removal of the ear canal stent, if the canal appears inflamed, Luong and colleagues recommend placing a Pope wick.[1]

In a retrospective study of 24 patients with ASEEC, Bajin and colleagues[31] reported that the atretic plate was located at the bony-cartilaginous junction in 37.5%, in the cartilaginous canal in 33.3% and rarely in the bony canal. Jacobsen and Mills reported that simple surgical excision of the fibrous plug was inadequate. It was associated with 100% failure in patients studied over a period of 18 years and the use of a split skin graft resulted in a patent ear canal in 70% of the cases.[34]

Battelino and colleagues[65] reported intraoperative application of 1 mL of 0.4 mg/mL of mitomycin C for 4 minutes to the EAC to prevent adhesions and restenosis in a small prospective study. One patient required reapplication postoperatively. Of their 6 ears, 83.3% had adequate patency and improvement in ABG to 10dB or less between 3 and 14 months postop.

CLINICAL OUTCOMES

A successful postoperative outcome is the complete resolution of symptoms with closure of the air-bone gap. In one of the earliest postoperative outcome studies, Becker and Tos compared short- and long-term outcomes. They reported 90% of the patients had an initial air bone gap (ABG) of less than 20dB but this decreased to 61% after two years and 11% of patients had recurrence of ASEEC.[9] The authors also observed that cases with concomitant cholesteatoma had a problematic postoperative course.[9] Irrespective of the surgical technique used, Jacobsen and colleagues[34]'s 18-year follow-up showed recurrence in 21% within one to two years. They observed that 79% of patients had improvement in hearing with decrease in air bone gap from 29 to 17dB on average. However, this hearing improvement decreased over time.[34] In 2009, Magliulo compared his outcomes at twelve months postsurgery and five years postsurgery. He reported that excellent ABG results (ABG 0 to −10dB) decreased from 36% at 6-month follow-up to 12% at 5 years; and failures (ABG >30dB) increased to 24% at 5 years.[2] He concluded that irrespective of the correct surgical procedure, stable, long-term outcomes are unlikely in some patients. Ghani and colleagues[49] reported recurrent otorrhea in 56% of patients, restenosis in 33% and reoccurrence of conductive hearing loss five years after the surgical treatment of ASEEC. Keller and colleagues[10] have proposed 2 reasons for restenosis: 1. Incomplete excision of stenotic fibrous plug or inadequate coverage of denuded bone with skin grafts and 2. Persistent inflammatory disease in the ear canal. Concomitant middle ear disease or conductive deafness secondary to progressive tympanosclerosis after surgical procedure or previous inflammatory disease has been proposed as a cause of the conductive hearing loss in patients with ASEEC.[15] In the absence of a way to predict which patients will develop restenosis or have persistent hearing loss, there is a consensus that patients with ASEEC will require consistent long-term follow-up.[1] Slattery and colleagues[20] reported restenosis 9 years after surgery.

FUTURE DIRECTIONS

ASEEC treatment continues to be challenging. A few reports suggest limited success with topical tacrolimus ointment-soaked wick placement in the ear canal to prevent progression of the disease. Drug eluting stents have been proposed by Matin-Mann et al as proof of concept and may offer hope for ASEEC patients in future. Further research in stents eluting fibroblast activity inhibitors or similar agents in the wet stage of the disease to prevent inflammatory granulations from progressing to fibrosis is needed. Additionally, the development of drug eluting stents for long-term postsurgery use may help to prevent restenosis and worsening of the hearing loss.

SUMMARY

- Chronic otitis externa followed by surgical trauma, tumors, dermatologic conditions, and radiation treatment of head neck cancers can trigger inflammatory cascade leading to ASEEC.
- Complete excision of the stenotic ear canal plug, canaloplasty with or without meatoplasty, re-epithelialization of the EAC with STSGs and stenting of the ear canal are the mainstay of treatment.
- Meticulous postoperative care and prolonged follow-up are necessary.
- Long term clinical outcomes are unpredictable. Restenosis and persistent conductive hearing loss have been reported up to nine years after surgery

CLINICS CARE POINTS

- ASSEEC can involve either bony outer ear canal, medial cartilaginous ear canal or both.
- Aetiology can vary from trauma, tumor to inflammation.
- Clinical examination, Audiometry and CT scan help to diagnose the condition, its extent and degree of hearing loss.
- Surgery forms the mainstay of treatment.

DISCLOSURE

Nothing to disclose.

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