

A rare cause of conductive hearing loss: Posttraumatic external auditory canal atresia

Nadir görülen bir iletim tipi işitme kaybı sebebi: Posttravmatik dış kulak yolu atrezisi

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Abstract

Acquired external auditory canal atresia is an uncommon entity which can arise from a number of different causes including infection, trauma, neoplasia, inflammation and radiotherapy. Posttraumatic atresias are extremely rare, only 10% of atresias are attributed to trauma in most of the series. The presence of canal atresia causes associated morbidity, patients generally suffer from conductive hearing loss and persistent otorrhea. Surgery is the treatment of choice for posttraumatic atresias. The aim of surgery is to produce a dry, patent ear canal by removing the atretic segment and enlarging the bony canal. In this paper, we present a case of posttraumatic external auditory canal atresia and accompanying tympanic membrane perforation managed successfully 22 years after trauma.

Keywords: External ear canal, atresia, conductive hearing loss, canalplasty

Özet

Akkiz dış kulak yolu atrezisi enfeksiyon, travma, neoplaziler, inflamasyon ve radyoterapi gibi sebeplere bağlı gelişebilen nadir bir durumdur. Posttravmatik vakalar oldukça nadirdir, yapılan çalışmalarda akkiz dış kulak yolu atrezilerinin sadece %10 kadarının travma sonrası geliştiği saptanmıştır. Dış kulak yolu atrezisi iletim tipi işitme kaybı ve persistan otore gibi şikayetlere yol açabilir. Tercih edilmesi gereken tedavi yöntemi cerrahidir. Cerrahinin amacı atretik kısmı eksize edip kemik kanalı genişleterek kuru ve açık bir dış kulak yolu elde edilmesidir. Bu çalışmada, 22 yıl önceki travma hikayesi sonrası gelişen dış kulak yolu atrezisi ve eşlik eden timpanik membran perforasyonu olan bir hasta ve tedavisi sunulmaktadır.

Anahtar sözcükler: Dış kulak yolu, iletim tipi işitme kaybı, kanalplasti

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Introduction

Acquired external auditory canal (EAC) atresia is an uncommon entity. It can arise from a number of different causes including infection, trauma, neoplasia, inflammation and radiotherapy [1]. Its incidence has been estimated as 0.6 cases per 100.000 inhabitants, as reported in the largest reported series of patients treated for EAC stenosis [1]. EAC atresia results in a blind skin-lined canal just lateral to the tympanic membrane and commonly presents as conductive hearing loss. This situation could either caused by a congenital malformation of the external auditory canal or it may be acquired. The intervening segment between the medial aspect of the patent EAC and tympanic membrane usually consists of fibrous plug. This entity has been referred to by several

different terms including medial meatal fibrosis, EAC atresia, and medial canal or EAC stenosis [1-5]. The most common cause of acquired EAC atresia is chronic otitis externa, hence most of the EAC atresia cases presented in the literature are postinfectious, posttraumatic cases are extremely rare [6, 7]. Whatever the cause is, atresia of the external auditory canal is a situation which is very difficult to manage, and recurrence rate is very high after treatment. Paparella and Kurkjain introduced the basic surgical principles of excising the fibrous plug, enlarging the cartilaginous and bony canal, and following this, re-covering the canal [8], Since then, modifications on this technique have been introduced primarily to prevent the most common postoperative complication, restenosis.

In this report, we present a case of posttraumatic external auditory canal atresia and accompanying tympanic membrane perforation managed successfully 22 years after trauma.

Case report

A 27 year old female patient attended to our outpatient clinic with the complaint of unilateral hearing loss for over 20 years in her left ear. She had a history of falling down from three metres high on her left side when she was 5 years old. She realized a mild hearing loss just after the trauma, and her hearing loss worsened gradually in 6 months. Her hearing loss stayed stable since then. She had no history of otorrhea or otalgia. In otoscopic examination, left EAC was found to be completely atretic. The pure tone audiometry showed 40 dB conductive type hearing loss of left ear (Figure 1). Temporal computerized tomography (CT) scan showed fibrous plug with a thickness of approximately 10 mm. in the left EAC (Figure 2). There was no cholesteatoma seen in the CT scan, and the middle ear cavity and ossicles were appeared to be normal. Surgery was planned to remove the stenotic segment of the EAC, to enlarge the bony ear canal and to recreate an epithelial lined EAC with a split thickness skin graft. First, we excised the soft tissue plug via an endaural incision and we enlarged the bony part of the ear canal circumferentially with the help of cutting and diamond burrs. After enlarging the EAC, we realised the subtotal perforation of the tympanic membrane. We performed underlay myringoplasty with temporalis muscle fascia to fix this situation. Then we harvested split thickness skin graft sized approximately 2*1 cm. from retroauricular region and we placed on denuded bony EAC. The graft was stabilized in place with small wedges of silicone rubber (Silastic sponge). These sponges were removed three weeks after surgery.

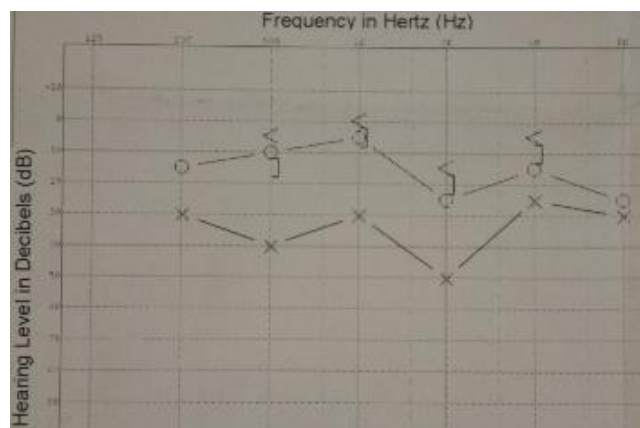


Figure 1. Preoperative audiogram of the patient.

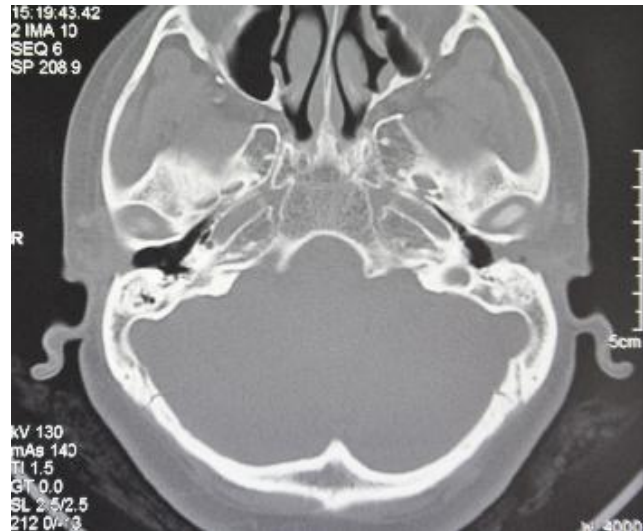


Figure 2. Temporal CT showing left external ear canal atresia.

At the 18th month follow up, the patient's tympanic membrane was intact and there was no sign of restenosis on EAC (Figure 3). The pure tone audiometry showed an improve in hearing with a closure of the air-bone gap to within 20 dB (Figure 4).



Figure 3. Postoperative 18th month view of the external ear canal and tympanic membrane.

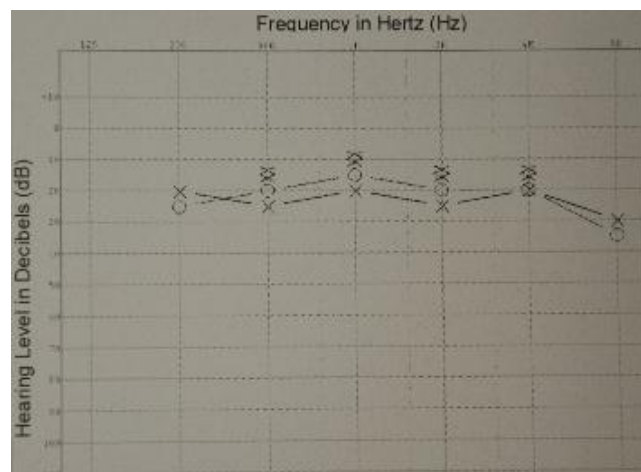


Figure 4. Postoperative 18th month audiogram of the patient.

Discussion

EAC atresia can be congenital or acquired. The most common cause of acquired EAC atresia is otitis externa. Chronic inflammation of the EAC results in a subepithelial infiltration of inflammatory cells, and this inflammatory process results in fibrotic changes to the canal [9], leading to EAC stenosis. Trauma is another possible cause of EAC atresia. Iatrogenic trauma from prior otologic surgery is a common inciting event [10], but direct trauma to EAC is a rare cause of atresia. In one of the largest series on this subject, there was only one acquired EAC atresia due to previous trauma in a total of 49 patients [7]. Selesnick et al. [4] reviewed 15 reports on this topic and they reported that chronic infection was the leading cause of this disorder in 54.1% of the patients, followed by postsurgical (20.2%) and traumatic (11%). Magluilo also reported a similar rate, in his series of 41 cases, he found a 10% rate of posttraumatic cases [6]. Acquired atresia can be treated medically or surgically but medical management plays a limited role in treatment of posttraumatic atresias since the goal of medical therapy is to control the underlying infection and prevent the formation of granulation tissue. Surgery should be the treatment of choice in posttraumatic EAC atresia. The goal of the surgery is to remove the fibrous plug, widening the bony EAC, exposing the tympanic membrane and to recreate an epithelial lined EAC. The surgical techniques used in the management of canal stenosis have varied over the years, with similar results. In the early years, Soliman et al. [11] removed a wedge of skin from the meatal floor following widening the bony canal and excision of the stenotic tissue, leaving 13 of 16 patients with good results. Adkins and Osguthorpe covered the skin-deficient canal with a transposition flap in eight cases, with no recurrence [12]. McDonald et al. [13] used a split thickness skin graft in 22 cases, with two re-stenosing. Bell used bilateral rotation skin flaps in nine cases with no recurrence [14]. McCary et al. [10] used split thickness grafts in 18 cases, with one recurrence. No matter which technique is selected, the fibrous plug should be completely resected, because residual fibrous tissue has been associated with high incidence of recurrence [1]. Published recurrence rates range from 6% to 27% in different studies [1, 3, 5, 11, 13], with 100% in patients which only the fibrous plug is removed [7]. Becker and Tos [1] presented the largest review of patients that underwent surgical repair with complete resection of the fibrous plug. In 53 ears with a median follow-up of 5 years, complete resection resulted in closure of the air-bone gap to within 20 dB for 61% of the patients and recurrence of stenosis was reported in six ears (11%). An important component of EAC stenosis repair following resection of the fibrous plug is a canalplasty and if necessary, meatoplasty [5]. Also, in EAC atresias, the denuded canal wall should not be allowed to granulate as this will lead to recurrence of atresia; some form of canal lining is required. Different techniques have been used; transposition flaps [12], full thickness skin grafts [15] and most commonly, split thickness skin grafts [1, 3, 4, 6, 16], but all techniques have some degree of recurrence. Although recurrence of EAC stenosis has been documented up to 9 year after surgery, majority of restenosis cases occur after the first postoperative year [2, 16, 17]. In our case, we removed the fibrous plug completely, we enlarged the EAC and we used split thickness skin graft for reepithelization, and the patient had a normal, self cleansing EAC at 18th month follow up. Even if endaural approach has been advocated to have poorer results then retroauricular approach, [7] it did fine in our case.

In conclusion, acquired external auditory atresia is a very rare cause of conductive hearing loss, and direct trauma to external ear can cause this situation. Complete resection of fibrous plug with canalplasty and re-epithelization of EAC with split thickness skin grafts should be the treatment of choice to achieve a patent external ear canal for a long period of time.

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