Categorical data are shown as numbers (percentage, %). Categorical data were compared with χ^2 test. For all statistical analyses, $P < 0.05$ was deemed significant.

RESULTS

In group 1, 33 (82.5%) were female and 7 (17.5%) were male. In group 2, 28 (82.4%) were female and 6 (17.6%) were male. The average age in group 1 is 56.1 ± 7.9 years and in group 2 is 55.2 ± 12.4 years. The groups were similar in terms of age and gender ($P = 0.987$ and $P = 0.216$, respectively).

In the preoperative, OSDI scores (32.4 ± 6.5 vs. 4.8 ± 1.3) and corneal staining scores (2 (1–3) vs. 0 (0–1)) were higher and Schirmer 1 (14.7 ± 6.6 vs. 25.5 ± 6.4 mm) and TBUT (9 (3–16) vs. 14 (11–18) second) were lower in group 1 compared with group 2 (all, $P < 0.001$). In the postoperative first week, OSDI scores (46.2 ± 11.6 vs. 4.8 ± 1.3) were higher, and TBUT (7 (3–11) vs. 14 (11–18) second) were lower in group 1 compared with group 2 (both, $P < 0.001$). The ocular surface parameters were similar between the groups in the first and third postoperative months. (Fig. 1)

DISCUSSION

Epiphora is one of the most common eye problems encountered in oculoplasty clinics. The drainage of tear secretion starts from the puncta located on the medial part of both eyelids, passes through the inferior and superior canaliculi and accumulates in the lacrimal sac. It then travels along the nasolacrimal duct and drains into the inferior nasal meatus. Therefore, stenosis or obstruction at different locations of this lacrimal passage may cause epiphora. In various studies in the literature, nasolacrimal duct blockage was found in ~40% of patients with epiphora. A study by Williams et al reported that obstruction at any location in the nasolacrimal duct was the most common cause of epiphora (33.3%). In addition, 57% of the study patients were female and the mean age was 66 years.[4] Similarly, in our study, the majority of patients were middle-aged to elderly women.

Ocular surface innervation is carried out by afferent fibers of the trigeminal nerve and regulates tear secretion. Tear secretion is decreased in cases of nasolacrimal obstruction and has been reported to improve after treatment of the obstruction.[5] In addition, changes in tear osmolarity after EE-DCR have also been reported.[6] In our study, there was a statistical difference in all parameters between the patient and healthy group in the preoperative period. In the postoperative first week, the corneal staining score and Schirmer I test returned to normal. All parameters returned to normal in the first and third postoperative months. One of the underlying reasons for the early recovery of the corneal staining score may be the exclusion of patients with meibomian gland atrophy, which causes long-term (evaporative type) dry eye disease. Another issue could be the exclusion of patients with inflammatory autoimmune diseases such as Sjogren's Disease, which can cause chronic ocular surface disease.

There are suggestions that there is a decrease in the tear secretion reflex in eyes with PANDO due to epiphora and that ocular surface tests normalize with the return of this reflex in the postoperative period.[7,8] Previous studies have reported that tear meniscus measurements (tear meniscus height, area, and volume) decreased significantly after EE-DCR operation.[9] Compared with healthy control eyes, ocular surface, and tear stability parameters, which were initially worse, caught up with healthy eyes one month after surgery. Therefore, the ocular surface and tear film layer should be examined in the early postoperative period, but it should be kept in mind that the effect of surgery does not appear immediately.

All patients in our study reported that epiphora disappeared in the postoperative period. The reduction of epiphora with surgery is essential for accurate evaluation of the ocular surface and surgical success. Although the EE-DCR technique is a successful operation with updated methods, epiphora may occur again in a very small group of patients in the postoperative period. Although this can be explained by the duration of nasolacrimal duct obstruction, physical irritation of the silicone tube and postoperative ocular pathologies, these symptoms cannot be standardized depending on the patient.

The first limitation of our study is that ocular surface parameters were not evaluated after the removal of the silicone tube. This may have overestimated the surgical success. In addition, the relatively short follow-up period is another limitation. Therefore, although anatomical success was achieved in all our cases, evaluation after the removal of the silicone tube is necessary. The high average age due to the nature of the disease may also be a limitation. Conjunctivachalasis that occurs with aging and lacrimal hyposecretion masked by PANDO may cause changes in measurement parameters. There is a need for multicenter and large participatory studies on this topic.

In conclusion, EE-DCR is an important method for the restoration of deteriorated tear stability. The level of ocular surface disorders and tear instability observed in the preoperative period should not be ignored in the postoperative period. Restoration of these disorders may be an important determinant of patient satisfaction and visual quality after surgery.

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OPEN

Traumatic Neuroma in the External Auditory Canal

Seung Ho Shin, MD, PhD, Hye Min Kim, MD, PhD,† and Eun Jin Son, MD, PhD**

Abstract: Traumatic neuroma is the reparative proliferation of axons and Schwann cells at the proximal end of a severed nerve following injury or surgery. Traumatic neuromas with or without clinical symptoms have rarely been reported in the external auditory canal. A 50-year-old woman with a history of trauma visited our otorhinolaryngology clinic with a 7 × 5-mm mass localized on the anterior wall of the external auditory canal. The mass was easily removed via surgical excision and was histopathologically diagnosed as a neuroma. No signs of recurrence were observed after excision. Herein, the authors present this case, along with a review of the literature.

Key Words: External auditory canal, neuroma, pathology

A traumatic neuroma (TN) is a nodular mass of regenerating axons and Schwann cells that develops at the proximal end of a severed nerve after trauma or surgery. Traumatic neuroma is considered a reparative process consisting of the hyperplastic proliferation of neural fibers and connective tissue.[1] Several cases of TNs of the head and neck have been reported at sites such as the glossopharyngeal, inferior alveolar, and facial nerves and the maxillary division of the trigeminal nerve.[2–4] However, TN in the external auditory canal (EAC) has not yet been reported in the literature. Here, we present the radiologic, clinical, and histopathological findings in a case of TN in the EAC.

CASE REPORT

A 50-year-old woman visited our otorhinolaryngology clinic with a complaint of an incidentally discovered mass in the right EAC. The patient did not report any symptoms, such as otalgia, otorrhea, or paresthesia. The patient had a history of temporomandibular joint injury after being hit on her right jaw with a steel pipe. However, the history of underlying medical diseases, such as hypertension, diabetes, hepatitis, and tuberculosis, was unremarkable. Otoscopic examination revealed a 7 × 5-mm diameter smooth ovoid mass covered with normal skin (Fig. 1A). Pure-tone audiometry revealed normal hearing and no conductive hearing loss on either side.

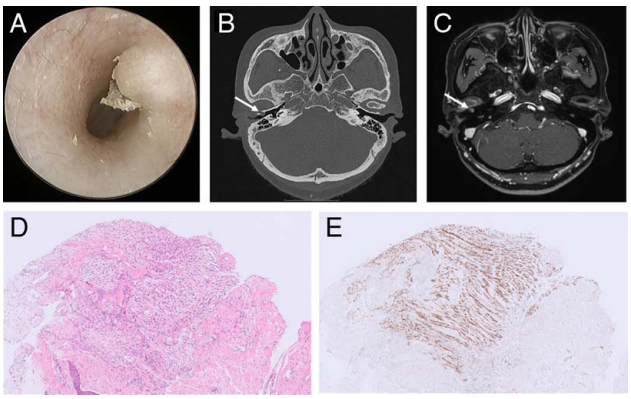

FIGURE 1. (A) Otoscopic examination shows a 7 × 5 mm, smooth, well-defined mass on the anterior wall of the right external auditory canal. (B) Computed tomography shows an ~7-mm diameter soft tissue lesion in the anterior wall of the external auditory canal with erosion of the bony wall (arrow). (C) Magnetic resonance imaging shows a small enhancing lesion in the anterior wall of the external auditory canal associated with adjacent osteoarthritis involving the right temporomandibular joint (arrow). (D) Histopathologically, the proliferation of circumscribed, unencapsulated spindle cells arranged in short bundles composed of axons and Schwann cells is observed (hematoxylin-eosin, ×100). (E) Immunohistochemical staining is strongly but diffusely positive for S-100 protein (×100).

Computed tomography revealed an ~7-mm diameter soft tissue lesion in the anterior EAC with erosion of the bony wall (Fig. 1B). The middle ear, inner ear, and mastoid cavity were intact. Magnetic resonance imaging revealed a small enhancing lesion in the anterior wall of the right EAC, associated with adjacent osteoarthritis involving the right temporomandibular joint (Fig. 1C).

Surgical excision was performed under general anesthesia via a transcanal approach using a microscope. The mass originated from the anterior wall of the EAC with bony erosion. It was completely removed with minimal external auditory skin defects. Histopathology revealed the proliferation of circumscribed, unencapsulated spindle cells arranged in short bundles comprising axons, and Schwann cells (Fig. 1D), and the histopathological diagnosis was neuroma. Immunohistochemical staining revealed strong positivity for S-100 (Fig. 1E). At the 6-month follow-up, there were no signs of recurrence.

DISCUSSION

Traumatic neuromas most commonly develop in the extremities after surgical procedures (amputations), and TN of the head and neck are relatively rare.[1] Several cases of TNs in the head and neck region at sites such as the inferior alveolar, lingual, trigeminal, glossopharyngeal, and great auricular nerves have been reported.[2–4] However, to the best of our knowledge, TN in the EAC has not yet been reported in the literature.

The pathophysiological mechanism of TN is related to excessive hyperplasia and fibrous-tissue proliferation after nerve injury.[1] Traumatic neuromas develop at the proximal end of the sensory nerves. Because motor nerves have limited potential for regeneration, TNs do not occur in the motor nerves.[1,4] Traumatic neuroma in the anterior, posterior, superior, and inferior walls of the EAC canal could originate from sensory nerves, such as the auriculotemporal nerve from the CN V3, cervical plexus (C2, C3), sensory branch of the facial nerve, and auricular branch of the vagus nerve, respectively.[5] Generally, it is difficult to determine the nerve origin during clinical examinations. In this case, we suspected that the tumor originated from the auriculotemporal nerve because the mass was localized on the anterior wall of the EAC.

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Although pain is the most common symptom of TN, the majority of neuromas are asymptomatic. Previous studies have reported that the incidence of symptomatic neuromas in extremity amputations ranges from 4% to 32%. Our patient did not report any symptoms.[6]

Traumatic neuroma of the EAC should be differentiated from schwannomas, fibromas, lipomas, osteomas, neurofibromas, granulomas, sebaceous adenomas, and desmoplastic melanomas.[7] The diagnosis is confirmed based on the histopathological and immunohistochemical findings. Histologic characteristics include circumscribed, unencapsulated proliferation of spindle cells arranged in variable-sized, closely packed nerve bundles composed of axons, Schwann cells, endoneurial cells, perineurial cells, and prominent scar tissue with dense collagen. Traumatic neuromas are typically positive for S-100 protein. Magnetic resonance imaging shows a well-defined, ovoid lesion with intermediate signal intensity on T1-weighted images and intermediate-to-high signal intensity on T2-weighted images.[8]

The treatment of TN remains controversial. Conservative therapy, such as local injections of local anesthetics or steroids or surgery, is considered for patients experiencing pain.[4,9] Although our patient was asymptomatic, an excisional biopsy was performed for a definitive diagnosis because the tumor presented with bony erosion of the EAC wall on computed tomography. The prognosis of TN is good, and recurrence is rare, provided that complete excision is performed.[10]

We presented an extremely rare TM of the EAC canal. Although TN of the EAC is rare, it should be included in the differential diagnosis of benign tumorous lesions in the EAC.

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The Facial Artery Perforator Flap for Reconstruction of Facial Defects: Surgical Pearls and Clinical Series

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Background: Based on the knowledge of facial vascularity, facial artery perforator flaps could be used as potent tools for facial defect reconstruction. However, lack of experience and misconception of this technique limits the broad application in the clinical background. Here, we discussed surgical techniques based on our previous experience with facial artery perforator (FAP)-based facial defect reconstruction.

Methods: A retrospective review of 12 patients undergoing facial defect reconstruction using an FAP flap was performed, including 8 defects in the mid-facial part and 4 defects in the nasal area generally resulted from basal cell carcinoma (8 patients), squamous cell carcinoma (3 patients), and actinic keratosis (one patient).

Results: All patients received one-stage FAP flap reconstruction. The overall follow-up period was 6 to 12 months. All reconstructions were successful with satisfactory patient-reported outcome and no local recurrence. No significant complications were observed in most cases, except for one instance of partial flap loss.

Conclusions: Overall, taking advantage of FAP flaps will contribute to a good functional and esthetic outcome of facial defect reconstructions.

Key Words: Facial artery, facial artery perforator, facial artery perforator flap, facial reconstruction

The reconstruction of soft tissue defects after surgical excision in the facial regions constantly remains a challenge in the plastic and reconstructive field. The fundamental requirement for better clinical outcomes is the ability to innovatively use the tissue to renovate function and achieve esthetic results. Defects resulted from skin neoplasms resection surgery are frequently seen in clinical scenarios, like melanoma, basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and actinic keratosis (AK). In most cases, simple closure and skin graft are not

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