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Patulous Eustachian tube after Le Fort I Osteotomy in a Cleft Palate Patient

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Abstract

A 28 year old man with a repaired cleft palate presenting with symptoms of a patulous eustachian tube (PET) unilaterally after uneventful Le Fort I maxillary osteotomy is reported. PET is a rare condition which is unknown in relation to orthognathic surgery and may represent a rare complication.

Introduction.
Under normal circumstances, the Eustachian tube (ET) remains closed at rest but is opened during swallowing by muscle action associated with elevation of the soft palate. Patulous Eustachian tube (PET) is defined as an abnormal patency of the Eustachian tube and presents with symptoms of ear congestion or fullness, tinnitus and autophony (i.e., an abnormally loud perception of one’s own breathing and voice) (1).

A 28 year old man with repaired cleft palate who developed autophony after otherwise uneventful Le Fort I osteotomy is reported.

Case Report.
The patient was an otherwise healthy 28 year old man with a history of repaired unilateral cleft lip and palate in infancy at another hospital. He first presented to us as a young adult aged 25 years for correction of secondary skeletal cleft deformity and underwent standard orthognathic analysis. There had been no symptom or history of ear infections or hearing loss. Speech was noted to display minor articulation disorder consistent with malocclusion, and mild hypernasality considered not indicative for further investigation. Skeletal diagnosis was maxillary hypoplasia in all dimensions. After a period of presurgical orthodontics, conventional orthognathic surgery by means of Le Fort I osteotomy with advancement and downgrafting was carried out, with fixation by 4 titanium miniplates. During routine postsurgical orthodontics at about 3 months postoperatively, the patient noticed autophony and hearing his own breathing in the left ear. He also complained of fullness in the left ear, distortion of sounds and less tolerance to loud
sounds in the left ear. These symptoms were diminished when in the supine position and whenever nasal congestion was present. He independently sought hearing tests which reportedly were indicative of normal hearing. At about one year postoperatively, the patient decided to report his symptoms to the orthognathic surgeon who promptly requested specialist investigation.

Audiological and otological investigations included pure tone audiogram, immittance audiometry, videotoscopy and nasendoscopy.

Bilateral normal hearing sensitivity and middle ear function were observed on the pure tone audiogram (fig.1). Movements of the tympanic membrane were recorded as changes in acoustic immittance (fig.2). The left tympanic membrane demonstrated a change in acoustic immittance that was synchronous with the normal breathing pattern of the patient (fig.2a), while the right tympanic membrane showed no change in acoustic immittance with breathing (fig.2b). These fluctuations in acoustic immittance on the left side disappeared in the supine position and during cessation of breathing (fig.2c). In addition, synchronous movement of the left tympanic membrane and the chest wall was observed under videotoscopy (fig.3) and an enlarged and persistently open Eustachian tube on the left side was observed on endoscopy of the nasopharynx. On the basis of the above findings, a diagnosis of PET was made. All the above investigations were repeated one month later and noted to confirm the findings and diagnosis of PET.

The patient was given a thorough explanation of the nature and effects of the condition, and no treatment was prescribed in view of the tolerable symptoms. Severity and concern with the symptoms further diminished in the follow-up period of 30 months to date.
Discussion.

The prevalence of PET in the normal population is less than 10 percent (2), but only about 10 to 20 percent of affected individuals are sufficiently disturbed by the symptoms to seek medical advice (3). The exact pathophysiology of PET is not understood, and it is thought to be precipitated by sudden weight loss (2), allergy (4), irradiation (1), sniffing habit (5), chronic middle ear inflammation (6), or pregnancy and hormonal changes (7). The clinical symptom of autophony which abates on lying down, and is absent at night in bed and on rising, but soon recurs on getting out of bed, provides a strong diagnostic clue. Documentation of the diagnosis is best done by immittance audiometry as in this case, but other means include an inflation-deflation test (8), sonotubometry (9), manometric measurement in the nasopharynx and ear canal (10), and CT imaging (11). Depending on the aetiology, treatment reported includes weight gain, medication (like topical steroids, topical antihistamines (12), mucus-producing agents (13)), or myringotomy and ventilation tube placement (14). The ET reportedly may be closed by injection of autologous fat (15), diathermy (16), and pterygoid hamulotomy combined with transposition or transaction of the tendon of the tensor veli palatini muscle (9). Most treatment options, medical and surgical, are said to benefit a proportion of patients in any reported series. However, no specific treatment for the condition has been established to date. Consequently, a policy of non-intervention in mild cases is thought prudent.

There is no evidence to implicate surgical intervention in the form of maxillary osteotomy and advancement as a cause of PET. It is conceivable, at least theoretically, that a Le Fort I osteotomy could affect the ET indirectly through soft palate musculature attachments. Scarring is another potential aetiological factor since PET has been reported in association with radiation and fibrosis (1,8), however, PET is to our knowledge not a recognized complication of nasotracheal intubation. Given that the condition occurs independently in non-surgical patients, it can only be speculated whether it is a truly rare complication of Le Fort I in cleft palate patients or simply a coincidental event in this patient. This single case report should serve to flag the condition at this time.
References


Figure Legends

Figure 1. Air-conduction pure tone audiogram showing normal hearing.

Figure 2. Recordings from Immittance Audiometry showing:

a) Left Ear, Changes in acoustic immittance synchronous with breathing pattern.

b) Right Ear, No change in acoustic immittance during normal breathing.

c) Left Ear, No change in acoustic immittance during cessation of breathing.

Figure 3. Videotoscopy of left ear showed movement of tympanic membrane synchronous with movement of chest wall during normal breathing.
Figure 1
Figure 2

(a) Left ear, normal breathing

(b) Right ear, normal breathing

(c) Left ear, hold breathing