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ScienceDirect

British Journal of Oral and Maxillofacial Surgery 57 (2019) 566–571

BRITISH Journal of Oral and Maxillofacial Surgery

www.bjoms.com

Abnormalities of the muscles of the soft palate and their impact on auditory function in patients operated on for cleft palate: a case-control study

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Accepted 10 May 2019
Available online 31 May 2019

Abstract

Patients with cleft palate have a high incidence of otitis media (OM), which, even after palatal repair, can persist and lead to auditory dysfunction. We aimed to identify what predisposes such patients to develop OM and correlate it with auditory function. We designed a prospective case-control study of adults who had had their cleft palates repaired in childhood. We examined the anatomy of the eustachian tube and the paratubal muscles of the soft palate with magnetic resonance imaging (MRI), assessed auditory function, and correlated the results to evaluate the impact of the pathological anatomy on the function of the middle ear. A total of 64 ears in 32 patients were evaluated (Group A, n = 16) (Group B, controls = 16). MRI showed a short eustachian tube with a shortened tensor attachment in Group A when compared with Group B. The pterygoid hamulus was found to be intact in both groups, but extensive perihamular fibrosis was seen in Group A. A total of 15 ears in Group A had loss of hearing (prevalence ratio (PR) 1.08), (13 mild loss, and 2 moderate loss), and impedance audiometry showed effusion of the middle ear in 12 ears in 16 patients, and dysfunction of the eustachian tube with high negative pressure in the middle ear in four ears in the 16 patients (PR 4.6). These could be positively correlated with the pathological anatomy using Pearson's correlation coefficient. Otitis media is persistent and can lead to conductive hearing loss in adults who have been operated on for cleft palate. This can be attributed to abnormalities in the development of the eustachian tube and the paratubal musculature.

Keywords: otitis media; middle ear; tensor veli palatine; levator veli palatine; eustachian tube

Introduction

Even today cleft lip and palate are among the most common congenital anomalies. They primarily disrupt the anatomy of the oropharynx and can have deleterious effects on the comprehensive functioning of the stomatognathic system.[1] The disruption of the musculature of the soft palate can have far-reaching implications that often include auditory dysfunction. Cleft palate may lead to abnormalities of the tubal dilators, which can lead to the impairment of hearing,[2] and tensor tenotomy done during palatoplasty may exacerbate otitis media (OM).[3] Previous publications have described the pathogenesis of OM in children with cleft palate, and its progression is controversial because no studies to our knowledge have described the definitive site of this pathological anatomy and the impact of palatoplasty on the function of the middle ear. This has led to various attempts to manage OM, such as the insertion of prophylactic ventilatory tubes and the use of temporary hearing aids.[4] Defining the exact location of the problem and its effect on auditory function helps to establish guidelines for the management of OM in patients with cleft palate.[5] We aimed to define the site of pathological

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https://doi.org/10.1016/j.bjoms.2019.05.007

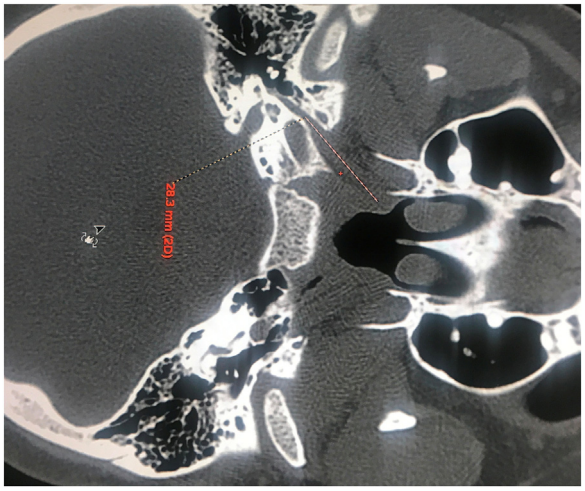

Fig. 1. Computed tomographic image of the length of cartilaginous eustachian tube.

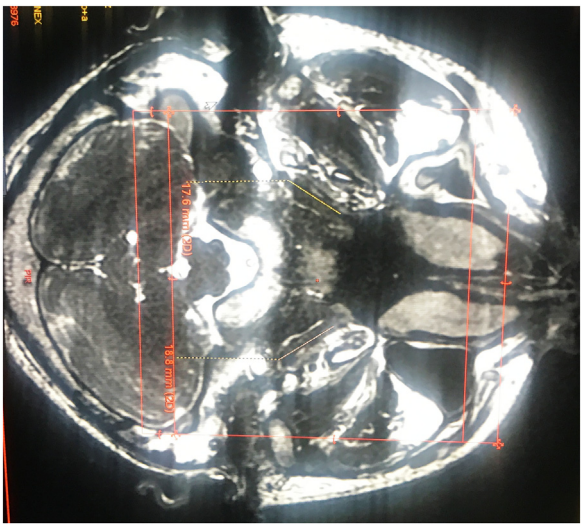

Fig. 2. Magnetic resonance image of the attachment of the tensor veli palatini.

anatomy that predisposes these patients to develop OM, and to correlate it with auditory function.

Patients and methods

We began a prospective, case-controlled clinical trial after approval by the Institutional Review Board (IRB No:SRMDC/IRB/2016/MDS/No.402). The study protocols were explained to all the patients and their informed consent was obtained. They were divided into two groups: group A (study group) included 24 patients who were listed for correction of secondary cleft deformities (secondary cheilorhinoplasty, n = 10; alveolar bone grafting, n = 4; and lip revision, n = 10). Group B (control group) consisted of healthy, age-matched volunteers.

Patients who had had palatoplasty in childhood without any interventions on the middle ear were included in group A, which was then further divided based on the Veau classification (Table 1). Patients with syndromes or who had isolated cleft lip were excluded. All patients included in group A had had two-flap palatoplasty with intravelar veloplasty done by two different surgeons.

Methods

The study was divided into two parts, the first of which included the study of the eustachian tube (Fig. 1) and paratubal musculature (Fig. 2) using MRI and CT. The following variables were recorded: the length of the eustachian tube; the length of the tensor veli palatini (TVP) muscle and its attachment to the eustachian tube; the location of levator veli palatini (LVP); the function of the eustachian tube during the Valsalva maneouvre; and the integrity of the pterygoid hamulus.

The second part of the study included the assessment of the function of the middle ear. This included otoscopic evaluation of the external auditory canal, pure-tone audiometric evaluation of the auditory function (0-90 dB range and frequency range of 500, 100, and 2000 Hz using Digi RS-1 audiometer, Graphic Electronics) and conductance across the middle ear using impedence audiometry (pressure range-+200 to -400 deca Pascals (DaPa) GSI-39 Auto-Tymp, Cardinal Health).

We compared all the variables that we recorded with the controls, and the significance of differences was evaluated using an unpaired *t* test or Fisher's exact test with Pearson's correlation coefficient where appropriate using IBM SPSS Statistics for Windows, version 22.0 (IBM Corp).

Results

A total number of 40 patients were enrolled in the study, 24 of whom were assigned to group A, and 16 to group B. Eight were excluded from group A (two with syndromic cleft lip and palate, and six with isolated cleft lip) based on the exclusion criteria. The remaining 16 patients were included in group A with a mean age (range) of 24.06 (18-32) years divided into groups: A2 (n = 1), A3 (n = 10), and A4 (n = 5). A total of 16 healthy volunteers with a mean age (range) of 23.81(18-32) years were included in group B (Tables 2 and 3).

Anatomical evaluation of the Eustachian tube and paratubal musculature showed that the mean (range) length of the eustachian tube was 22.58 (19.2- 27.1) mm and the TVP was 17.3 (14.4 – 18.9) mm. Those in group A were shorter than those in group B (eustachian tube 25.6 (22.7 – 29.2) mm and TVP 20.35 (17.7 – 22.7) mm) (Tables 2 and 4). These differences were significant (P = 0.0001). The mean length of the eustachian tube was shortest on the cleft side of group A3 followed by group A4 (Table 2).

The mean (range) percentage of attachment of the TVP to the eustachian tube was less in group A than in group

Table 1
Veau classification for cleft palate: distribution in Group A.

Class	Study group	Description
I	A1(n = 0)	Cleft of the soft palate only
II	A2 (n = 1)	Cleft of the hard and soft palate, to the incisive foramen
III	A3(n = 10)	Complete unilateral cleft involving the soft and hard palate, alveolar ridge, and lip
IV	A4 (n = 5)	Complete bilateral cleft involving the soft and hard palate, alveolar ridge, and lip

Table 2
Anatomical findings in groups A2, A3, and A4.

Case No.	Group	Length of eustachian tube (mm) (right)	Length of eustachian tube (mm) (left)	TVP (right)	TVP (left)	Function in eustachian tube (right)	Function in eustachian tube (left)	Middle ear (right)	Middle ear (left)
1	A2	21.5	23.6	17.1	17.3	Closed	Closed	Effusion	Effusion
2	A3(right)	21.3	21	17.5	18	Closed	Open	No effusion	No effusion
3	A3 (left)	21.2	19.7	17.7	17.7	Closed	Open	No effusion	No effusion
4	A3 (right)	23.4	24.5	16.1	17.4	Closed	Open	No effusion	No effusion
5	A3 (left)	20.9	20.5	17.1	16.8	Closed	Open	Sclerosis	Sclerosis
6	A3 (left)	22.3	21.3	17.6	15.9	Closed	Open	No effusion	No effusion
7	A3 (right)	22.1	23.2	16.4	20.2	Closed	Open	Fluid	Fluid
8	A3 (left)	23.3	22.6	16.9	16.8	Closed	Closed	Fluid	Fluid
9	A3 (right)	19.8	23.1	16.9	18.9	Closed	Closed	No effusion	No effusion
10	A3 (left)	20.7	19.2	15.4	14.4	Closed	Closed	Fluid	Fluid
11	A3 (right)	22.5	23.7	18.8	19	Closed	Closed	Fluid	Fluid
12	A4	22.4	23.1	19.1	18.9	Closed	Open	No effusion	No effusion
13	A4	23.5	21.4	17.4	16.9	Closed	Closed	Fluid	Fluid
14	A4	26.5	27.2	17.4	18.8	Closed	Closed	Fluid	Fluid
15	A4	27.1	27	17	17.2	Open	Open	Fluid	Fluid
16	A4	22.3	22.3	16.3	14.7	Closed	Closed	Fluid	Fluid
	Mean value	22.58	25.6	17.3	17.4				

Table 3
Audiometric findings in groups A2, A3, and A4.

Case No.	Group	Hearing decibel range (right)	Hearing decibel range (left)	Hall and Muller grading (right)	Hall and Muller grading (left)	Tympanogram (right)	Tympanogram (left)
1	A2	37	53	Mild	Moderate	A	B
2	A3(right)	23	13	Slight	Slight	A	A
3	A3(left)	34	21	Mild	Slight	A	B
4	A3 (right)	15	13	Slight	Slight	A	A
5	A3(left)	17	18	Slight	Slight	C	C
6	A3 (left)	15	13	Slight	Slight	B	B
7	A3 (right)	38	37	Mild	Mild	A	C
8	A3(left)	33	25	Mild	Mild	B	B
9	A3 (right)	27	22	Mild	Slight	A	A
10	A3 (left)	22	23	Slight	Slight	A	B
11	A3 (right)	38	32	Mild	Mild	A	B
12	A4	33	30	Mild	Mild	A	C
13	A4	17	16	Slight	Slight	A	A
14	A4	30	37	Mild	Mild	B	B
15	A4	32	43	Mild	Moderate	B	B
16	A4	23	25	Slight	Slight	A	A

Typanogram findings; A = normal, B = flat, clearly abnormal, and C = considerable negative pressure.

B: 77.5% (62.7% – 95.4%) compared with 79.9% (68.7% – 89.4%). Within group A the mean (range) percentage of muscle attachment was least in group A4, 71.65% (62% – 84%) compared with group A2, 76.2% and group A3, 80.47% (70% – 82.2%). LVP was attached to the inferomedial aspect of the membranous eustachian tube and the pterygoid hamulus was intact in both groups. An extensive degree of perihamular fibrosis was noticed bilaterally in group A.

Function of the eustachian tube

A total of 23/32 eustachian tubes in group A remained closed during the Valsalva manoeuvre compared with only two that remained closed in group B. This dysfunction in group A could be correlated with the shortened length of the TVP and its attachment to the eustachian tube (Tables 2 and 4).

Table 4
Anatomical findings in controls. There was no effusion in either ear of any patient.

Case No.	Length of eustachian tube (mm)		Length of tensor muscle (mm)		Percentage of tensor insertion to eustachian tube (%)		Tube function	
	Right	Left	Right	Left	Right	Left	Rest	Valsalva
1	23.6	23.9	19.9	20.0	84.3	83.68	Open	Open
2	23.3	23.2	19.9	19.6	84.4	85.77	Open	Open
3	23.7	23.8	20.2	20.9	89.4	87.81	Open	Open
4	22.7	23.2	20.3	17.9	89.4	77.15	Open	Occluded(no fluid)
5	26.8	24.6	20.7	18.7	77.2	76.01	Open	Open
6	24.0	24.2	17.7	21.0	73.75	86.77	Open	Open
7	28.3	29.1	19.8	19.9	75.95	75.38	Open	Open
8	28.1	29.2	19.3	22.0	68.68	75.34	Open	Open
9	26.6	24.2	19.9	21.0	74.81	86.77	Open	Open
10	27.4	28.0	19.6	19.8	71.54	70.71	Open	Open
11	23.9	23.5	20.9	20.2	87.4	85.95	Occluded	Open
12	26.5	26.9	20.7	19.3	78.1	71.7	Open	Open
13	26.2	26.0	22.5	22.7	79.7	78.00	Open	Open
14	25.4	25.6	20.5	19.9	80.7	77.7	Open	Open
15	27.9	28.7	21.8	22.0	78.13	76.7	Open	Occluded
16	24.3	26.4	20.6	22.0	84.7	83.3	Open	Open

Table 5
Audiological status in controls.

Case No.	Audiometry (type)		Audiogram (dBHL)		Otoscopic findings		Results	
	Right	Left	Right	Left	Right	Left	Right	Left
1	A	A	15	17	Intact	Mild retraction	Suggestive of normal functioning	Suggestive of normal functioning
2	A	A	17	17	Intact	Intact	Normal functioning	Normal functioning
3	A	C	20	17	Intact	Retracted	Normal functioning	Middle ear pathology
4	A	A	15	15	Intact	Intact	Normal functioning	Normal functioning
5	A	A	19	20	Intact	Intact	Normal functioning	Normal functioning
6	A	A	23	22	Intact	Intact	Normal functioning	Normal functioning
7	A	A	20	22	Intact	Intact	Normal functioning	Normal functioning
8	A	A	13	12	Intact	Intact	Normal functioning	Middle ear pathology
9	A	A	13	12	Intact	Intact	Normal functioning	Normal functioning
10	A	A	17	18	Intact	Intact	Normal functioning	Normal functioning
11	A	A	20	17	Intact	Intact	middle ear pathology	Normal functioning
12	A	A	19	17	Intact	Mild retraction	Normal functioning	Middle ear pathology
13	A	A	22	19	Intact	Intact	Normal functioning	Normal functioning
14	C	A	32	22	Retracted	Intact	Middle ear pathology	Normal functioning
15	A	A	25	21	Mild retraction	Intact	Middle ear pathology	Suggestive of normal functioning
16	A	As	20	19	Intact	Intact	Normal functioning	Normal functioning

Auditory function

A total number of 32 ears were assessed for auditory function in each group, and 15/32 ears in group A presented with mild to moderate hearing loss with a PR of 1.08. The mean (range) of hearing in group A was 26.4 (13 – 43) dB and in group B was 18.6 (12-32) dB (Tables 3 and 5).

Impedence audiometry showed that 71.8% of group A had abnormal tympanograms (Table 3), which was suggestive of middle ear abnormality, compared with five ears in group B that presented with abnormal tympanograms (Table 5) ($p < 0.0001$). The PR was 4.6. Fisher's exact test showed significant results ($P < 0.0001$) (Tables 3 and 5).

From the results we infer that the length of the eustachian tube, TVP, and the attachment percentage of the TVP to the eustachian tube are important factors that lead to dysfunction of the eustachian tube in patients with cleft palate, and lead to impaired auditory function with middle ear abnormalities.

Discussion

Auditory function is one of the most important sensory functions, the dysfunction of which affects the quality of life. Early hearing loss acts as a barrier for speech development that will interfere with a child's development.[6] Cleft palate is one of the common congenital conditions that affects middle ear function and leads to auditory problems. This has been attributed to the dysfunction of the eustachian tube and PTM.[7]

The eustachian tube is an incomplete, complex, osseocartilaginous tube that connects the middle ear to the nasopharynx.[8] It is suspended from the cranial base by medial and lateral tubal ligaments. Its inferolateral portion is membranous and is abutted by the TVP and Ostmann's fat pad, which act as a hypomochlion to aid in the opening of the eustachian tube during function.[9,10]

Tensor palatini, which inserts into the inferolateral position of the eustachian tube, acts as its primary dilator.[11,12] It extends from the skull base and cartilaginous eustachian tube to the pterygoid hamulus, where the TVP tendon winds around the hamulus and acts as a pulley to help open the eustachian tube. The second functional unit of the TVP extends from the hamulus into the soft palate to form the aponeurosis. At this point, it aids in tensing and raising the soft palate to achieve velopharyngeal competence.[13] Disruption of this anatomy in patients with cleft palate may impair the effective opening of the eustachian tube and lead to inflammation of the middle ear.[14]

Current publications on fetal studies have shown that the eustachian tube is hypoplastic in fetuses with cleft palate,[15] but this has not to our knowledge been discussed in adults. Our clinical trial shows that the cartilaginous eustachian tube and TVP remain short in adults who have been operated on for cleft palate, but we did not find a significant difference in the percentage attachment of the tensor palatine to the eustachian tube. This can be attributed to the mutual shortage in the lengths of the eustachian tube and TVP.

Apart from this anatomical abnormality, we have noticed that the TVP in patients with cleft palate often fails to dilate the eustachian tube. This was more pronounced by lack of function on the cleft side compared with the non-cleft side in group A3, which emphasises that the tensor palatine muscle could be hypoplastic (together with hypoplasia of the levator), as it is involved with the opening of this membranous eustachian tube at the torus tubarius.[16]

The impact of integrity of the pterygoid hamulus, and insertion of the TVP tendon, on the function of the middle ear have been extensively debated. Sehhati-Chafai-Leuwer et al have discussed that integrity of the hamulus and tensor tendon is essential to prevent OM in patients with cleft palate.[17] This is in contradiction to the observation in our study in which we found that though the hamulus was intact there was a high incidence of OM in patients who had been operated on for cleft palate (group A). Apart from this, the effect of procedures like tensor tenotomy and tensor tenopexy have been extensively debated,[18,19] with no definitive conclusion. The TVP should be considered anatomically as a single muscle with two functional units: the first of which extends between the eustachian tube and pterygoid hamulus, and the second from the hamulus into the soft palate with the TVP/hamulus acting as a free pulley system.[20]

Palatoplasty by any of the techniques of veloplasty that have been described so far involves the correction of the latter, which may not change the functioning of the tensor proximal to the pterygoid hamulus. Clinical studies by Flores et al on the impact of TVP preservation, transection, and transection with tenopexy, stated that even tensor tenopexy merely reduced the need for myringotomy but did not abolish it, emphasising the progressive nature of OM irrespective of the technique of repair.[20] They also stated that even after transection of the tensor the effect of the free pulley of the TVP on the eustachian tube is not lost, as there is partial fibrous attachment of the ligament to the hamulus, but this could not be delineated in our study as a result of perihamular fibrosis.

Considering all these factors, we consider that, hypoplasia of the TVP of developmental origin may be the primary cause of middle ear issues in these patients, and disruption of the tendon/hamular fracture done as a part of palatal repairs acts as an additional factor that can promote the progression of OM. This can be supported by the variable degree of OM in patients with similar cleft patterns, who were operated on using the same technique by a single surgeon.[21,22]

Long-term dysfunction of the middle ear can have a deleterious impact on auditory function. Chronic infection of the middle ear can lead to the seepage of bacterial toxins into the internal ear and sensorineural hearing loss.[20,23,24]

Studies have shown reduced accuracy of hearing secondary to OM in children with cleft palate,[23] and there is also evidence that this may impair the development of speech.[26,27] Most current studies have focused their assessments on children up to 6 years of age who had no clefts. The justification for this has been attributed to the change in the angulation of the eustachian tube to the cranial base with time,[19] and so the role of this in children with clefts remains controversial.

Our study provides evidence that the OM in patients with cleft palate persists into adulthood, although it may remain quiescent with episodes of acute exacerbation. This tended to have an adverse outcome on the auditory function in 15/32 ears in group A that had hearing loss.

Correlating the hearing loss variables with the pathological anatomy shows that it is more profound in patients with bilateral clefts (dB range: 17 - 43), which is similar to hearing loss on the cleft side in patients with unilateral cleft lip and palate (dB range: 13 - 38). This is irrespective of the integrity of the pterygoid hamulus. This correlates with the short length of eustachian tube with corresponding insertion of the TVP.

Our study shows, therefore, that the pathology of the eustachian tube and hypoplasia of the paratubal muscles have an impact on the function of the middle ear, which in turn lead to auditory dysfunction in patients with cleft palate that persists into adulthood. The nature of the cartilage of the eustachian tube and the status of the lateral lamina could not be assessed, however. Reviews of studies pertaining to these factors on fetal specimens with cleft palate have suggested that the lateral lamina is shorter with a slit-like eustachian tube could contribute to dysfunction of the eustachian tube.[15]

This discussion raises two important issues that will need to be addressed in future studies: the prophylactic insertion of a ventilatory tube, and the role of eustachian tube dilators.

Considering that the problem persists at the level of the eustachian tube and paratubal muscles, the prophylactic insertion of a ventilatory tube is not necessary and should be restricted to patients with the appropriate symptoms and effusion. Temporary hearing aids should be considered to overcome the morbidity associated with insertion of ventilatory tubes.[20,23] Focus should be directed towards early identification of auditory dysfunction and its treatment with temporary hearing aids[25] or bone-anchored hearing aids[28] as these aid in the development of speech during the formative years and improve the quality of life. Developing artificial eustachian tube dilators will be the future for management of these issues in patients with clefts.[29]

Conflict of interest

We have no conflicts of interest.

Ethics statement/confirmation of patients' permission

Ethics approval was obtained from the Institutional Review Board. The patients' informed consent was obtained.

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