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Adenoid hypertrophy causing obstructive sleep apnea in children after pharyngeal flap surgery

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Abstract

Purpose Pharyngeal flap surgery used for treatment of velopharyngeal insufficiency (VPI) may be followed by obstructive sleep apnea (OSA) especially if the patient has developed adenoid hypertrophy. However, adenoidectomy may adversely affect speech in these patients. The aim of this study was to assess the effectiveness of transnasal endoscopic power-assisted adenoidectomy in relieving OSA in patients with adenoid hypertrophy who underwent pharyngeal flap surgery, and the impact of the procedure on their speech.

Methods Transnasal endoscopic power-assisted adenoidectomy for nine children presenting with adenoid hypertrophy was performed. The patients had previously undergone pharyngeal flap surgery for treatment of VPI. Flexible nasopharyngoscopy was used in the diagnosis of adenoid hypertrophy. Pre- and postoperative polysomnography with measurement of apnea–hypopnea index (AHI) was done. Additionally, auditory perceptual assessment of speech (APA) and nasalance scores was measured pre- and postoperatively.

Results All patients were shown to have OSA by polysomnography, and a larger adenoid size was significantly associated with a higher AHI. We achieved a significant improvement in AHI after adenoidectomy. However, six patients still demonstrated OSA, albeit with a reduced severity. Speech was not adversely affected postoperatively as the APA and nasalance scores showed non-significant changes.

Conclusion Adenoid hypertrophy may be encountered in children who undergo pharyngeal flap surgery, which may cause OSA. Transnasal endoscopic power-assisted adenoidectomy is a safe and effective method for treatment of OSA in those patients without prejudicing the pharyngeal flap, and it has no adverse effect on speech.

Keywords Adenoid hypertrophy · Pharyngeal flap · Obstructive sleep apnea · Velopharyngeal insufficiency · Adenoidectomy

Introduction

Velopharyngeal insufficiency (VPI) is the incomplete closure of the velopharyngeal port during speech. It leads to air leakage into the nose with resultant hypernasality and nasal emissions during speech articulation [1]. A structural palatal defect is the main cause of VPI, and it is frequently seen after cleft palate repair. Also, VPI may be found in patients with submucous cleft palate (SMC) and occult SMC. However, the frequency of VPI after palatoplasty which may need surgical correction reported in the literature varies between 15 and 45% [2, 3]. Surgical techniques used for the treatment of VPI are either palatal or pharyngeal procedures. Pharyngeal procedures are designed to narrow the velopharyngeal port and include pharyngeal flap surgery, sphincter pharyngoplasty, or

This case series study was conducted in the Departments of Otolaryngology of Cairo University, Aswan University, and Fayoum University, Egypt in the period from March 2011 to February 2018.

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posterior wall augmentation. Pharyngeal flap surgery is a common procedure and considered the black horse treatment for VPI. It involves using a myomucosal flap from the posterior pharyngeal wall which is better to be superiorly based and is inserted in the soft palate forming a bridge in the central part of the velopharynx [3–5]. This procedure has potentially beneficial effects for speech, with success rates ranging from 74 to 98% [1, 4, 5].

Partial or complete upper airway obstruction is the leading cause of obstructive sleep apnea (OSA). It is a common illness in children, affecting 2–4%, with a peak incidence between 1 and 8 years of age [6, 7]. OSA is characterized by repeated oxygen desaturation during sleep which provokes cortical arousals and leads to disturbed sleep patterns. Children with OSA may have concentration deficits, reduced learning ability, developmental delay and even school failure [8]. Adenotonsillar hypertrophy is the most common cause of OSA in pediatric patients, consequently the American Academy of Pediatrics recommended tonsillectomy and adenoidectomy as the first line of treatment for pediatric OSA [8, 9]. Velopharyngeal surgery used for treatment of VPI may cause OSA in children as it causes narrowing of the velopharyngeal port [3]. Pharyngeal flap surgery is associated with the worst effect on airway, it may be accompanied with OSA in 40% of patients [4]. If a child underwent such surgical procedures and developed adenoid hypertrophy, the treatment should be directed for relieving the airway obstruction without affecting the speech. The aim of this study was to assess the effectiveness of transnasal endoscopic power-assisted adenoidectomy on the relief of OSA in patients with adenoid hypertrophy who underwent pharyngeal flap surgery, and the impact of the procedure on speech.

Materials and methods

This study included nine patients with adenoid hypertrophy. The patients had been referred from the Phoniatric Unit of our tertiary referral center which provides speech therapy for VPI patients. The study was conducted in the period from March 2011 to February 2018. The patients had previously undergone superiorly based pharyngeal flap surgery (mean age at surgery was 6.11 ± 1.14 years) for treatment of VPI after cleft palate repair, and they were undergoing a routine regimen of postoperative speech therapy. After a mean duration of 1.51 ± 0.73 years following pharyngeal flap surgery, all patients developed manifestations suggestive of OSA including night symptoms (snoring with observed apnea and restless sleep) as well as daytime symptoms (hypersomnolence) [10, 11]. Patients who presented with craniofacial anomalies were excluded from the study. Table 1 summarizes the clinical characteristics of the patients. We obtained informed consent from the parents of the patients, and we followed the principles outlined in the Declaration of Helsinki. Also, the study protocol was approved by the Research Ethics Committee of our institute (N-64-2011).

All patients were subjected to the following:

Preoperative assessment

Medical history was obtained from the parents of the children, with confirmation of OSA symptoms. Otolaryngologic examination was performed for detection of associated craniofacial anomalies, nasal pathology and size of the tonsils and adenoid. The size of the tonsils was categorized from 0 to 4 based on the percentage of oropharyngeal airway occupied by the two tonsils [12]. Adenoid hypertrophy was initially diagnosed by flexible nasopharyngoscopy performed by the Phoniatrician before referral (Fig. 1). Adenoid size

Table 1 Clinical characteristics of the patients

Patient	Age (year)	Sex	Adenoid size	Tonsillar size	AHI		APA score		NS for oral sentences		NS for nasal sentences	
					Pre-	Post-	Pre-	Post-	Pre-	Post-	Pre-	Post-
1	7.0	Male	Grade II	0	7	1	3	4	11.5	12.2	40.4	41.5
2	8.5	Female	Grade II	1	5	0.5	5	4	11.4	10.5	38.7	35.8
3	6.3	Female	Grade III	1	8	2	5	3	12.0	12.1	39.8	40.2
4	9.7	Female	Grade II	1	12	3	4	6	10.8	11.3	39.5	42.1
5	5.8	Male	Grade III	2	6	0.5	2	2	12.0	10.7	37.0	37.8
6	10.2	Male	Grade IV	1	14	1	3	2	12.5	11.0	40.0	39.2
7	12.0	Female	Grade II	1	8	2	5	3	11.0	11.0	38.5	39.2
8	7.2	Male	Grade IV	1	18	3	2	3	10.4	10.9	41.7	42.1
9	9.0	Male	Grade III	0	9	0.5	5	4	11.1	10.8	36.0	36.7

AHI apnea–hypopnea index, *APA* auditory perceptual assessment; *NS* nasalance score, *Pre-* preoperative, *Post-* postoperative

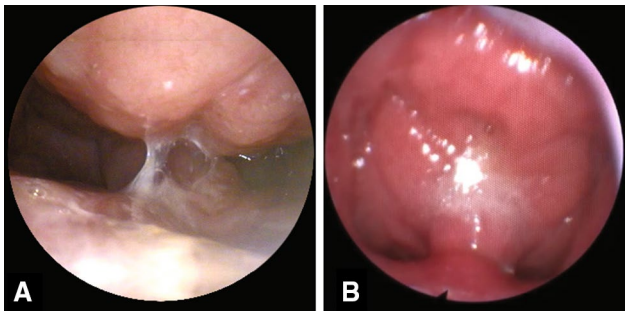

Fig. 1 Flexible nasopharyngoscopic views of the velopharynx. **a** Preoperative view shows adenoid hypertrophy grade II with the pharyngeal flap below it, **b** postoperative view shows the pharyngeal flap after removal of the adenoid

was categorized I–IV according to the degree of choanal obstruction by the adenoid tissue [13]. Also, ear examination was performed for detection of middle ear effusion.

Overnight polysomnography (PSG) were used in assessment of all patients (lab-based study for at least 6 h in a quiet, dark room). The Apnea/hypopnea index (AHI) and minimum O_2 saturation were measured. The AHI was categorized as follows: < 1.0, normal; 1–4.99, mild; 5 to < 9.99, moderate; and ≥ 10.0, severe [14]. The last author (AA) analyzed the PSG data.

Speech assessment was performed using auditory perceptual assessment (APA) and nasometry. APA included measurement of hypernasality, nasal emission of air and weak pressure consonants, and each item was graded on a 5-point scale (0–4) in which 0 indicates normal and 4 indicates severe affection. A lower grade achieved on this scale indicates less dysfunction. Measurement of nasalance score (nasally expressed acoustic energy is compared to the orally expressed energy) was performed using a Nasometer (Model 6200; Kay Elemetrics Corp., Lincoln Park, NJ). The nasometric data were obtained while the patients read and/or repeated standardized nasal and oral sentences.

Operative procedure

All operations were performed by the first author (MA). Under general anesthesia with oral endotracheal intubation, the patient was placed in the routine position for endoscopic sinus surgery with the head up. Both nasal cavities were decongested with xylometazolinee 0.5 mg/mL; 0º and/or 30º of 2.7 mm diameter nasal endoscope (Karl Storz Hopkins® II Optik endoscope, 18 cm length) were used. A camera (Karl Storz GmbH & Co KG; Tuttlingen, Germany) was connected to the endoscope and the view was displayed on a monitor. A microdebrider (Stryker® Core Powered Instrument Driver Console 5400–50) was introduced in front of the endoscope through one nasal side. The adenoid tissue was removed from below upwards, with saline irrigation and an oscillating mode of up to 2000 rpm. Hemostasis was achieved using cotton pledgets soaked with saline–adrenaline 1/200,000. The same procedure was performed from the other nasal side. Care was taken to avoid injury of the Eustachian tube orifices, soft palate, posterior pharyngeal wall as well as the pharyngeal flap during the procedure. After completion of adenoid removal, we packed the postnasal space with a piece of gauze for few minutes.

Postoperative assessment

After strict monitoring of breathing, patients were discharged from hospital on the second postoperative day. At the end of the first and second postoperative weeks, patients were assessed for wound healing using 0º of 2.7-mm diameter nasal endoscope (Karl Storz Hopkins® II Optik endoscope, 18 cm length). At a follow-up visit, 6 months postoperatively, the patients underwent PSG, APA, and nasometric assessment (same parameters as those assessed preoperatively).

Statistical methods

Data were coded and summarized using Statistical Package for Social Sciences version 20.0 for Windows. Quantitative variables are presented as mean ± standard deviation, and categorical data as frequency and percentage. Comparison of preoperative and postoperative results of APA, nasometric assessment and AHI was done using a paired sample *t* test. A one-way ANOVA was used to test the association between categorized adenoid size and AHI. $P < 0.05$ was considered statistically significant.

Results

This case series study was conducted on nine children who underwent pharyngeal flap surgery for treatment of VPI. They presented with adenoid hypertrophy and OSA. The mean age of the patients at presentation was 8.41 ± 2.03 years. The parents of all children gave a history suggesting OSA. Otolaryngologic examination showed no craniofacial anomalies and no nasal pathology. Otoscopy showed ventilation tubes in place bilaterally in seven patients, and no ear abnormalities in two patients. Tonsillar size was graded as 0 in two patients, 1 in six patients and 2 in one patient. Adenoid hypertrophy was scored as grade II in four patients with choanal obstruction of less than 75%, grade III in three patients with choanal obstruction more than 75% and grade IV in two patients with complete choanal obstruction (Table 1). Patients with adenoid hypertrophy grade II showed the pharyngeal flap centrally

with appropriately sized lateral ports; however, we could not assess the velopharyngeal ports in the other patients due to difficulty in passing the nasopharyngoscope through the obstructed choana. No patients developed intraoperative or postoperative complications.

Preoperative PSG showed OSA in all patients; moderate in six patients and severe in three patients with a mean AHI of 9.66 ± 4.21 and a mean minimum O_2 saturation of 88.33 ± 1.80. A larger adenoid size was significantly associated with a higher AHI ($P = 0.02$). Postoperatively, PSG showed complete relief of OSA in three patients demonstrating normal AHI, whereas six patients demonstrated mild OSA with a mean AHI of 1.50 ± 1.03 and a mean minimum O_2 saturation of 92.44 ± 2.30. Comparison between the preoperative and postoperative AHI and minimum O_2 saturation showed significant changes with $P < 0.001$.

The mean preoperative baseline of APA was 3.77 ± 1.30, while the postoperative score was 3.44 ± 1.23. The difference between preoperative and postoperative values was non-significant with $P > 0.05$. Also, the preoperative nasalance scores were 39.06 ± 1.74 for the nasal sentences and 11.41 ± 0.66 for the oral sentences, while postoperative scores were 39.40 ± 2.30 for the nasal sentences and 11.16 ± 0.60 for the oral sentences. The changes were not statistically significant for either nasal or oral sentences with $P > 0.05$.

Discussion

In this study, we performed transnasal endoscopic power-assisted adenoidectomy for nine children who had previously undergone pharyngeal flap surgery for treatment of VPI. All patients had OSA as shown by PSG, and a larger adenoid size was significantly associated with a higher AHI score. We achieved a significant improvement in AHI postoperatively. In addition, speech was not adversely affected by adenoid removal as the APA and nasalance scores did not show significant changes. We opted for the transnasal endoscopic technique to avoid injury of the pharyngeal flap during the procedure. Also, we used the micodebrider as the tool of choice, as it is a cold instrument and less likely to induce fibrosis.

Pharyngeal flap surgery that is used for treatment of VPI usually narrows the upper airway and may cause OSA in some patients. Sometimes, the surgeon may take down the flap to relieve airway obstruction [4, 15]. Hypertrophied adenoid represents the most common nasopharyngeal swelling and it is the most common cause of pediatric OSA either alone or in association with tonsillar hypertrophy [8, 9, 11]. However, the adenoid may act as a pad in the posterior pharyngeal wall against the palate facilitating velopharyngeal closure particularly in patients with VPI. Following adenoidectomy, this compensatory mechanism may not be functional [11]. Treating adenoid hypertrophy in a patient with pharyngeal flap surgery is a surgical challenge, especially if the patient has OSA.

Hubbard et al. [16] reported that adenoidectomy in VPI patients may carry a high risk of increased nasality as the adenoid may support the weakly mobile palate. However, Abdel-Aziz [11] treated OSA in cleft palate patients with tonsillectomy and/or partial adenoidectomy, and achieved a significant improvement of AHI with complete relief of OSA in 70.6% of patients. Indeed, complete adenoidectomy was contraindicated as the patients had weak palatal mobility, contrary to the patients in our present study as the velopharynx was already obturated by a pharyngeal flap. Also, some authors [17, 18] performed partial adenoidectomy using a transnasal endoscopic approach for children with submucosal cleft palate, they achieved significant relief of nasal obstruction, but PSG was not used as an objective assessment method. They confirmed precise clearance of the choana without obvious speech affection.

Katzel et al. [4] reported that pharyngeal flap surgery may be associated with a morbidity of OSA in about 40% of patients. In this instance, the surgeon may take down the flap with the possibility of deleterious effects on speech. Chegar et al. [15] advised performing adenotonsillectomy as a first-stage treatment prior to pharyngeal flap surgery to minimize the postoperative obstructive airway effect. However, removal of the asymptomatic adenoid as a first step for patients with VPI is a risky procedure, as it may have adverse effects on speech, consequences which need parental understanding and compliance [11, 16]. The patients in this study were referred to our institute with adenoid hypertrophy after receiving treatment for VPI. As such, the target of our intervention was to relieve the airway obstruction without worsening speech. Residual airway obstruction with partial improvement of OSA after adenoidectomy in six of our patients may have been caused by the obstructive effect of the pharyngeal flap.

Campos et al. [5] reported that pharyngeal flap surgery may be followed by a high incidence of OSA; however, they detected no significant difference in incidence of OSA between cleft palate patients who did and did not undergo the operation. Although their study was conducted on middle-aged adults, it suggests no major impact of the pharyngeal flap on the airway. The results of their study support the hypothesis of our study which is that adenoid hypertrophy was the main contributor to airway obstruction.

Our study may have some weaknesses; it included a small sample of patients due to the rarity of concurrent presentation of adenoid hypertrophy and pharyngeal flap. Also, we did not know if adenoid hypertrophy was already present before undergoing pharyngeal flap surgery or not; however, our patients developed manifestations suggestive of OSA

after a mean duration of 1.51 years post-surgery which may mean development of adenoid hypertrophy after pharyngeal flap surgery. As such, we recommend careful airway examination in patients of VPI before velopharyngeal surgery. Moreover, we advise adenoid removal for patients presented with adenoid hypertrophy before performing pharyngeal flap surgery. We did not evaluate the effect of tonsillar hypertrophy on the severity of AHI as the maximum tonsillar grade was 2 which may not have a major impact on the airway. In addition, we could not assess the velopharyngeal port preoperatively in all patients due to obstruction of the view by the hypertrophied adenoid, leading to difficulty in passing of the nasopharyngoscope through the obstructed choana.

Conclusion

Adenoid hypertrophy may be encountered in children who underwent pharyngeal flap surgery for treatment of VPI. It may cause obstructive sleep apnea in such patients. Transnasal endoscopic power-assisted adenoidectomy is a safe and effective method in alleviating the upper airway obstruction without prejudicing the pharyngeal flap, and it has no adverse effect on speech.

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Compliance with ethical standards

Conflict of interest There are no conflicts of interest.

Ethical approval The study protocol was approved by the Research Ethics Committee of the Faculty of Medicine of Cairo University (N-64-2011).

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