

Chapter

Therapeutic Approaches in Chronic Adenoiditis

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Abstract

Adenoid tissue diseases (acute adenoiditis, adenoid hypertrophy, and chronic adenoiditis) typically occur in childhood. Adenoid hypertrophy seems to be related to many factors, such as infections, passive smoking, and low vitamin D levels, while the role of allergy still remains controversial. Chronic adenoiditis incidence has increased in recent years, as a result of higher rates of upper airway infections and biofilm formation, with multiple potential clinical complications. Diagnosis is typically clinical, with physical examination and nasal endoscopy. The treatment can be medical or surgical. Non-surgical treatment of chronic adenoiditis with intranasal steroids and leukotriene inhibitors has proven to be effective, reducing the size of the adenoid tissue and symptoms. On the other hand, adenoidectomy is one of the commonest ENT surgical procedures with excellent outcomes and rare adverse events. Curettage adenoidectomy is widely used by many ENT surgeon, but presents risk of residual adenoidal tissue, especially in peritubular and superior nasopharynx regions. In the last years, different surgical techniques have been proposed to reduce surgical risk and morbidity, such as electrocautery adenoidectomy, microdebrider adenoidectomy, and coblation adenoidectomy. Intranasal or transoral endoscopes enabled a great control of surgical field and a complete removal of adenoid tissue.

Keywords: adenoids, adenoid hypertrophy, adenoidectomy, coblation, suction diathermy, microdebrider

1. Introduction

The adenoids or nasopharyngeal tonsils are aggregates of lymphoid tissue located at the posterior wall of the nasopharynx, at the level of the soft palate. Together with the lingual and palatine tonsils they form the Waldeyer's ring, providing an immediate barrier against upper respiratory tract infections and promoting immunity against microorganisms from outside [1].

Histologically, they are composed of epithelial cells, lymphocytes, macrophages, and dendritic cells [2]. Adenoids as well as tonsils are composed mainly of B lymphocytes (50–65%), while T cell lymphocytes comprise 40% of all adenoid and tonsillar lymphocytes. Only 3% is represented by mature plasma cells [3].

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The adenoids are present at birth, develop progressively throughout childhood, and reach their maximum size at about age seven. They then decrease in size due to physiological atrophy during puberty, becoming almost absent by adulthood.

Adenoiditis is therefore a typical disease in childhood and adolescence [1].

2. Adenoiditis

Adenoiditis occurs when there is inflammation of the adenoid tissue resulting from infection, allergies, or inflammation such as irritation from stomach acid as a component of laryngopharyngeal reflux (LPR) [1].

It can be distinguished in acute adenoiditis, adenoid hypertrophy, and chronic adenoiditis.

2.1 Acute adenoiditis

Acute adenoiditis is often the consequence of a viral upper airway infection, often followed by bacterial overinfection [1]. It presents most frequently with the following symptoms: high fever, severe nasal obstruction, mouth breathing, and yellow snot dripping from the posterior pharyngeal wall. In particular, nasal obstruction in infants can lead to dysthia, increased neutrophils, high C-reactive protein (CRP) levels in the peripheral blood, and enlarged adenoids on the lateral image of the pharynx [4, 5].

2.2 Adenoid hypertrophy/vegetation

Adenoid hypertrophy/vegetation represents the most frequently observed clinical condition in ENT medical practice. Its clinical manifestations include mouth breathing, snoring, and adenoid-face. Typically nasal obstruction gets worse in the supine position, leading to sleep apnea in severe cases (obstructive sleep apnea-hypopnea syndrome or OSAHS) [6]. Factors contributing to sleep apnea include obesity, allergies, asthma, gastroenterological reflux disorder (GERD), abnormalities in the physical structure of the face or jaw, and various medical and neurological conditions [2].

Infections are the main cause of adenoid hypertrophy. Interestingly, Epstein-Barr virus (EBV) and Human Bocavirus (HBoV) have been detected throughout the year in samples of children with asymptomatic chronic adenotonsillar diseases, thus attesting their constant presence in the lymphoepithelial tissues of the upper respiratory tract and a pathogenetic potential for development of lymphoid hypertrophy and chronic inflammation [7].

Passive smoking is one of the most notorious risk factors for airway infections in children, such as pharyngitis, rhinitis, sinusitis, otitis, laryngitis, bronchitis, and pneumonia. It has been evidenced in many studies that cigarette smoking reduces the Th1/Th2 ratio and increases proinflammatory molecules, leading to structural changes in the respiratory nasal mucosa, such as impaired ciliary activity and mucociliary function [8]. Moreover, IFN- γ production by CD8+ T cells is severely reduced, thus promoting respiratory tract infections [9].

A recent study by Shin et al. [10] evidenced the correlation between low vitamin D levels and adenotonsillar hypertrophy, emphasizing the importance in measuring serum 25(OH)D level in children with sleep-disordered breathing (SDB) symptoms and increased sizes of the adenoids and tonsils.

The role of allergy in adenoidal disease is still controversial. Sadeghi-Shabestari et al. [11] demonstrated a significant correlation between a positive skin prick test and the high level of serum IgE in patients with adenotonsillar hypertrophy (ATH) rather than other children, while a study by Modrzynski and Zawisz [12] reported that children with allergic rhinitis appeared to be more susceptible to adenoidal hypertrophy (AH). They showed that adenoid hypertrophy was more frequently represented in children with allergic rhinitis related to hypersensitivity to dust mites, than in children with other allergic diseases (asthma/atopic dermatitis) or with no allergies. Furthermore, a hypersensitivity to plant pollen allergens and mold allergens was more frequent in children with AH than in children without AH, demonstrating the role of allergic rhinitis (AR) as the main cause of allergic inflammation around the adenoid.

Another focus of studies is the relevance of localized allergic response in the nasal mucosa of local allergic rhinitis (LAR), in the absence of systemic atopy [13]. In this regard, it has been evidenced that specific immunoglobulin E (sIgE) antibodies are produced locally [13–15] and the local production of total IgE and sIgE antibodies to *Dermatophagoïdes pteronyssinus* (DP) has been demonstrated in the adenoid tissues of atopic children [16].

A recent study by Cho et al. [17] reported a higher sIgE-positive rate in local tissue than in serum. Moreover, 36.2% of children with sIgE-negative serum resulted positive for sIgE in adenotonsillar tissue, suggesting that local allergic inflammation may play an important role in adenotonsillar tissues. These results are in line with a study by Zhang et al. [18] that reported inconsistency in the expression of sIgE antibodies between adenotonsillar tissues and serum.

Furthermore, this study showed that the serum and/or adenotonsillar tissue of 70.6% of children with adenotonsillar hypertrophy (ATH) were sensitized to more than one allergen, suggesting that children with ATH are more likely to have concomitant allergies compared to the general population [17].

However, other studies have found no direct correlation between allergies and adenotonsillar hypertrophy [19–22].

2.3 Chronic adenoiditis

Chronic adenoiditis (**Figure 1**) occurs frequently in children (mainly those aged 3–7 years) [23], showing many of the same manifestations of adenoid hypertrophy, such as continuous or intermittent snoring, mouth breathing, and dry mouth.

However, clinical manifestations, such as fever, increased leucocytes, granulocytes, and elevated CRP levels, are rarely observed when chronic adenoiditis is not severe [6].

Polymicrobial infections and biofilm formation are often the main causes of this chronic disease [24].

A bacterial biofilm (BF) is a membrane-like substance formed by the polysaccharide matrix, fibrous proteins, and proteolipid proteins secreted by bacteria attached to the surfaces of tissues. BFs contain various types of bacteria and even DNA and RNA [25].

The main bacteria are responsible for nasopharyngeal biofilm formation are the otopathogens (*H. influenzae*, *M. catarrhalis*, and *S. pneumoniae*), also leading to middle ear infections [26–30].

It has been suggested that the presence of bacterial biofilm may act as the primary source of infection for other closely related structures, leading to rhinosinusitis, pharyngitis, tonsillitis, and otitis media [24, 31, 32], and may be partially responsible

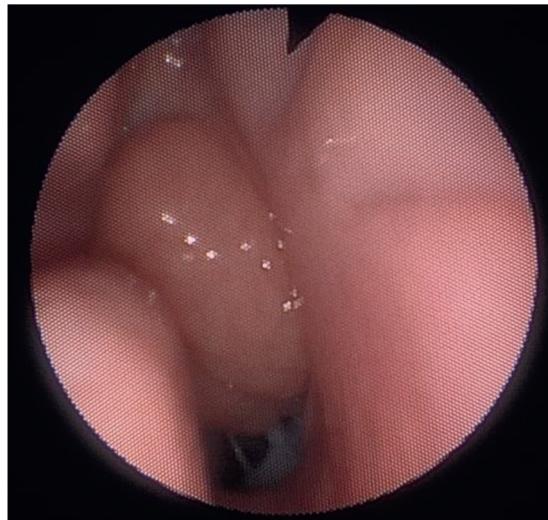


Figure 1.
Chronic adenoiditis.

for the ineffectiveness of traditional antibiotic treatment. This seems to be due to the physical barrier formed by the extracellular matrix, which blocks antibiotic diffusion within the biofilm, as well as some characteristics of the biofilm, such as reduced bacterial replication in the inner layers and resistance mechanisms acquired as a result of the quorum-sensing process [29].

2.3.1 Clinical complications of chronic adenoiditis

Chronic adenoiditis is a long-term infection (especially bacterial infection) that may involve close anatomical structures, leading to acute or chronic infections, such as rhinitis, rhinosinusitis, chronic pharyngitis, cobblestone throat, soft palatitis, abscesses of the posterior pharyngeal wall, and abscesses of the peripharyngeal wall. As observed in all infections, chronic adenoiditis can result in lymphadenitis, typically presenting as an enlargement of the laterocervical and intraglandular-parotid lymph nodes.

Recent studies evidenced that adenoid diseases are one of the main causes of post-nasal drip syndrome (also known as upper airway cough syndrome, UACS) in children, and thus, responsible for chronic cough. Adenoid involvement should be suspected when chronic cough appears or worsens upon postural change or occurs after falling asleep or waking up in the morning.

Moreover, chronic adenoiditis may induce several “infectious immune” diseases, such as rheumatic fever, glomerulonephritis, nephrotic syndrome, and anaphylactoid purpura. Regarding autoimmune nephropathy, it seems that the immune mechanism that causes glomerulonephritis from palatine tonsillitis may be the same as adenoiditis [6].

Given the close relationship between the pharynx and the adenoids with the atlantoaxial joint and the shared venous and lymphatic circulation, an inflammatory involvement of the cervical spine from an upper airway infection has been described. This rare syndrome is known as Grisel’s syndrome, presenting with spontaneous luxation or subluxation of the atlas on the axis, secondary to hyperemia and laxity of the atlanto-axial joints [33].

Chronic adenoiditis represents one of the main causes of ozostomia, together with chronic tonsillitis, tonsillar calculus, and gastrointestinal function disorders, such as gastroesophageal reflux and laryngopharyngeal reflux [6].

Chronic or recurrent middle ear disease is a frequent complication of chronic adenoiditis, due to bacterial colonization of the middle ear along the Eustachian tube, inducing suppurative otitis media [34]. Furthermore, adenoid hypertrophy can obstruct the auditory tube, leading to increased tympanic pressure and secretory otitis media [35].

3. Adenoids and middle ear

The adenoids are located on the posterior wall of the nasopharynx, and they may extend laterally to the ostium of the Eustachian tube, creating a close anatomical and functional relationship with the middle ear, and suggesting how chronic adenoiditis may be complicated by the development of chronic or recurrent middle ear disease. Clinical manifestations of middle ear disease include the presence of serous or mucous fluid in the middle ear, persisting for at least 3 months (chronic otitis media with effusion, OME [36]) (**Figures 2 and 3**); repeated acute middle ear infections, with at least three episodes within a period of 6 months or more than four episodes in a period of 12 months (recurrent acute otitis media, RAOM) [37]; long-lasting



Figure 2.
Myringotomy in OME.



Figure 3.
Temporary ear tube placement.

purulent ear discharge through a persistent perforation of the tympanic membrane (chronic suppurative otitis media, CSOM) [38].

It has been demonstrated that the bacterial biofilm was more frequently polymicrobial near the Eustachian tube orifice [30] and it has been hypothesized its potential role as a source of otopathogens, capable of migrating and colonizing middle ear mucosa through an impaired Eustachian tube [31]. Furthermore, it has been evidenced that nasopharyngeal biofilm-producing otopathogens is more frequently represented in the nasopharynxes of young children with RAOM without chronic adenoiditis, suggesting that nasopharyngeal biofilms are independently involved in the development of recurrent middle ear infections, regardless of the presence of adenoidal hypertrophy [28].

4. Diagnosis

There are currently no universal indications for the diagnosis of chronic adenoiditis. Historically, the two instruments used for diagnostic purposes were the posterior rhinoscopy (with laryngeal mirror) and nasopharyngeal radiological examination.

Lateral X-ray imaging and local computed tomography scanning can show increased adenoid size with the possible obstruction of the upper airway [6]. However, the lack of correlation between the level of obstruction evidenced by X-ray and nasal symptoms has been demonstrated [23]. Moreover, the radiological risk from X-ray exposure should not be underestimated in children [39].

Nowadays, nasal endoscopy is considered the gold standard for diagnosis of the disease. In particular, the flexible fiber-optic scop is widely used in children, allowing a better examination of the nasal cavities and nasopharynx [2].

Many classifications have been proposed for adenoid hypertrophy, but the most relevant for the degree of obstruction of the nasopharynx is that of Parikh et al. [40]: grade 1 for adenoid tissue not in contact with adjacent structures; grade 2 for adenoid tissue in contact with the torus tubarius; grade 3 for adenoid tissue in contact with the vomer; grade 4 for adenoid tissue in contact with the soft palate.

Children typically complain of foreign body sensations at the pharynx, adhesion of the sputum, and postnasal dripping. Endoscopic examination can show mucosal edema on the surface of adenoids with the presence of different degrees of mucus or pus adhesion, while physical examination can evidence retropharyngeal folliculitis and cobblestone-like changes, the adhesion of mucinous, or purulent secretion. Other symptoms may be associated with chronic adenoiditis, such as nasal obstruction, running nose, sneezing, dry throat, and headache [6].

5. Therapeutic approaches: non-surgical therapy

The use of intranasal steroids (INS) is well known for reducing adenoid size and symptoms and it is most often the first line of treatment. A metanalysis by Alisha Chohan et al. [41] in 2015 evaluated the role of mometasone in children with adenoidal hypertrophy. Mometasone caused improvements in outcomes of nasal obstruction, snoring, total nasal symptoms, pure tone audiometry, otitis media with effusion, and quality of life. The doses used range from 100 to 400 mg, for a duration of treatment between 4 and 9 weeks. The effect of mometasone on different outcomes appeared at 6 weeks and remained till 12 weeks.

Other intranasal steroids, such as Fluticasone propionate, Beclomethasone dipropionate, and Flunisolide, are associated with improved nasal obstruction, mouth breathing, apnea, and adenoid size, reducing the rate of surgery for adenoid hypertrophy, especially in children with allergic rhinitis. Proposed mechanisms for the INS activity in adenoid hypertrophy include direct lympholytic action, inhibition of inflammation, and alteration of adenoidal bacterial flora [42].

Leukotriene inhibitors (such as Montelukast, Zafirlukast, and Pranlukast) have been proposed as non-surgical therapy for adenoidal hypertrophy. Montelukast, an oral cysteinyl leukotriene receptor antagonist indicated as preventive therapy for the inflammatory component of asthma and allergic rhinitis, has been shown to cause a reduction of adenoid size in 76% of patients [43].

A 2021 review by Ji et al. [44] on the effect of antileukotrienes on children with obstructive sleep apnea evidenced that leukotriene inhibitors improve sleep disorders and the quality of life in children with mild-to-moderate OSA, alone and in addition to intranasal steroids, reducing tonsillar and adenoid size. Side effects such as headache, nausea, and vomiting were reported only in one study.

Systemic or local antibacterial treatments are effective for chronic adenoiditis induced by bacterial infection [6], but not sufficient modes of treatment for adenoid hypertrophy. Additionally, many viruses are often associated with adenoid hypertrophy [45].

6. Therapeutic approaches: surgical therapy

Adenoectomy with or without tonsillectomy is one of the most common ENT procedures performed in the pediatric population. The most common indication for this procedure is obstructive sleep-disordered breathing and obstructive sleep apnea syndrome (OSAS). Other indications include chronic or recurrent otitis media with effusion, chronic rhinorrhea, nasal obstruction, sinusitis, and chronic adenoiditis. The AAO-HNS 2021 guidelines for adenoectomy are fully described in **Table 1** [46].

The traditional technique is a curettage adenoectomy that is used by most ENT surgeons. In the last years, different surgical techniques have been proposed to reduce surgical risk and morbidity: electrocautery adenoectomy, microdebrider adenoidectomy, and coblation adenoectomy. The ideal technique should be quick, easy to perform, minimize postoperative pain, with a low rate of postoperative complications and relapse rate.

In the traditional adenoectomy, the main bulk of adenoids is removed using curette or adenotome blindly (**Figure 4**), without direct visualization of the nasopharynx. Digital palpation at the end of the procedure is used by many ENT surgeons to confirm the complete removal of adenoid tissue. When the adenoids extend toward the peritubular region or intranasally, adequate removal can be challenging, just by a blind curettage adenoectomy [47].

In order to provide direct visualization of surgical field the use of angled mirrors and, in the last years, of endoscopes during adenoectomy is becoming popular.

A study performed by Ark et al. [48] showed that the direct visualization of nasopharynx during adenoectomy is necessary to fully remove the adenoid tissue. In this study, a group of patients that underwent a traditional adenoectomy was inspected using a laryngeal mirror and only an on-fifth of them had no residual adenoid tissue. The commonest residual site was the nasopharynx roof near the choanal opening, followed by the peritubular region.

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History (One or more required)	<ol style="list-style-type: none"> 1. Four or greater episodes of recurrent purulent rhinorrhea in prior 12 months in a child <12 years of age. One episode should be documented by intranasal examination or diagnostic imaging. 2. Persisting symptoms of adenoiditis after two courses of antibiotic therapy. One course of antibiotics should be with a B-lactamase stable antibiotic for at least 2 weeks. 3. Sleep disturbance with nasal airway obstruction persists for at least 3 months. 4. Hyponasal speech. 5. Otitis media with effusion >3 months or associated with additional sets of tubes. 6. Dental malocclusion or orofacial growth disturbance is documented by an orthodontist or dentist. 7. Cardiopulmonary complications include cor pulmonale, pulmonary hypertension, and right ventricular hypertrophy associated with upper airway obstruction. 8. Otitis media with effusion (age 4 or greater). <p>For infectious conditions, it is recommended that documentation of infections be obtained. For hypertrophy and other noninfectious conditions documentation should include information regarding growth, weight gain, daytime performance issues such as behavior and attention, and any medical condition necessitating removal of the adenoids. Adenoid size is immaterial when the indication is sinusitis, adenoiditis, or otitis media with effusion. Allergic symptoms should have been treated with an adequate trial of allergy therapy before evaluation for non-infectious conditions.</p>
Physical Examination (required)	<ol style="list-style-type: none"> 1. Physical examination (required) 2. Description of uvula, palate, tonsils, nasal airway, and cervical lymph nodes. 3. Evaluation of adenoids by mirror, palpation, nasal endoscopy, or imaging only as necessary. 4. Assessment for signs of hypernasal speech or risk factors for postop voice disturbance
Tests (If abnormality suspected by history, physical examination)	<ol style="list-style-type: none"> 1. Coagulation and bleeding evaluation based on personal or family history 2. Radiographs (lateral neck or cephalometric) 3. Sleep tape recording (if documentation of snoring or apnea is required) 4. Polysomnography in children at high risk for respiratory compromise

Table 1.
AAO-HNS 2021 guidelines for adenoidectomy.

A study by Ezzat et al. [49] performed in a cohort of 300 patients reported that an endoscopic examination after a curettage adenoidectomy showed residual adenoid tissue that required to be removed in 14.5% of cases. In this case, percentage of revision adenoidectomy after 2 years was only 0.85%. Patients that did not undergo an endoscopic examination after the procedure had a revision rate adenoidectomy of 5.6%, showing that the use of an endoscope significantly reduces the incidence of recurrence and the need for revision surgery.

In the '90s, the advent of endoscopic sinus surgery (ESS) popularized the use of intranasal endoscopes and endoscopic adenoidectomy becoming popular, permitting direct visualization of the surgical field and better removal of the adenoid tissue, mostly from the superior part of the nasopharynx and peritubal region [50].

Cannon et al. [50] called the Endoscopic Assisted Adenoidectomy (EAA) a “natural progression of endoscopic technology to allow a more complete adenoidectomy.” Intranasal or transoral endoscopes can be used following traditional adenoidectomy, or in combining techniques with curettes, suction diathermy, microdebriders, and coblator [2].



Figure 4.
Two different-sized Shambaugh adenotomes and adenoid curettes.

A Network Meta-analysis performed in 2023 by Ya-Lei Sun et al. [51] compared four approaches available for adenoidectomy (curettage adenoidectomy, suction diathermy adenoidectomy, powered vacuum shaver adenoidectomy, and plasma field ablation adenoidectomy). It evidenced that there were no significant differences between these techniques for operative time, intraoperative blood loss, and incidence of postoperative residual tissue, while plasma field ablation adenoidectomy showed lower postoperative pain scores than curettage adenoidectomy.

6.1 Traditional adenoidectomy

The traditional adenoidectomy technique includes curettes and/or adenotomes to remove the adenoidal tissue, with patient placed in the Rose position and under general anesthesia *via* oro-tracheal intubation.

The surgeon is placed at the head of the patient (**Figure 5**) and the instruments pass transorally to reach the nasopharynx and the adenoid pad, so as to remove the lymphatic nasopharyngeal tissue (**Figures 6 and 7**).

The procedure is usually performed blindly, and digital palpation confirms the full removal of adenoid tissue. Following that, removal of mucus and clots is performed through nasal irrigation. After several saline irrigations, hemostasis is usually performed by placing a gauze pack in the nasopharynx for some minutes. Cold curettage potentially minimizes morbidities associated with thermal injury, but the blind procedure can theoretically damage the Eustachian tube or pharyngeal muscles and leaves residual tissue, especially in peritubular and superior nasopharyngeal region [47].

The traditional adenoidectomy can be performed using a laryngeal mirror or an endoscope. The latter can be used following a curettage adenoidectomy to remove residual adenoid tissue transnasally with Blakesley forceps [52] or curette [53].



Figure 5.
Surgical position during a traditional adenoidectomy.



Figure 6.
External view of a traditional adenoidectomy performed with a Shambaugh adenotome.

Alternatively, the curettage adenoidectomy can be performed directly under an endoscopic guide, transnasally [54] (**Figure 8**) or transorally [55]. The advantage of these methods is the direct visualization of surgical field that would decrease the rate of residual adenoids and potential injury of the Eustachian tube (**Figure 9**).

6.2 Suction cautery adenoidectomy

In the 1980s, the use of suction electrocautery to control bleeding during curettage adenoidectomy became popular. Subsequently, suction diathermy ablation of the adenoids become a popular alternative to traditional adenoidectomy, resulting being

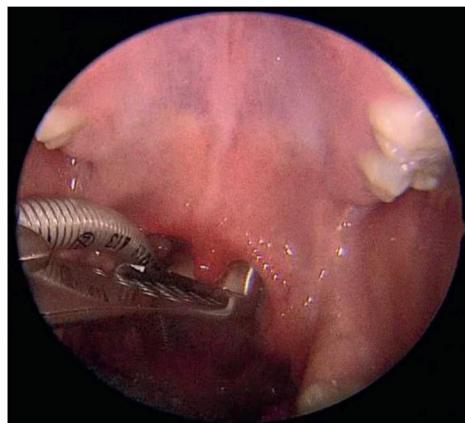


Figure 7.
Internal view of a traditional adenoidectomy performed with a Shambaugh. In the first place the tonsillar tissue adenotome.

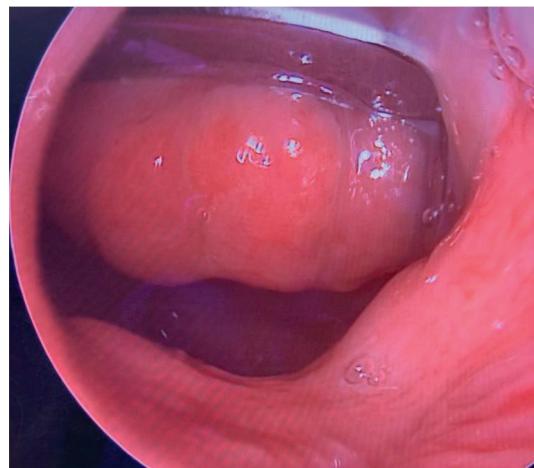


Figure 8.
Curette adenoidectomy under endoscopic vision.

the most common surgical method, as referred to in a 2007 survey among the members of the American Society of Pediatric Otolaryngology [56]. The most common advantage according to this technique is the lower intra-operative bleeding loss and the ability to precisely remove adenoid tissue in the choanal region.

The procedure is done under general endotracheal anesthesia and the adenoid plate is visualized indirectly through a mirror placed in the oropharynx or a transoral angled endoscope. Diathermy ablation of the adenoid pad is performed using an insulated, curved Frazier-type suction system, and a monopolar is applied to the non-insulated portion of the suction or suction coagulator. The suction electrocautery device is applied to the adenoid pad starting from the superior part. The adenoidal tissue shrinks, as the suction device evacuates the smoke. The procedure is completed when the choanae are clearly visible and the nasopharynx presents a smooth level contour. It is important not to traumatize soft palate, Eustachian tube, or pharyngeal wall, so as to avoid scarring and nasopharyngeal stenosis [57].

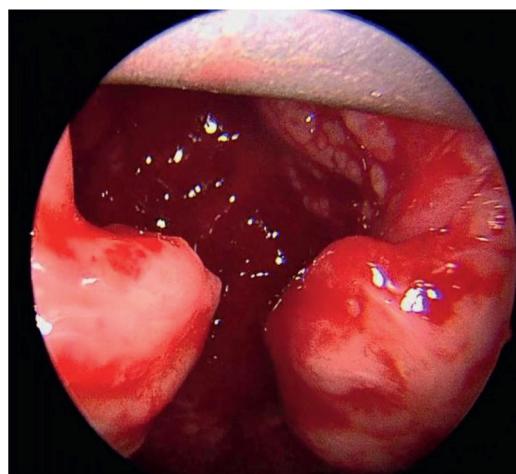


Figure 9.
Transoral endoscopic view after traditional adenoidectomy.

A 2009 meta-analysis by Reed et al. [58] demonstrates that suction cautery adenoidectomy has a significantly lower rate of intra-operative hemorrhage and decreases operative time compared to the curette adenoidectomy keeping equivalent short-term outcomes.

6.3 Coblation adenoidectomy

The word Coblation derives from “Controlled Ablation.” Intended as a low-thermal technology for tissue removal, it recently became popular in ENT procedures. The principle of coblation is that of producing a plasma field using radiofrequency through an electrically conductive fluid, like isotonic sodium chloride solution, so as to remove tissue at a low temperature (40–70°C). Other procedures based on electro-surgical devices such as electrocautery adenoidectomy reach temperatures around 400–600°C. Coblation technology allows tissue removal with a low risk of thermal injury, creating a stable plasma layer of only 100–200 μm thickness around the active electrode, allowing precise tissue excision [59].

Endoscopic-assisted coblation adenoidectomy combines both the advantage of scope view and coblation technology. Several studies have shown that coblation adenoidectomy presents lower post-operative pain than curette adenoidectomy, reducing the use of post-operative drugs and loss of working days for parents, due to faster post-surgical healing. The concomitant use of the endoscope and the small wand tip of coblator allow selective removal of adenoid tissue reaching both the most cranial part of adenoid pad and the intranasal extension, preserving structures such as the Eustachian tube and pharyngeal mucosa. Another advantage is the ability to ablate and coagulate at the same time, as referred disadvantages are the need for a pediatric endoscopic set, the longer pre-operative set-up time, the need for a learning curve in pediatric endoscopy, and the inability to perform a histopathological examination [51, 59–61].

In a study conducted by Bidaye et al. [59] comparing curette adenoidectomy and endoscopic assisted coblation adenoidectomy, it was seen that the percentage of adenoid remnant is 40% with the traditional technique, while it was 0% with the coblator, with

significantly lower postoperative pain in the coblation group. The same results were observed in Hapalia et al.'s [60] and Di Rienzo Businco et al.'s [61] comparative studies, between endoscopic coblation technique and cold curettage adenoidectomy, showing a lower residual adenoid rate and postoperative pain with the former technique.

6.4 Microdebrider adenoidectomy

Power-assisted instruments, such as microdebriders, were first used in endoscopic sinus surgery to treat inflammatory diseases and became popular in adenoidectomy in the last years. The adenoid pad can be removed under laryngeal mirror or under an endoscopic vision, thus permitting a better view of the field. Endoscopes can be used transanally (0° scope) or transorally (45° or 70° scopes). The first adenoidectomy with power-assisted instruments performed under transnasal endoscopic view was first reported by Yanagisawa in 1997 [62].

Costantini et al. [63] proposed a trans-oral adenoidectomy using 40° curved microdebriders and 70° scopes. The instrument is introduced through the mouth and the smooth tip of the microdebrider is placed into the recess between the side vegetations and the tubaric ostium to remove the adenoid pad without damaging the Eustachian tube. At the end of resection, a gauze packing is transorally placed in the nasopharynx to perform adequate hemostasis. This procedure offers an improved visual field and extreme precision in removing the adenoid tissue. Compared with the adenotome or curette removal, it permits to remove adenoid tissue in the most important sites: the choanal and tubaric regions. The continuous suction of the microdebrider during the resection permits for maintenance of a bloodless field, and thus, post-operative bleeding loss is extremely low, comparable with adenoidectomy techniques [51].

In transnasal power-assisted adenoidectomy with transnasal endoscopic control [64], both instruments pass through the nostril (**Figures 10** and **11**). Under endoscopic vision, the shaver cannula is passed into the nose with the suction switched off, so as to not damage the nasal mucosa. The adenoid pad is removed under endoscopic control with care not to lacerate the torus tubarius. Working from proximal to distal the adenoid tissue is removed under a bloodless field thanks to the continuous suctions. A small inferior rim of adenoid tissue can be left intact intentionally, thus preserving the velopharyngeal sphincter.

However, in the presence of a bulky adenoid pad, a pure microdebrider adenoidectomy is time consuming. One possibility is to perform the first step with standard adenoid instruments (curette and adenotome) and then enable microdebriders to remove, if needed, the residual adenoid tissue under a transnasal endoscopic view [65].

Sometimes, the use of transnasal microdebrider under transnasal endoscopic view can be difficult, especially in younger children. The insertion through the nose of both instruments is challenging and the maneuverability is limited. The Transoral Endonasal-Controlled Combined Adenoidectomy (TECCA) technique [66] permits better maneuverability of the instruments in case of narrow nasal spaces and a complete clearance of nasopharyngeal area. As a first step, a traditional transoral adenoidectomy is performed. After that, the residual adenoid is checked with transnasal endoscopy with 0° rigid fiber optic. If residual adenoid tissue is still present, it gets fully removed with curved microdebriders (**Figure 12**).

This procedure carries no additional risk compared to other techniques and is effective to remove the peritubular and superior adenoidal tissue without damaging the surrounding structures [67].

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Figure 10.
Endoscope (0°), curved and straight microdebriders, curette, and Shambaugh adenotome.

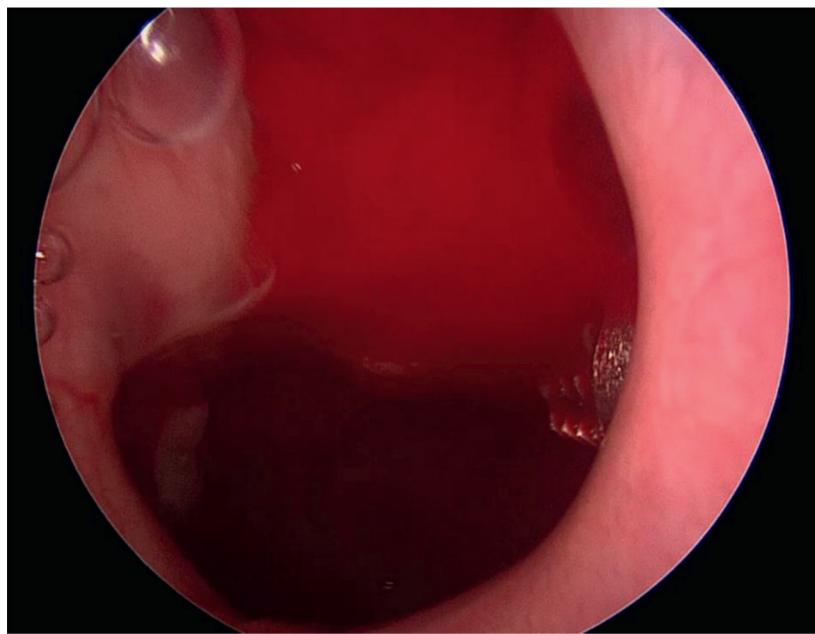


Figure 11.
Transnasal power-assisted adenoidectomy with transnasal endoscopic control adenotome.

Endoscopic power-assisted adenoidectomy is a safe procedure, with minimal intra-operative and post-operative bleeding loss, low post-operative pain, and faster recovery. The endoscopic view gives greater control of the surgical field allowing a complete clearance of adenoid tissue [68].

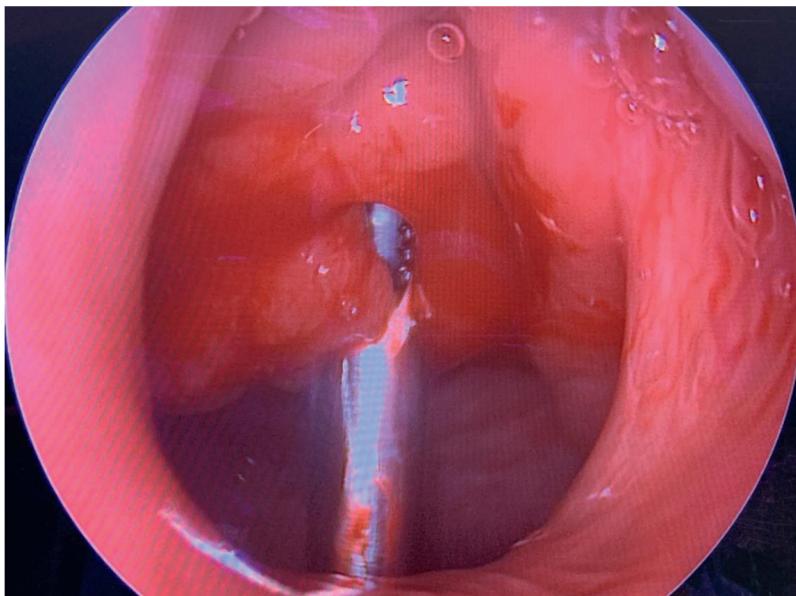


Figure 12.
TECCA technique.

7. Adenoidectomy complication

Adenoidectomy in children is generally a safe procedure with a low rate of complications. The most frequent is hemorrhage, distinct in primary (within 24 hours following surgery) and secondary (after 24 hours following surgery). The rate of the former is between 0.5 and 0.8%, while the latter is very low. The risk of bleeding is additive if a tonsillectomy is associated with the procedure [69, 70].

Velopharyngeal insufficiency (VPI) is a rare but well-recognized complication of adenoidectomy with an impact on the communication skills of a child. It occurs when an inadequate velopharyngeal closure is achieved during speech and/or swallowing, with hypernasal voice quality, nasal regurgitation, and poor speech output. VPI following adenoidectomy is usually transient and should resolve in 3–6 months. Patients with cleft palate, bifid uvula, poor palatal motion, and deep pharynx have a higher risk of developing VPI and should undergo a partial superior adenoidectomy so as to decrease risk. Above 50% of patients with VPI will respond to speech therapy and, in the case of treatment failure, various surgical options are available [71].

Grisel syndrome or non-traumatic atlantoaxial rotary subluxation is a rare adenoidectomy complication, with an increased rate in Down syndrome children. Clinical symptoms are torticollis, pain during neck rotations, and fever. Radiograms and CT scans of the cervical spine are required to confirm the diagnosis. Surgical treatment is indicated in case of high-grade instability or failure of conservative treatment [72].

Nasopharyngeal stenosis is the interruption of the normal communication between nasopharynx and oropharynx, due to the fusion of tonsillar pillars and soft palate to the oropharyngeal posterior wall, creating a strong fibrous wall. It is a rare complication that occurs in excessive electrocautery and dissection.

Its management is surgical: Bilateral Z-pharyngoplasty and palatal eversion are the treatment of choice [73].

Other, very rare, complications described in the literature, are vertebral osteomyelitis, cerebrospinal fluid leakage, and temporomandibular joint dysfunction [45].

8. Conclusions

Adenoideectomy is one of the most common ENT procedures. Traditional curettage adenoideectomy is widely used and it is performed blindly, without direct visualization of the surgical field. In the last years, several other techniques have been proposed: electrocautery adenoideectomy, microdebrider adenoideectomy, and coblation adenoidectomy, with a low rate of residual adenoid tissue and post-operative complications. "Blind" curettage adenoideectomy remains a procedure with good outcomes, but it can fail in obtaining full removal of adenoid pad, especially in the peritubaric, superior, and choanal regions.

Combination of one of the previous surgical techniques along with the direct visualization of the surgical field with endoscopes (transnasally or transorally), allows complete removal of the adenoid tissue and avoids damages to the surrounding structures.

Conflict of interest

The authors declare no conflict of interest.

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References

- [1] Bowers I, Shermetaro C. Adenoiditis. [Updated 2022 Nov 22]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Available from: <https://www.ncbi.nlm.nih.gov/books/NBK536931/>
- [2] Brambilla I, Pusateri A, Pagella F, Caimmi D, Caimmi S, Licari A, et al. Adenoids in children: Advances in immunology, diagnosis, and surgery. *Clinical Anatomy*. 2014;27(3):346-352. DOI: 10.1002/ca.22373 Epub 2014 Feb 18
- [3] Cummings W, Charles S. Text Book of Otolaryngology. 5th ed. Elsevier, Vol. 32010. Chapter 181
- [4] Spector S, Bautista AG. Respiratory obstruction caused by acute tonsillitis and acute adenoiditis. *New York State Journal of Medicine*. 1956;56(13):2118-2119
- [5] Karpova EP, Kharina DV. Vozmozhnosti ratsional'noi farmakoterapii adenoidita u detei [The possibilities for the rational pharmacotherapy of adenoiditis in the children]. *Vestn Otorinolaringologii*. 2016;81(5):73-76. Russian. DOI: 10.17116/otorino201681573-76
- [6] Wang H. Chronic adenoiditis. *The Journal of International Medical Research*. 2020;48(11):300060520971458. DOI: 10.1177/0300060520971458
- [7] Günel C, Kırdar S, Ömürlü İK, Ağdaş F. Detection of the Epstein-Barr virus, human bocavirus and novel KI and KU polyomaviruses in adenotonsillar tissues. *International Journal of Pediatric Otorhinolaryngology*. 2015;79(3):423-427. DOI: 10.1016/j.ijporl.2015.01.007 Epub 2015 Jan 15
- [8] Elwany S, Ibrahim AA, Mandour Z, Talaat I. Effect of passive smoking on the ultrastructure of the nasal mucosa in children. *The Laryngoscope*. 2012;122(5):965-969. DOI: 10.1002/lary.23246 Epub 2012 Mar 23
- [9] Marseglia GL, Avanzini MA, Caimmi S, Caimmi D, Marseglia A, Valsecchi C, et al. Passive exposure to smoke results in defective interferon-gamma production by adenoids in children with recurrent respiratory infections. *Journal of Interferon & Cytokine Research*. 2009;29(8):427-432. DOI: 10.1089/jir.2008.0108
- [10] Shin JH, Kim BG, Kim BY, Kim SW, Kim SW, Kim H. Is there an association between vitamin D deficiency and adenotonsillar hypertrophy in children with sleep-disordered breathing? *BMC Pediatrics*. 2018;18(1):196. DOI: 10.1186/s12887-018-1178-8
- [11] Sadeghi-Shabestari M, Jabbari Moghaddam Y, Ghahri H. Is there any correlation between allergy and adenotonsillar tissue hypertrophy? *International Journal of Pediatric Otorhinolaryngology*. 2011;75(4):589-591. DOI: 10.1016/j.ijporl.2011.01.026 Epub 2011 Mar 5
- [12] Modrzynski M, Zawisza E. An analysis of the incidence of adenoid hypertrophy in allergic children. *International Journal of Pediatric Otorhinolaryngology*. 2007;71(5):713-719. DOI: 10.1016/j.ijporl.2006.12.018 Epub 2007 Feb 5
- [13] Altintoprak N, Kar M, Bayar Muluk N, Oktemer T, Ipci K, Birdane L, et al. Update on local allergic rhinitis. *International Journal of Pediatric Otorhinolaryngology*. 2016;87:105-109.

DOI: 10.1016/j.ijporl.2016.06.008 Epub 2016 Jun 7

[14] Shin SY, Ye YM, Eun YG, Kim SW, Cho JS, Park HS. Local IgE-mediated hypersensitivity to Alternaria in pediatric adenoid tissue. International Journal of Pediatric Otorhinolaryngology. 2012;76(10):1423-1428. DOI: 10.1016/j.ijporl.2012.06.015 Epub 2012 Jul 6

[15] Shin SY, Choi SJ, Hur GY, Lee KH, Kim SW, Cho JS, et al. Local production of total IgE and specific antibodies to the house dust mite in adenoid tissue. Pediatric Allergy and Immunology. 2009;20(2):134-141. DOI: 10.1111/j.1399-3038.2008.00756.x Epub 2008 Jul 24

[16] Brandtzaeg P. Potential of nasopharynx-associated lymphoid tissue for vaccine responses in the airways. American Journal of Respiratory and Critical Care Medicine. 2011;183(12):1595-1604. DOI: 10.1164/rccm.201011-1783OC Epub 2011 Mar 18

[17] Cho KS, Kim SH, Hong SL, Lee J, Mun SJ, Roh YE, et al. Local atopy in childhood adenotonsillar hypertrophy. American Journal of Rhinology & Allergy. 2018;32(3):160-166. DOI: 10.1177/1945892418765003 Epub 2018 Apr 12

[18] Zhang X, Sun B, Li S, Jin H, Zhong N, Zeng G. Local atopy is more relevant than serum sIgE in reflecting allergy in childhood adenotonsillar hypertrophy. Pediatric Allergy and Immunology. 2013;24:422-426

[19] Costa EC Jr, Sabino HA, Miura CS, Azevedo CB, Menezes UP, Valera FC, et al. Atopy and adenotonsillar hypertrophy in mouth breathers from a reference center. Brazilian Journal of Otorhinolaryngology. 2013;79(6):663-667. DOI: 10.5935/1808-8694.20130123

[20] Carr E, Obholzer R, Caulfield H. A prospective study to determine the incidence of atopy in children undergoing adenotonsillectomy for obstructive sleep apnea. International Journal of Pediatric Otorhinolaryngology. 2007;71(1):19-22. DOI: 10.1016/j.ijporl.2006.08.011 Epub 2006 Sep 18

[21] Griffin JL, Ramadan HH, Adham RE. Prevalence of IgE-mediated hypersensitivity in children with adenotonsillar disease. Archives of Otolaryngology—Head & Neck Surgery. 1994;120(2):150-153. DOI: 10.1001/archotol.1994.01880260022005

[22] Alexopoulos EI, Bizakis J, Gourgoulianis K, Kaditis AG. Atopy does not affect the frequency of adenotonsillar hypertrophy and sleep apnoea in children who snore. Acta Paediatrica. 2014;103(12):1239-1243. DOI: 10.1111/apa.12774 Epub 2014 Sep 28

[23] Cassano P, Gelardi M, Cassano M, Fiorella ML, Fiorella R. Adenoid tissue rhinopharyngeal obstruction grading based on fiberendoscopic findings: A novel approach to therapeutic management. International Journal of Pediatric Otorhinolaryngology. 2003;67(12):1303-1309. DOI: 10.1016/j.ijporl.2003.07.018

[24] Zuliani G, Carron M, Gurrola J, Coleman C, Haupert M, Berk R, et al. Identification of adenoid biofilms in chronic rhinosinusitis. International Journal of Pediatric Otorhinolaryngology. 2006;70(9):1613-1617. DOI: 10.1016/j.ijporl.2006.05.002 Epub 2006 Jun 16

[25] Kania R, Vironneau P, Dang H, Bercot B, Cambau E, Verillaud B, et al. Bacterial biofilm in adenoids of children with chronic otitis media. Part I: A case control study of prevalence of biofilms in adenoids, risk factors

and middle ear biofilms. *Acta Otolaryngologica*. 2019;139(4):345-350. DOI: 10.1080/00016489.2019.1571282 Epub 2019 Feb 26

[26] Torretta S, Drago L, Marchisio P, Mattina R, Clemente IA, Pignataro L. Diagnostic accuracy of nasopharyngeal swabs in detecting biofilm-producing bacteria in chronic adenoiditis: A preliminary study. *Otolaryngology–Head and Neck Surgery*. 2011;144(5):784-788. DOI: 10.1177/0194599810394955

[27] Torretta S, Drago L, Marchisio P, Gaffuri M, Clemente IA, Pignataro L. Topographic distribution of biofilm-producing bacteria in adenoid subsites of children with chronic or recurrent middle ear infections. *The Annals of Otology, Rhinology, and Laryngology*. 2013;122(2):109-113. DOI: 10.1177/00034894132200206

[28] Torretta S, Marchisio P, Drago L, Baggi E, De Vecchi E, Garavello W, et al. Nasopharyngeal biofilm-producing otopathogens in children with nonsevere recurrent acute otitis media. *Otolaryngology–Head and Neck Surgery*. 2012;146(6):991-996. DOI: 10.1177/0194599812438169 Epub 2012 Feb 21

[29] Nazzari E, Torretta S, Pignataro L, Marchisio P, Esposito S. Role of biofilm in children with recurrent upper respiratory tract infections. *European Journal of Clinical Microbiology & Infectious Diseases*. 2015;34(3):421-429. DOI: 10.1007/s10096-014-2261-1 Epub 2014 Oct 16

[30] Drago L, Cappelletti L, De Vecchi E, Pignataro L, Torretta S, Mattina R. Antiadhesive and antibiofilm activity of hyaluronic acid against bacteria responsible for respiratory tract infections. *APMIS*. 2014;122(10):1013-1019. DOI: 10.1111/apm.12254 Epub 2014 Apr 4

[31] Torretta S, Drago L, Marchisio P, Ibba T, Pignataro L. Role of biofilms in children with chronic adenoiditis and middle ear disease. *Journal of Clinical Medicine*. 2019;8(5):671. DOI: 10.3390/jcm8050671

[32] Marzouk H, Aynehchi B, Thakkar P, Abramowitz T, Goldsmith A. The utility of nasopharyngeal culture in the management of chronic adenoiditis. *International Journal of Pediatric Otorhinolaryngology*. 2012;76(10):1413-1415. DOI: 10.1016/j.ijporl.2012.06.012 Epub 2012 Jul 9

[33] Fernández Cornejo VJ, Martínez-Lage JF, Piqueras C, Gelabert A, Poza M. Inflammatory atlanto-axial subluxation (Grisel's syndrome) in children: Clinical diagnosis and management. *Child's Nervous System*. 2003;19(5-6):342-347. DOI: 10.1007/s00381-003-0749-6 Epub 2003 Jun 3

[34] Tomonaga K, Kurono Y, Chaen T, Mogi G. Adenoids and otitis media with effusion: Nasopharyngeal flora. *American Journal of Otolaryngology*. 1989;10(3):204-207. DOI: 10.1016/0196-0709(89)90064-1

[35] Kocyigit M, Ortekin SG, Cakabay T, Ozkaya G, Bezgin SU, Adali MK. Frequency of serous otitis media in children without otolaryngological symptoms. *International Archives of Otorhinolaryngology*. 2017;21(2):161-164. DOI: 10.1055/s-0036-1584362 Epub 2016 Jun 3

[36] American Academy of Family Physicians, American Academy of Otolaryngology-Head and Neck Surgery, American Academy of Pediatrics Subcommittee on Otitis Media With Effusion. *Otitis media with effusion*.

Pediatrics. 2004;113(5):1412-1429. DOI: 10.1542/peds.113.5.1412

[37] Gates GA, Klein JO, Lim DJ, Mogi G, Ogra PL, Pararella MM, et al. Recent advances in otitis media. 1. Definitions, terminology, and classification of otitis media. *The Annals of Otology, Rhinology & Laryngology. Supplement.* 2002;188:8-18. DOI: 10.1177/00034894021110s304

[38] Jensen RG, Koch A, Homøe P. The risk of hearing loss in a population with a high prevalence of chronic suppurative otitis media. *International Journal of Pediatric Otorhinolaryngology.* 2013;77(9):1530-1535. DOI: 10.1016/j.ijporl.2013.06.025 Epub 2013 Jul 29

[39] Cho JH, Lee DH, Lee NS, Won YS, Yoon HR, Suh BD. Size assessment of adenoid and nasopharyngeal airway by acoustic rhinometry in children. *The Journal of Laryngology and Otology.* 1999;113(10):899-905. DOI: 10.1017/s0022215100145530

[40] Parikh SR, Coronel M, Lee JJ, Brown SM. Validation of a new grading system for endoscopic examination of adenoid hypertrophy. *Otolaryngology - Head and Neck Surgery.* 2006;135(5):684-687. DOI: 10.1016/j.otohns.2006.05.003

[41] Chohan A, Lal A, Chohan K, Chakravarti A, Gomber S. Systematic review and meta-analysis of randomized controlled trials on the role of mometasone in adenoid hypertrophy in children. *International Journal of Pediatric Otorhinolaryngology.* 2015;79(10):1599-1608. DOI: 10.1016/j.ijporl.2015.07.009

[42] Scadding G. Non-surgical treatment of adenoidal hypertrophy: The role of treating IgE-mediated inflammation. *Pediatric Allergy and*

Immunology. 2010;21(8):1095-1106. DOI: 10.1111/j.1399-3038.2010.01012.x

[43] Shokouhi F, Meymaneh Jahromi A, Majidi MR, Salehi M. Montelukast in adenoid hypertrophy: Its effect on size and symptoms. *Iranian Journal of Otorhinolaryngology.* 2015;27(83):443-448

[44] Ji T, Lu T, Qiu Y, et al. The efficacy and safety of montelukast in children with obstructive sleep apnea: A systematic review and meta-analysis. *Sleep Medicine.* 2021;78:193-201. DOI: 10.1016/j.sleep.2020.11.009

[45] Schupper AJ, Nation J, Pransky S. Adenoidectomy in children: What is the evidence and what is its role? *Current Otorhinolaryngology Reports.* 2018;6(1):64-73. DOI: 10.1007/s40136-018-0190-8

[46] AAO-HNS. Clinical Indicators: Adenoidectomy [Internet]. 2021. Available from: <https://www.entnet.org/resource/clinical-indicators-adenoidectomy/>

[47] Buchinsky FJ, Lowry MA, Isaacson G. Do adenoids regrow after excision? *Otolaryngology-Head and Neck Surgery.* 2000;123(5):576-581. DOI: 10.1067/mhn.2000.110727

[48] Ark N, Kurtaran H, Ugur KS, Yilmaz T, Ozbuduroglu AA, Mutlu C. Comparison of adenoidectomy methods: Examining with digital palpation vs. visualizing the placement of the curette. *International Journal of Pediatric Otorhinolaryngology.* 2010;74(6):649-651. DOI: 10.1016/j.ijporl.2010.03.012

[49] Ezzat WF. Role of endoscopic nasal examination in reduction of nasopharyngeal adenoid recurrence rates. *International Journal of Pediatric Otorhinolaryngology.*

2010;74(4):404-406. DOI: 10.1016/j.ijporl.2010.01.016

[50] Cannon CR, Replogle WH, Schenk MP. Endoscopic-assisted adenoidectomy. *Otolaryngology–Head and Neck Surgery*. 1999;121(6):740-744. DOI: 10.1053/hn.1999.v121.a98201

[51] Sun YL, Yuan B, Kong F. Comparison between different approaches applied in pediatric adenoidectomy: A network meta-analysis. *The Annals of Otology, Rhinology, and Laryngology*. 2023;132(2):207-216. DOI: 10.1177/00034894221081612

[52] Huang HM, Chao MC, Chen YL, Hsiao HR. A combined method of conventional and endoscopic adenoidectomy. *The Laryngoscope*. 1998;108(7):1104-1106. DOI: 10.1097/00005537-199807000-00028

[53] Regmi D, Mathur NN, Bhattacharai M. Rigid endoscopic evaluation of conventional curettage adenoidectomy. *The Journal of Laryngology and Otology*. 2011;125(1):53-58. DOI: 10.1017/S0022215110002100

[54] Wan YM, Wong KC, Ma KH. Endoscopic-guided adenoidectomy using a classic adenoid curette: A simple way to improve adenoidectomy. *Hong Kong Medical Journal*. 2005;11(1):42-44

[55] El-Badrawy A, Abdel-Aziz M. Transoral endoscopic adenoidectomy. *International Journal of Otolaryngology*. 2009;2009:949315. DOI: 10.1155/2009/949315

[56] Skilbeck CJ, Tweedie DJ, Lloyd-Thomas AR, Albert DM. Suction diathermy for adenoidectomy: Complications and risk of recurrence. *International Journal of Pediatric*

Otorhinolaryngology. 2007;71(6):917-920. DOI: 10.1016/j.ijporl.2007.03.001

[57] Elluru RG, Johnson L, Myer CM 3rd. Electrocautery adenoidectomy compared with curettage and power-assisted methods. *Laryngoscope*. 2002;112(8 Pt 2 Suppl 100):23-25. DOI: 10.1002/lary.5541121409

[58] Reed J, Sridhara S, Brietzke SE. Electrocautery adenoidectomy outcomes: A meta-analysis. *Otolaryngology - Head and Neck Surgery*. 2009;140(2):148-153. DOI 10.1016/j.otohns.2008.11.030

[59] Bidaye R, Vaid N, Desarda K. Comparative analysis of conventional cold curettage versus endoscopic assisted coblation adenoidectomy. *The Journal of Laryngology and Otology*. 2019;133(4):294-299. DOI: 10.1017/S0022215119000227

[60] Hapalia VB, Panchal AJ, Kumar R, et al. Pediatric adenoidectomy: A comparative study between cold curettage and coblation technique. *Indian Journal of Otolaryngology and Head & Neck Surgery*. 2022;74(Suppl. 2):1163-1168. DOI: 10.1007/s12070-020-02247-4

[61] Di Rienzo BL, Angelone AM, Mattei A, Ventura L, Lauriello M. Paediatric adenoidectomy: Endoscopic coblation technique compared to cold curettage. *Acta Otorhinolaryngologica Italica*. 2012;32(2):124-129

[62] Yanagisawa E, Weaver EM. Endoscopic adenoidectomy with the microdebrider. *Ear, Nose & Throat Journal*. 1997;76(2):72-74

[63] Costantini F, Salamanca F, Amaina T, Zibordi F. Videoendoscopic adenoidectomy with microdebrider. *Acta Otorhinolaryngologica Italica*. 2008;28(1):26-29

- [64] Al-Mazrou KA, Al-Qahtani A, Al-Fayez AI. Effectiveness of transnasal endoscopic powered adenoidectomy in patients with choanal adenoids. *International Journal of Pediatric Otorhinolaryngology*. 2009;73(12):1650-1652. DOI: 10.1016/j.ijporl.2009.08.019
- [65] Pagella F, Pusateri A, Giourgos G, Matti E. Evolution of the adenoidectomy in the endoscopic era. *Advances in Endoscopic Surgery*. Ed Intechopen; 2011:131-154. DOI: 10.5772/23177
- [66] Pagella F, Pusateri A, Matti E, Giourgos G. Transoral endonasal-controlled combined adenoidectomy (TECCA). *The Laryngoscope*. 2010;120(10):2008-2010. DOI: 10.1002/lary.21070
- [67] Pagella F, Pusateri A, Canzi P, et al. The evolution of the adenoidectomy: Analysis of different power-assisted techniques. *International Journal of Immunopathology and Pharmacology*. 2011;24(4 Suppl):55-59. DOI: 10.1177/03946320110240S411
- [68] Wadhera R, Vashist A, Kumar P, Ghai A. Endoscopic adenoidectomy with microdebrider. *Indian Journal of Otolaryngology and Head and Neck Surgery*. 2022;74(Suppl 2):1314-1318. DOI: 10.1007/s12070-021-02416-z
- [69] Tomkinson A, Harrison W, Owens D, Fishpool S, Temple M. Postoperative hemorrhage following adenoidectomy. *The Laryngoscope*. 2012;122(6):1246-1253. DOI: 10.1002/lary.23279
- [70] Lowe D, Brown P, Yung M. Adenoidectomy technique in the United Kingdom and postoperative hemorrhage. *Otolaryngology-Head and Neck Surgery*. 2011;145(2):314-318. DOI: 10.1177/0194599811403119
- [71] Lambert EM, You P, Kacmarynski DS, Rosenberg TL. Adenoidectomy and persistent velopharyngeal insufficiency: Considerations, risk factors, and treatment. *International Journal of Pediatric Otorhinolaryngology*. 2021;149:110846. DOI: 10.1016/j.ijporl.2021.110846
- [72] Pini N, Ceccoli M, Bergonzini P, Iughetti L. Grisel's syndrome in children: Two case reports and systematic review of the literature. *Case Reports in Pediatrics*. 2020;2020:8819758. Published 2020 Nov 12. DOI: 10.1155/2020/8819758
- [73] Abdel-Fattah G. Palatal eversion for the treatment of combined nasopharyngeal stenosis and tonsillar pillars adhesion. *International Journal of Pediatric Otorhinolaryngology*. 2016;90:227-230. DOI: 10.1016/j.ijporl.2016.09.028