Revision adenoidectomy—A retrospective study

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Summary

Background: Adenoid “re-growth” is a poorly understood phenomenon. While parents often express concerns regarding the potential for adenoid “re-growth”, little information exists in the literature about its incidence and causation.

Purpose: To establish the incidence and possible contributing factors leading to adenoid re-growth in children.

Design: Retrospective case series review.


Methods: The charts of 106 patients who underwent revision adenoidectomy between 1995 and 2006 were reviewed. Thirty-four patients were excluded because the primary adenoidectomy was performed elsewhere or initially only a partial adenoidectomy was performed. In the remaining 72 patients, demographic data, clinical presentation, associated medical conditions and findings at surgery were studied.

Results: During the 11-year study period, 13,005 adenoidectomies or adenotonsillectomies were performed; 72/13,005 (0.55%) underwent revision adenoidectomy. The mean (± S.D.) age at presentation for primary adenoidectomy was 3.68 ± 2.9 and 7.69 ± 4.04 years for secondary (“revision”) adenoidectomy with an average time interval of 4.3 years between surgeries. Age at initial adenoidectomy was not a significant factor in predicting revision adenoid surgery. 29/72 (40%) underwent a reflux work up including scintiscan with gastric emptying, 24 h pH probe, or laryngoscopy. 28/29 (96%) were diagnosed with reflux. At least 15/72 (21%) were reported to have symptoms consistent with adenoid re-growth which were found to be caused by tubal tonsil hyperplasia.
1. Introduction

Many parents request information about their child’s potential for adenoid re-growth before their child undergoes adenoidectomy. However, otolaryngologist’s themselves have little information available to answer these questions. Adenoid re-growth is a poorly understood phenomenon with few studies addressing either its incidence or causation.

Adenoidectomy is one of the most common surgeries in children, performed principally to help relieve conditions such as recurrent otitis media, rhinosinusitis, and nasopharyngeal airway obstruction with sleep disturbance. Yet, after a successful adenoidectomy, some patients develop nasal obstructive symptoms suggestive of adenoid re-growth. After ruling out other pathologies such as environmental nasal allergies, turbinate hypertrophy or septal deviation revision adenoidectomy may be considered. Examination of the nasopharynx might reveal varying degrees of hyperplasia of remaining lymphoid tissue and/or varying degrees of tubal tonsillar hyperplasia. The pathogenesis of hypertrophy of this lymphoid tissue is unknown. Therefore, the purpose of this study is to establish the incidence and identify factors associated with adenoid re-growth in children.

2. Methods

A computer search for CPT codes 42835 and 42836 (adenoidectomy, sec, <12 years and adenoidectomy, sec, age 12+ over) was preformed. The charts of all patients who underwent revision adenoidectomy who previously had primary adenoidectomy as an adenoidectomy alone and/or as an adenotonsillectomy were identified. All patients were operated at a tertiary care children’s hospital during an 11-year study period from 1995 to 2006. All patients had been managed by one of five pediatric otolaryngologists who all used the same adenoidectomy technique—a sharp adenoid curette for removal and a suction bougie cautery set at 30 W for hemostasis. Charts were retrospectively reviewed and studied for clinical presentation of symptoms of adenoid re-growth. Associated conditions such as extraesophageal reflux disease (EERD), inhalant allergies and otitis media were also noted. A prior history or present symptoms for EERD were identified and diagnostic evaluations for EERD, such as gastric scintiscan/gastric emptying, 24 h dual channel pH probe, microlaryngoscopy, bronchoscopy and esophagoscopy were noted.

To estimate incidence, a computer search was completed during the same time period for CPT codes 42820, 42821, 42830 and 42831 (adenotonsillectomy <12 years, adenotonsillectomy 12+ years, adenoidectomy, primary <12 years, and adenoidectomy, primary 12+ years).

3. Results

A total of 13,500 primary adenoidectomies were performed over an 11-year period. A computer search for secondary adenoidectomy identified 106 patients who underwent revision adenoidectomy. Thirty-four (34) were excluded because the initial adenoidectomy was performed elsewhere or only partial adenoidectomy was done previously. Ultimately, 72 patients underwent revision adenoidectomy resulting in an incidence of between 0.55 and 0.80%. Thirty-four patients were females and 38 were male. Of the patients who underwent revision adenoidectomy, mean surgical age (±S.D.) for primary adenoidectomy was 3.6 ± 2.9 and a median age of 2.9 years (Fig. 1A). Revision adenoidectomy was performed at 7.8 ± 4.0 and a median age of 7.0 years. (Fig. 1C) The average time interval between primary adenoidectomy and revision adenoidectomy was 4.2 years. In order to compare the age of patients in the general population that underwent adenoidectomy to those who later underwent revision surgery, we obtained the age of a random sample of 1167/13,500 children who underwent primary adenoidectomy at our institution. The average age of the 1167 patients at adenoidectomy was 4.1 ± 3.2 and median age of 3.0 (Fig. 1B). Therefore, age at initial adenoidectomy was not a significant factor in predicting revision adenoidectomy.

The clinical presentation of adenoid re-growth was similar to the symptom complex prior to the initial adenoidectomy (Table 1).

As for otologic manifestations, 53/72 (74%) had otitis media during the perioperative period of the initial and revision adenoidectomy. Among patients with otitis media, nasal obstructive symptoms were
usually present 71/72 (98.6%) among patients who were undergoing primary and revision adenoidecto-
my. 31/72 (43%) had bilateral tympanostomy tube (BTT) placement at the time of revision ade-
noidectomy, 22/72 (31%) had BTT or laser assisted tympanic membrane fenestration (OtoLAM) prior to revision adenoidectomy and interestingly, 3/72 (4%) had BTT for the first time during the revision adenoidectomy. 19/72 (26%) had no history of ear infection.

Evaluation for EERD was performed in 29/72 (40%). Each one of these 29 patients had at least one test for EERD including gastric scintiscan/gas-
tric emptying, 24 h dual channel pH probe, flexible fiberoptic laryngoscopy, microelaryngoscopy, bronchoscopy with bronchoalveolar lavage, and eso-
phagoscopy with biopsy. 28/29 (96%) had at least one test positive for reflux. Also, younger children less than 1-year-old were more likely to have posi-
tive studies for reflux 4/5 (80%) (Fig. 2). 2/28 were diagnosed with EERD prior to the initial adenoi-
dectomy while the remainder of patients were diagnosed during the interval between the initial and revision adenoidectomy.

As for intraoperative findings, tubal tonsils caus-
ing nasopharyngeal obstruction (Fig. 3) were speci-
ically mentioned in the operative reports in 15/72 (21%) patients. Of these 15 patients with tubal tonsillar hyperplasia, nearly half, 7/15 (46%) had been diagnosed with reflux. The remaining 8/15 (54%) were undetermined since no reflux work up was performed.

The degree of obstruction reported when com-
paring primary and revision adenoidectomy revealed more obstruction during initial adenoi-
dectomy than revision adenoidectomy. 85% had more extensive adenoid obstruction at 60–100% of the nasopharynx during the initial adenoidectomy compared to 56% who had lesser obstruction at 30–
59% of the nasopharynx during the revision adenoidectomy (Fig. 4).

Allergies were diagnosed in 10% of patients who underwent revision adenoidectomy—6% in associa-
tion with documented reflux and another 4% present-
ing allergy alone. In comparison, 28% had reflux alone and the remaining 62% who had revision adenoidectomy had no associated condition identified (Fig. 5).

4. Discussion

In the pediatric population, adenoid hyperplasia is associated with nasal obstruction, rhinosinusitis, obstruc-
tive sleep apnea, and recurrent otitis media. Enlargement of the adenoids during early and mid-

| Table 1 Clinical presentation during the primary and revision adenoidectomy |
|---------------------------------|------|------|
| Obstructive airway symptom       | Primary (%) | Revision (%) |
| Snoring                         | 94        | 94       |
| Nasal congestion/mouth breathing | 76       | 82        |
| Noisy breathing                  | 83       | 80        |
| Rhinorrhea                      | 72       | 69        |
| Otitis media                    | 69       | 74        |
| Sleep disturbance               | 93       | 92        |
number and duration of episodes of otitis media have been well established in randomized trials [4,5]. Similarly a significant decrease in the number of episodes of pediatric rhinosinusitis after adenoidectomy has been described [6].

Adenoid re-growth may be the result of the same presumed causes for primary adenoid hyperplasia. Adenoidectomy may place a greater burden on the remaining lymphoid tissue in the nasopharynx, resulting in progressive hypertrophy of the remaining lymphoid tissues within the nasopharynx over time. 51/72 (71%) of patients who had revision adenoidectomy for adenoid recurrence had their primary adenoidectomy at less than 4 years of age (Fig. 1A). In comparison, 47/72 (65%) had their revision adenoidectomy between age 3 and 8 (Fig. 1C). Patients who had primary adenoidectomy at less than 1 year of age had the shortest time interval between the primary and revision adenoidectomy averaging 2.59 years versus 4.2 years overall. This group of patients presented earliest with symptoms necessitating revision adenoidectomy. Interestingly, this age group also had highest incidence of reflux (4/5).

Symptoms of adenoid re-growth observed in our study occurred at a mean age of about 7.8 years. This time frame is similar to that reported by Emerick and

Fig. 2  Age in relationship to patients with documented reflux. Prevalence of reflux not fully established yet since reflux workups were not commonly done during the early years of the study because of less understanding about pathophysiology of extraesophageal reflux.

Fig. 3  (A) Adenoid re-growth after adenoidectomy causing symptomatic nasal obstructive symptoms and eustachian tube obstruction. (B) Tubal tonsil hyperplasia on the lateral portions of the nasopharynx obstructing the posterior choana on the right.

Fig. 4  Degree of adenoid obstruction in nasopharynx during initial and revision adenoidectomy.

Fig. 5  Associated conditions with adenoid re-growth.
Cunningham [7], mean age of 7 years, 2 months, and
by Honda et al. [8], age range of 5–11 years.

Another reason proposed for re-growth is inadequate removal at initial adenoidectomy, i.e. poor surgical technique. A cross-sectional study of 35 patients from 175 randomly selected children from a total of 774 patients who underwent adenoidectomy over a 2-year period were found to have no significant re-growth (as determined by recurrence of symptoms) after follow up 2–5 years later [9]. Their findings had been attributed to complete removal of the adenoid tissue using a indirect visualization technique. Thus, it is conceivable that incomplete removal of adenoid tissue could still be occurring during the time of initial adenoidectomy, however, this is difficult to prove. We recommend a technique that allows for indirect visualization of the nasopharynx using mirrors for complete a removal as possible, while sparing the tori tubarius.

Patients evaluated for complaints pertaining to primary or recurrent adenoid obstructive disease do not always undergo an etiologic work-up for the cause of the hypertrophy. Given the current availability of very few studies that can explain the causation even from a hypothetical viewpoint, many adenoidectomies are done for symptomatic relief sometimes without addressing the underlying cause. Some practitioners have recommended decreasing the size of the adenoids by application of topical nasal steroid sprays, implicating an inflammatory response in the pathogenesis. In a study by Demain and Goetz, reduction of adenoid tissue as well as nasal airway obstructive symptoms in children were reported [10].

A detailed review of systems and physical examination correlated to the patient’s symptomatology can be very helpful. Conditions warranting further evaluation include environmental and/or food allergies and extraesophageal reflux. The presence of symptomatic adenoid obstructive disease may signal co-existing (and even causative) problems that may have a more subtle presentation such as EERD. In this retrospective study, 28/29 (96%) patients who were studied for EERD were diagnosed with EERD. Only 2/28 were diagnosed with reflux before the initial adenoidectomy. 22/28 were diagnosed with EERD during the interval between the initial and revision adenoidectomy and 4/28 were discovered to have reflux during the time of the revision adenoidectomy. All of the patients having tubal tonsillar hyperplasia who had evaluation for reflux had at least one positive reflux finding. This data strongly suggests that tubal tonsillar hyperplasia is associated with extra-esophageal reflux disease. The increased prevalence of documented reflux disease in patients undergoing revision adenoidectomy may be due to a higher index of suspicion for reflux in patients with recurrent nasal symptoms post-adenoidectomy. Recent increased awareness of the affects of extraesophageal reflux on the pharynx has lead to a higher index of suspicion for reflux in our patients.

In the study, 4/5 children less than 1 year of age were found to have positive studies for reflux. They were also underwent revision adenoidectomy earliest. Similar findings were noted in a study by Carr et al. [11] in another study of 95 children by Carr et al., 88% of the children age 1 year or less had GERD and about 32% of those older than 1 year had GERD diagnosed concluding that practitioners should be aware that infants age 1 year or less with symptomatic adenoid enlargement usually have GERD [12]. Furthermore, Wenzl et al showed that infants and children have a much higher proportion of reflux but of the non-acidic type [13].

The epithelium lining the nasopharynx, including the adenoids and the tubal tonsil, is prone to reflux injury. The contribution of EERD to adenoid re-growth and tubal tonsillar hyperplasia must be considered. Brief exposure of the upper aerodigestive tract and lower airways to the refluxate can have a significant impact. The esophagus has intrinsic anti-reflux defenses that prevent mucosal injury (bicarbonate production, mucosal tissue resistance [14] and esophageal motor function with acid clearance [15]) whereas the pharynx and the larynx do not [16]. Studies had shown that extraesophageal reflux can cause considerable damage to the less resistant respiratory epithelium of the larynx, trachea, nose, and middle ear [17]. Inflammation with resulting hypertrophy of the remaining lymphoid such as the adenoids, lingual tonsils and tubal tonsils is possible.

A retrospective study is limited by incomplete data. Another limitation of this study is the probable underestimation of the real incidence of symptomatic adenoid re-growth. Some children may have undergone surgery by another practice or may have moved from the area. In addition, the true incidence of the co-morbidities of allergies and extraesophageal reflux could not be determined. Of course, association does not indicate causation, and thus, further study of the impact of allergen exposure or refluxate on lymphoid tissue is warranted.

5. Conclusions
The incidence of adenoid re-growth is low and is estimated at less than 1%. A number of patients were identified with tubal tonsillar hyperplasia causing obstructive symptoms. Allergies were
uncommon, but extraesophageal reflux was commonly identified when investigation was completed for this condition. Prospective multi-institutional studies may be helpful to elucidate the incidence and nature of adenoid re-growth in children.

References