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Comparison of nasal mucociliary clearance in adenoid hypertrophy with or without otitis media with effusion ☆☆☆

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ABSTRACT

Objective: To investigate pre- and postoperative mucociliary clearance in patients with adenoid hypertrophy or combined with otitis media with effusion.

Methods: Patients were divided into two groups: Group 1—patients with adenoid hypertrophy (AH), and Group 2—patients with AH and otitis media with effusion (AHOME). In all patients, AH size was recorded, and the Andersen saccharin and methylene blue tests were conducted before and 1 month after surgery to obtain mucociliary clearance time (MCT). Nasal cavity length was measured intraoperatively to establish mucociliary clearance velocity (MCV). Patients with allergic rhinitis, active infection, and history of nasal or ear surgery were excluded.

Results: This study included 64 patients with a mean age of 8.34 ± 2.98 years (range: 3–18 years). Pre- and postoperative MCT were 14.60 ± 4.83 and 9.48 ± 2.63 min in Group 1 and 16.03 ± 4.31 and 12.12 ± 3.78 min in Group 2, respectively. Pre- and postoperative MCV were 0.77 ± 0.30 and 1.16 ± 0.42 mm/min in Group 1 and 0.67 ± 0.16 and 0.89 ± 0.28 mm/min in Group 2, respectively. MCT and MCV were significantly improved postoperatively in both groups ($p < 0.001$). In addition, the postoperative MCT and MCV of Group 1 were significantly better than those of Group 2 ($p < 0.001$). Exposure to cigarette smoking and adenoid size had negative correlations with mucociliary clearance.

Conclusions: Otitis media was associated with impaired mucociliary clearance and further studies should be performed to demonstrate the causes of this deficiency.

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1. Introduction

Adenoid hypertrophy (AH) is lymphoid tissue on the nasopharynx roof that produces nasal obstruction. It may cause nasal discharge and stagnation of secretion [1] that damages nasal physiology due to disaffected mucociliary clearance. The upper respiratory tract is a long path with multiple recesses, including paranasal sinuses, Eustachian tubes, and middle ear cavities. Disorders along this tract may affect adjacent structures. The

relationship between AH and otitis media with effusion (OME), as well as the effect of AH on nasal mucociliary clearance, have been examined [2,3]. However, the effect of AH with OME (AHOME) on nasal mucociliary clearance has not been reported. In this study, pre- and postoperative mucociliary clearance in AH and AHOME patients was compared.

2. Materials and methods

This study included 64 patients with AH (31 with OME) age 3–18 years. Primary complaints included frequent snoring, nasal discharge, mouth breathing, and hyponasal voice. OME diagnosis was based on otoscopy and tympanogram. Air-fluid level or bubbles, lateralized position, and presence of increased vascularity in tympanic membrane were present in OME. Treatment with antibiotics (when needed), decongestants, and topical steroids was attempted before considering surgery. Patients were examined with 0° rigid endoscopy, and adenoid tissue size was categorized

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based on the choana obstruction grading described by Cassano et al. [4] (Grade 1: 25–50%, Grade 2: 50–75%, Grade 3: 75–100%). Patients were placed into two groups: Group 1—AH and Group 2—AHOME. Andersen saccharin test [5] (AST) was conducted before and 1 month after surgery to measure MCT. A 1.5 mm saccharin particle soaked in methylene blue was placed on the nasal cavity floor, ~10 mm behind the anterior end of the inferior turbinate. Patients were asked not to sniff, sneeze, smoke, eat, or drink during the test and to report when sweet perception occurred. Presence of methylene blue in the nasopharynx was used to establish test reliability. Adenoidectomy and tympanostomy tube insertion were performed as needed for each group. Patients whom glue were not found after myringotomy were excluded from study. Nasal cavity length was defined as the distance between the upper medial incisor and the inferior edge of the soft palate [6]. MCV was calculated by dividing nasal cavity length by MCT, which eliminates the effect of variables on MCT. Exposure to cigarette smoke was also recorded. Patients who had allergic rhinitis, severe inferior turbinate hypertrophy, underlying immunodeficiency, cystic fibrosis, prior nasal or adenoid surgery, active infection and lack of follow-up findings were excluded.

Pre- and postoperative MCT and size of adenoid, exposure to cigarette and AH-AHOME was compared with one-way ANOVA. Later, difference between pre- and postoperative MCT values was found and nasal MCT improvement values were calculated. MCT improvement values and size of adenoid, exposure to cigarette smoke and presence of OME initially analyzed with one-way ANOVA as univariate analysis. Because of observing significant correlation between MCT improvement values, cigarette and OME and non-significant correlation between adenoid size and MCT improvement values, to research the correlation between MCT improvement, OME and exposure to cigarette smoke multivariate analysis was applied with two-way ANOVA as both two variables accepted as independent. After statistical analysis, because cigarette and presence of OME was dicotom, post hoc was not applied additionally.

Data analysis used SPSS 16.0 (SPSS Inc., Chicago, IL, USA). Analyses performed included the Chi-square test, *t*-test, and Pearson's correlation test; statistical significance was defined as $p < 0.05$.

Ethics committee approval was obtained.

3. Results

Sixty-four patients, 36 male (56.2%) and 28 female (43.8%), with a mean age of 8.34 ± 2.98 years, were included in this study. Age and gender were not different between the groups ($p = 0.762$ and $p = 0.615$, respectively).

Pre- and postoperative MCT and MCV values are shown in Table 1. MCT and MCV were improved postoperatively in both groups. In addition, postoperative MCT and MCV values in Group 1

showed significantly more improvement than those in Group 2 ($p < 0.001$).

Grades 2 versus 3 adenoid sizes differed significantly according to occurrence of OME ($p < 0.041$). Both pre- and postoperative MCT were better in Grade 2 patients ($14.12 \rightarrow 9.58$ min) than Grade 3 patients ($16.65 \rightarrow 11.82$ min) ($p = 0.010$ and $p = 0.015$, respectively). However, when the effect of adenoid size on MCT improvement was investigated by one-way ANOVA, no significant difference was observed ($p = 0.922$). After observing adenoid size was not correlated with MCT improvement values, the effect of cigarette and presence of OME over nasal MCT improvement was analysed with multivariate analysis.

In preoperative comparison, no relationship was found between cigarette smoke exposure and OME ($p = 0.695$). However, cigarette smoke exposure had a significant negative effect on postoperative MCTs ($p = 0.001$) in AHOME patients. Cigarette smoke exposure (14.00 ± 3.72 min) versus no exposure (9.86 ± 2.41 min) ($p = 0.001$). After this, to research the effect of cigarette smoke exposure and presence of OME over MCT improvement, multivariate analysis was applied where both two parameters were accepted as independent and resulted that both two parameters had significant effect on MCT improvement ($p = 0.033$ and $p = 0.048$, respectively).

4. Discussion

The prevalence of OME was estimated at 34.8–41.1% based on a large survey of 8261 children under the age of 6 years in the United States [7], and an association between AH and OME has been reported. AH in children produces respiratory tract infections by causing mechanical obstruction, which acts as a source of infection, and by impairing nasal mucociliary clearance (MCC). In addition, adjacent respiratory tract areas, such as paranasal sinuses and middle ear cavities, lose defenses, thus leading to inflammation and infection. Recent studies reported that removing adenoid tissue prevents OME and increases MCV [8,9]. However, the relationship between MCC and OME had not to our knowledge been investigated.

MCT can be measured by means of AST [5] and scintigraphy [10]; however, AST is the most common and appropriate method for children. Test values may vary based on humidity, temperature, cigarette smoke exposure, nasal obstruction severity, environmental pollutants [11], oxygen therapy, anesthetics, and respiratory diseases (chronic obstructive pulmonary disease, cystic fibrosis, primary ciliary dyskinesia, bronchiectasis, and asthma). The mean MCT exhibits a wide range (4.4–9.9 min) in healthy children [12,13].

In this study, MCT improved in all patients, and postoperative MCT fell within normal limits in Group 1 (AH). Pre- and postoperative MCT in Group 2 (AHOME) were significantly worse than those in Group 1, and postoperative MCT fell into the normal

Table 1
Preoperative and postoperative nasal mucociliary clearance times and velocity scores. Examination of mucociliary clearance time (MCT; min) and mucociliary clearance velocity (MCV; mm/min) in patients with varying adenoid size (Grades 1–3), exposure to cigarette smoke, and presence (+) or absence (–) of otitis media with effusion (OME).

| | | N | Pre MCT | Post MCT | Pre MCV | Post MCV | p |
|-----------------|-------------|----|-------------|-------------|------------|------------|-------|
| Adenoid size | Grade-1 | 5 | 13.4 ± 2.6 | 9 ± 1.7 | 0.7 ± 0.1 | 1.1 ± 0.2 | 0.001 |
| | Grade-2 | 24 | 14.12 ± 5.2 | 9.58 ± 3.0 | 0.8 ± 0.3 | 1.2 ± 0.4 | 0.001 |
| | Grade-3 | 35 | 16.85 ± 4.1 | 11.82 ± 3.6 | 0.6 ± 0.2 | 1.0 ± 0.4 | 0.001 |
| Cigarette smoke | Exposed | 38 | 16.0 ± 5.2 | 11.9 ± 3.7 | 0.7 ± 0.3 | 0.9 ± 0.3 | 0.001 |
| | Non-exposed | 26 | 14.7 ± 3.5 | 9.1 ± 2.3 | 0.7 ± 0.2 | 1.2 ± 0.4 | 0.001 |
| OME (+) | | 31 | 16.0 ± 4.3 | 12.1 ± 3.7 | 0.69 ± 0.2 | 0.89 ± 0.2 | 0.001 |
| OME (–) | | 33 | 14.6 ± 4.8 | 9.4 ± 2.6 | 0.77 ± 0.3 | 1.16 ± 0.4 | 0.001 |

range. To clarify the relationship between MCT impairment and OME, the effects of cigarette smoke exposure and adenoid size were investigated. Although preoperative MCT did not differ according to adenoid size and cigarette smoke (Fig. 1), Group 2 had significantly worse postoperative MCT scores (Fig. 2). Statistical analyses revealed no relationship between adenoid size and MCT improvement. Subsequent univariate analyses were performed and indicated that cigarette smoke exposure and OME were both associated with significant MCT impairment.

The effect of cigarette smoke exposure on nasal cilia and MCT is controversial [14,15]. Decreased ciliary beat frequency and number of cilia as well as direct effects of cigarette smoke toxins are blamed for MCC impairment. In our study, no significant difference in MCT was found between smoke-exposed and non-exposed patients preoperatively ($p=0.372$); however, postoperative MCT combined with smoke exposure was worse than that in non-exposed individuals ($p=0.001$). But for this study it could be accepted as a limitation that the effect of cigarette smoked at home over children was not proved with objective parameters like blood cotinine levels. Surely further studies about this subject will be helpful at clarifying the pathophysiology of the disease.

Another factor that may negatively affect MCT is adenoid tissue size. The relationship between nasal obstruction and MCT as well as between adenoid tissue size and OME were investigated previously. However, the relationship between MCT and OME had not been examined. Acharya et al. [16] reported a significant correlation between Grade 3 adenoid size and OME. In this study, a significant correlation was found between OME and an adenoid size of Grade 2 or 3. Furthermore, MCTs were significantly impaired in Grade 2 and 3 adenoid sizes preoperatively ($p=0.015$ and $p=0.010$, respectively) (Spearman's correlation test $p=0.004$, $r=0.360$). In postoperative measurement, MCT improved in each adenoid group, with no significant differences among preoperative adenoid grade categories.

Collectively, OME and cigarette smoke exposure were highly related to MCT impairment, which eventually affected tubal and middle ear ciliary function independent of adenoid size. This impairment led to persistent infections, interrupted the middle ear's effusion healing process, and likely produced persistent OME.

This process generates a self-sustaining pathogenesis for AH and related diseases. AH creates an obstruction harbors infection,

which yields mucous stagnation, and decreased MCC and nasal pathogen removal. As a result, adenoid and adjacent tissues become a source of persistent inflammation, which further impairs MCC. Breaking this cycle is normally approached through adenoidectomy. Although improvements following adenoidectomy are often reported, the present study did not observe improved MCT in the AHOME group postoperatively. Clearly, another step is required to resume normal nasal physiology, and the best intervention would directly target MCC.

In addition, Wake et al. [17] reported a positive correlation between MCC and middle ear cell beat frequency in healthy patients. Based on this report, MCT should be adjusted to incorporate the middle ear cell healing process, especially for long-term follow-up in patients with OME. MCT could become a simple, inexpensive, and comfortable method of assessing middle ear cell healing. One limitation to our study was the absence of long-term follow-up MCT results.

5. Conclusion

To the best of our knowledge, this study was the first to report nasal MCC impairment in AHOME patients. Our results suggest that impaired nasal MCC might play a critical in OME etiology, suggesting that an OME treatment regimen should include nasal mucocurative interventions.

Conflict of interest

None declared.

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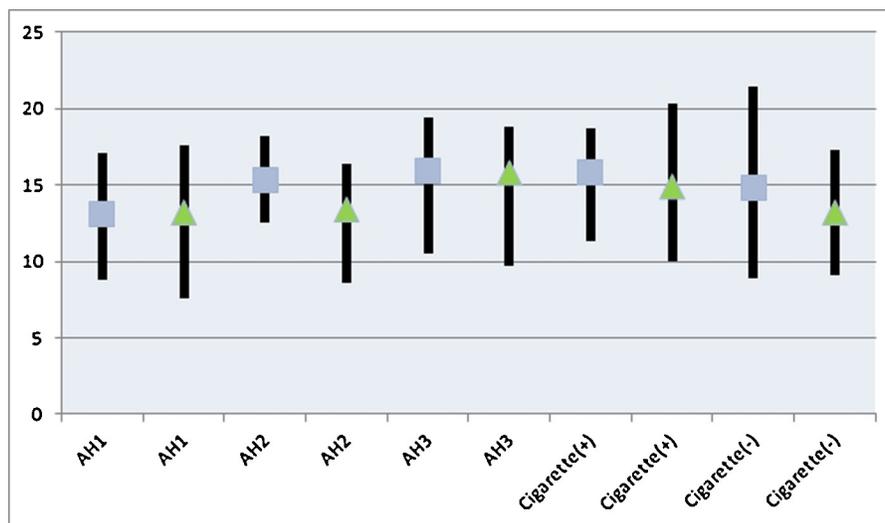


Fig. 1. Preoperative MCTs of OME (+) and (-) patients according to adenoid size and exposure to cigarette smoke. Blue square: OME (+); green triangle: OME (-); AH1: Adenoid hypertrophy Grade 1; AH2: Adenoid hypertrophy Grade 2; AH3: Adenoid hypertrophy Grade 3. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

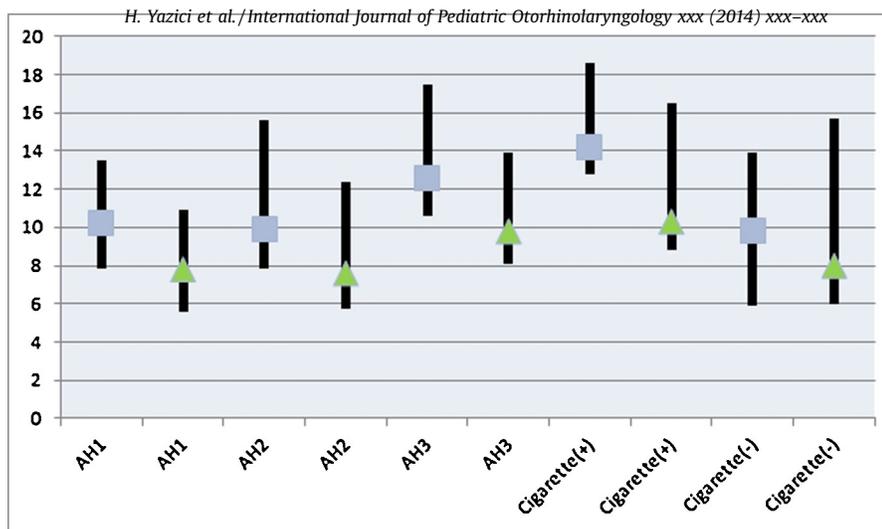


Fig. 2. Postoperative MCTs of OME (+) and (-) patients according to adenoid size and exposure to cigarette smoke. Blue square: OME (+); green triangle: OME (-); AH1: Adenoid hypertrophy Grade 1; AH2: Adenoid hypertrophy Grade 2; AH3: Adenoid hypertrophy Grade 3. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

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