ADENOID SIZE IN CHILDREN
WITH OTITIS MEDIA WITH EFFUSION

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SUMMARY – Otitis media with effusion (OME) is amongst the most common pediatric diseases and the most common cause of hearing loss in children. It is accepted that adenoid hypertrophy (AH) is related to OME incidence. Better understanding of the correlation between the relative size of AH and the incidence of persistent OME may provide evidence to support a more standardized approach to the diagnosis and treatment of OME. A retrospective study carried out between April 2016 and April 2018 collected data on 65 children aged 2-12 years, diagnosed with chronic OME and symptoms of AH, where conservative therapy failed. Pre-diagnostic data were collected from patient history, otoscopy, rhinoscopy, and oropharyngoscopy findings. Diagnostic workup included tympanometry, audiometry, and flexible nasal fiberoptic endoscopy. Adenoid grading was performed according to Cassano method after endoscopic visualization. Of the 65 patients, 37 were male and 28 were female. There was no statistically significant difference according to gender or average age. The highest incidence of persistent OME with AH was recorded in the youngest age groups (2-5 and 6-9 years). The most frequent AH grades were grade II (35.38%) and grade III (50.77%), yielding a statistically significant result. The most common presenting symptoms were hearing impairment, snoring, and nasal obstruction (100%, 64.62% and 60%, respectively). Higher AH grades are critical for persistence of OME and may cause conservative therapy failure.

Key words: Otitis media with effusion; Adenoid hypertrophy; Nasal fiberoptic endoscopy

Introduction

Otitis media with effusion (OME) is a disease defined by persistence of serous or mucous fluid in middle ear without signs of an acute infection. It is amongst the most common pediatric diseases and the most common cause of hearing loss in children. It is estimated that more than 50% of children are diagnosed with OME by the age of 1 year, and up to 90% of children by the time they have reached school age. Physicians must harbor high suspicion of this disease because OME does not appear with abrupt inflammatory symptoms and may go unnoticed as seemingly asymptomatic. However, symptoms such as sensations of aural fullness or hearing troubles may be apparent. Also, upon otoscopic observation, tympanic membrane may appear retracted and opacified, fluid levels or air bubbles may be visible, along with limited movement upon Valsalva maneuver. If left undiagnosed and untreated, persistent fluid impairs conductive hearing, diminishing the child’s perception in both noisy and quiet environments. The average hearing loss in OME includes a threshold of 25 dB and above 35 dB in 20% of cases, as compared to a child with normal hearing, who should not have hearing thresholds above 15 dB within the normal speech range. Furthermore, not only is hearing affected, but also the vestibular system, leading to poor balance. All of these conditions lead to poor speech development, intellectual lag, and difficulties in overall school performance.

Although the exact pathogenesis of OME is not clearly understood, it has been acknowledged that the etiology is multifactorial. However, two very signifi-
cant and interrelated factors are identified, i.e. adenoid hypertrophy (AH) and eustachian tube dysfunction. The eustachian tube permits pressure equalization in the middle ear via its opening in the nasopharynx. If there are obstructions, patency abnormalities, or poorly functional cilia, gases become absorbed, and the physiologic pressures become more negative, resulting in the pathognomonic transudate of OME. Children, compared to adults, have a higher risk to develop middle ear infections in general because their eustachian tube is anatomically narrower and horizontally placed, and has underdeveloped neuromuscular function. This leads to easier infection transfer and inclination towards pressure dysfunction. The adenoid, or the pharyngeal tonsil, is an antibody producing lymphatic tissue located in the superior part of the nasopharynx posteriorly, near the choana and opening of the eustachian tube. It grows during childhood, appearing largest in size in children between 3 and 7 years of age, and begins to regress in adolescence. Children younger than 7 years are more prone to asymptomatic effects of enlarged adenoid due to the relatively smaller volume of the nasopharynx and choanal opening. The prevalence of AH (pathologic enlargement) follows physiologic growth and regression pattern of the adenoid. Such symptoms may be a nasal sounding voice, difficulty breathing through the nose, night time snoring, and sleep disturbances. Such children rely on breathing through the mouth, thus maintaining a constantly ajar mouth for airflow. This chronic mouth breathing may later cause cranio-facial deformities and create the facial appearance called 'adenoid facies', presented as a 'long face' with an ajar mouth.

The etiology of AH is not well known, but allergies, upper respiratory infections, and chronic sinusitis have been recognized as preceding factors. These recurring infections lead to a hypertrophied and chronically infected adenoid, which then contributes to the pathogenesis of OME. Its influence on the pathogenesis of OME is two-fold: it may mechanically obstruct the eustachian tube, and its vegetations may serve as a reservoir of biofilm forming bacteria causing retrograde infections towards the eustachian tube and the middle ear. Other predisposing risk factors for OME include environmental factors such as living in lower socioeconomic conditions or exposure to smoke, and genetic factors such as cleft palate, ciliary dysfunction, and gastroesophageal reflux.

There is sufficient evidence that AH is an important co-factor in the development of OME, a very consequential disease. The intention of this study was to further investigate their correlation in order to provide evidence for designing a more standardized approach to the diagnosis and treatment of OME.

Subjects and Methods

The study selected 65 children from the Split University Hospital Centre, who had definitive indications for adenoidectomy with myringotomy with ventilation tube insertion between April 2016 and April 2018. All study children were initially treated conservatively for 3 months prior to any surgical intervention. Children younger than 2 years or older than 12 years were not included. Additionally, those with cleft palate, Down's syndrome, septal deviation, primary ciliary dyskinesia (Kartagener's syndrome), previous head or ear trauma, or previous myringotomy with ventilation tube insertion were excluded from the study.

Patient data were collected from pertinent diagnostic and preoperative tests and from surgical database. The initial screening consisted of the standard otolaryngologic workup including history, otoscopy, rhinoscopy, and oropharyngoscopy. During history taking, focus was put on questioning both the children and their parents about any complaints, such as hearing disturbances, sleep disturbances, nasal obstruction, allergies, etc. Diagnostic workup included tympanometry, audiometry, and flexible nasal fiberoptic endoscopy (NFE). During clinical examination, otoscopy findings noted the presence of a retracted and opacified tympanic membrane. On tympanometry, all patients had B type recordings, and tonal audiograms showed a conductive hearing loss of 25-35 dB. Audiogram results were only available for children aged ≥7 years due to the lack of patient cooperation at younger ages.

For the diagnosis and quantification of AH, the gold standard NFE was performed. The results were classified according to the method of Cassano. The grades were thus determined according to the percentage of choanal opening obstruction by the adenoid, as follows: grade I, adenoid obstructs less than 25% of the choanal opening; grade II, adenoid occupies 25%-50% of the choanal opening; grade III, adenoid occupies 50%-75% of the choanal opening; and grade IV, adenoid obstructing 75%-100% of the choanal opening.
Original images (Fig. 1) taken for this study show different AH categories.

Data analysis was performed by using MedCalc 18.2.1 program version (MedCalc Software, Ostend, Belgium). The level of statistical significance was set at \( p<0.05 \). The mean ± standard deviation (SD), median, and ranges were used to describe numerical variables. Kruskal Wallis test was performed to analyze any statistical differences in numerical variables among study groups. Analysis between two groups was done by Mann-Whitney U test. Statistical analysis of association of categorical variables was calculated by the \( \chi^2 \)-test, which yielded \( \chi^2 \) and \( p \). The Spearman coefficient correlation rho was also used to measure statistical dependence between two variables.

**Results**

Of the 65 patients included, 37 were boys and 28 were girls, age range 2-12 years (average age, 6 years). Overall, there was no statistically significant age difference \( (p=0.281) \). The patients were divided into 3 age categories.
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There were 32 children in the 2-5 age group, 30 children in the 6-9 age group, and three children in the 10-12 age group (Table 1). In each age group, the number of males and females was balanced, thus no statistical difference was noted ($\chi^2=0.352; p=0.839$). The highest incidence of OME with AH occurred in the 2-5 and 6-9 age groups, with a frequency of 49.23% and 46.15%, respectively (Fig. 2). Table 2 shows distribution of AH grades according to our sample. Grade I was recorded in two patients, which accounted for only 3.08% of total AH grading. Grade II was recorded in 23 (35.38%), grade III in 33 (50.77%), and grade IV in 7 (10.77%) cases. Accordingly, grades II and III were most frequently recorded, together accounting for 84.6% of cases. In order to get uniform distribution, the expected number of children per grade should be 16. Thus, according to the $\chi^2$ analysis, the observed versus expected number of children per grade should be 16.

### Table 1. Age and gender distribution of patients (N=65)

| Age group (years) | Total | Male | Female | \( p^* \) |
|-------------------|-------|------|--------|----------|
| 2-5               | 32 (49%) | 19 (51%) | 13 (46%) | 0.839 |
| 6-9               | 30 (46%) | 16 (43%) | 14 (50%) |      |
| 10-12             | 3 (5%)   | 2 (2%)   | 1 (4%)   |      |
| Total             | 65 (100%) | 37 (100%) | 28 (100%) |      |

Data presented as absolute number and percentage in parentheses; *$\chi^2$-test*

### Table 2. Distribution of adenoid hypertrophy grades found by flexible endoscopy

| Grade | Frequency | %  |
|-------|-----------|----|
| I     | 2         | 3.08 |
| II    | 23        | 35.38 |
| III   | 33        | 50.77 |
| IV    | 7         | 10.77 |

### Table 3. Relationship between age groups and adenoid hypertrophy grades

| Age group (years) | Grade I | Grade II | Grade III | Grade IV | Total   |
|-------------------|---------|----------|-----------|----------|---------|
| 2-5               | 1       | 11       | 18        | 2        | 32 (49.23%) |
| 6-9               | 0       | 10       | 15        | 5        | 30 (46.15%) |
| 10-12             | 1       | 2        | 0         | 0        | 3 (4.62%)   |
| Total             | 2 (3.08%) | 23 (35.38%) | 33 (50.77%) | 8 (12.31%) | 65 (100%) |

Data presented as absolute number and percentage in parentheses
expected data resulted in a statistically significant difference ($\chi^2=35.9; p<0.001$).

Table 3 compares patient age groups to AH grade. In the 2-5 age group, AH grades I-IV were recorded in 1, 11, 18 and 2 cases, respectively. In the 6-9 age group, AH grades I-IV were recorded in 0, 10, 15 and 5 cases, respectively. In the 10-12 age group, grade I was recorded in only one patient and grade II in two patients. According to Spearman's rho coefficient, the correlation between age groups and AH grade was not statistically significant ($rs=0.013; p=0.921$). When comparing genders (Fig. 3), excluding the groups of two patients with grade I AH, there was no statistically significant gender difference in the distribution of grades II, III and IV ($\chi^2=1.45; p=0.484$).

### Table 4. Presenting symptoms in patients with adenoid hypertrophy (N=65)

| Presenting symptom | n  | %   |
|--------------------|----|-----|
| Hearing impairment | 65 | 100 |
| Mouth breathing    | 37 | 56.92 |
| Nasal obstruction  | 39 | 60 |
| Snoring            | 42 | 64.62 |
| Sleep disturbances | 28 | 43.08 |
| Voice changes      | 21 | 32.31 |
| Headache           | 14 | 21.54 |
| Epistaxis          | 8  | 12.31 |

Considering data collected from patient histories, all the patients presented with hearing impairment. In addition to hearing concerns, the most frequently presenting symptoms of AH were snoring (64.62%), nasal obstruction (60%), and mouth breathing (56.92%) (Table 4). The remaining presenting symptoms reported included sleep disturbances, voice changes, headache, and epistaxis.

### Discussion

The results revealed that the youngest age groups (2-5 and 6-9 age groups) had a higher prevalence of OME, totaling 95.34% of study patients, with 49.23% in the 2-5 age group and 46.15% in the 6-9 age group. A similar study conducted in Kochi, India, which included children aged 3-12 years, also showed that OME was most prevalent in the 5-7 age group, which accounted for 59.5% of the study sample ($p<0.01$). However, the results of the above-mentioned study differ from ours, i.e. the youngest age group (3-5 years) had a prevalence of only 13.33% compared to 49.23% in the present study. Overall, these statistics show that patients of younger age are more likely to have OME.

Our results did not show a statistically significant gender difference in the prevalence of OME ($p=0.281$). This is comparable to the study by Khayat et al., which...
found no statistically significant gender difference either\textsuperscript{13}. However, this does not correlate to the study conducted on 467 pediatric patients in Fiji. Their results showed that male gender was in fact a significant predictor of OME\textsuperscript{20}. Another study conducted in Nigeria also concluded on the male predominance with a ratio of 7:1\textsuperscript{15}. Thus, for now, the role of gender and incidence cannot be defined with certainty.

Our study showed a statistically significant incidence of patients, already at the stage of interventional treatment, with AH grades II and III. In fact, 35.38% of the patients had grade II AH, and 50.77% had grade III AH, yielding a total of 84.6% of all study patients with OME having a high grade of AH. The observed distribution was statistically different from the expected uniform distribution ($\chi^2=35.9; p<0.001$). This leads to a conclusion that those children with failed conservative treatment for OME and therefore scheduled for adenoidectomy and myringotomy are most likely suffering from grade II or III AH. Zhu et al. also consider that high-grade AH is related to unsuccessful treatments of OME. In their study, recurrence of OME was significantly affected by grades III and IV AH ($p<0.05$)\textsuperscript{21}. Similarly, Nwosu et al. found high-grade AH to be a statistically significant risk factor for OME\textsuperscript{22}. Another study, conducted similarly to ours, which classified AH according to the method by Clemens and McMurray that compares enlargement with vertical height rather than choanal opening area, recorded grade III AH in the majority of patients\textsuperscript{11}. However, another study designed similarly to ours, did not result in a similar conclusion. The authors also examined adenoid via flexible endoscope and graded tissue in four stages according to choanal opening obstruction; however, study results showed no significant correlation between persistent OME and AH\textsuperscript{23}. A study by Skoludik et al. compared both adenoid size by choanal obstruction and by eustachian tube ostium obstruction. Their results showed that it is not the size of the adenoid (choanal obstruction) that is relevant, but rather its contact with torus tubarius\textsuperscript{24}. A limiting factor to our study could have been that adenoid contact with torus tubarius was not investigated, but we were focused only on adenoid tissue in relation to choanal opening.

As for the presenting signs of AH, all our patients had hearing impairment (100%), and the investigation revealed a conductive hearing loss of 25-35 dB. Other most common presenting signs of AH were mouth breathing (56.92%), nasal obstruction (60%), and snoring (64.62%). The remaining presenting symptoms reported were sleep disturbances, voice changes, headache, and epistaxis. In a study by Vijayan et al., mouth breathing was the most common presenting factor (78.1%), followed by complaints of nasal obstruction (73%) and decreased hearing (52.5%)\textsuperscript{18}. However, in one study, the presenting signs did not seem to correlate with our results, with cough and catarrh being the most common signs (73.1% and 69.2%, respectively), and mouth breathing being one of the least common signs (15.4%)\textsuperscript{15}. Thus, although there are different signs that are repeatedly observed in association with AH, it cannot be concluded that there is a specific combination of symptoms that can be used exclusively to diagnose the severity of AH.

Although there is other research that was found to support our results, our study results may not be used as evidence for strong association between AH and persistent OME. Regression analysis, an analysis that requires a control group, should have been performed to produce stronger results.

**Conclusions**

According to the results, we conclude that the size of AH is critical in OME persistence, and that children with a higher grade of AH have a higher risk of OME persistence, leading to conservative treatment failure and requiring surgical intervention. Study results point to a conclusion that there is no difference between genders or individual age groups at risk. The most common presenting symptoms of AH were recorded, including hearing impairment, snoring, nasal obstruction, and mouth breathing. A practicing physician who encounters a child with any of the above-mentioned complaints must stay alert for the potential diagnosis of AH and proceed with additional diagnostic workup.

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Sažetak

VELIČINA ADENOIDNIH VEGETACIJA U DJECE S UPALOM SREDNJEG UHA S IZLJEVOM

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Kronična upala srednjeg uha s izljevom jedna je od najučestalijih pedijatrijskih bolesti i najčešći uzrok gubitka sluha u djece. Poznato je da je adenoidna hipertrofija (AH) povezana s učestalošću upale srednjeg uha s izljevom. Bolje razumijevanje njihove povezanosti bi moglo ponuditi dokaz za novi standard u dijagnostici i liječenju kronične upale srednjeg uha s izljevom. U ovo retrospektivno istraživanje provedeno od travnja 2016. do travnja 2018. godine bilo je uključeno 65 djece u dobi od 2-12 godina s dijagnozom kronične upale srednjeg uha s izljevom i simptomima AH, kod koje je konzervativna terapija bila neuspješna. Svoj djeci je uzeta detaljna heteroanamneza i pred-dijagnostičke pretrage koje uključuju otoskopiju, rinoskopiju i orofaringoskopiju. Dijagnostičke pretrage uključuju fiberendoskopiju epifaringa i audiološku obradu koja obuhvaća timpanometriju i tonalnu audiometriju. Gradiranje AH je napravljeno prema sustavu Cassano s endoskopskom vizualizacijom adenoidnih vegetacija. Od 65 djece, bilo je 37 dječaka i 28 djevočica, bez statistički značajne razlike u broju i spolu u svim dobним skupinama. Rezultati rada pokazuju da je najčešća incidencija kronične upale srednjeg uha s izljevom i AH u mladoj dobi (2-5 i 6-9 godina). Najčešći gradusi su bili AH II. (35,38%) i AH III. (50,77%) (p<0,001). Najčešći prezentirajući simptomi bili su gubitak sluha (100%), noćno hrkanje (64,62%) i otežano disanje na nos (60%). Veći gradus AH povezan je s većom učestalosti upale srednjeg uha s izljevom te može uzrokovati neuspjeh konzervativne terapije.

Ključne riječi: Upala srednjeg uha s izljevom; Adenoidna hipertrofija; Nazalna fiberendoskopija