The effect of adenotomy, allergy and smoking on microbial colonization of upper aerodigestive tract in children

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ABSTRACT

Aim: To evaluate microbial colonization of upper aerodigestive tract in children and to investigate the influence of adenoid hypertrophy, allergy and exposition to cigarette smoke on presence of pathogens.
Methods: In 43 children with adenoid hypertrophy and 17 healthy children bacterial culture was performed by a swab from middle nasal meatus, nasopharynx and tonsils. The effect of adenotomy, presence of allergy and exposure to passive smoking on bacterial colonization were investigated.
Results: Identification of potentially pathogenic bacteria in upper aerodigestive tract was significantly higher in children with adenoid hypertrophy compare to control group. Adenotomy was associated with significantly decreased colonization by potential pathogens. Allergy was diagnosed in 33 % children with adenoid hypertrophy. Presence of allergy and exposure to tobacco smoke were associated with significantly more often colonization by potentially pathogenic bacteria in the upper aerodigestive tract.
Conclusion: Increased colonization of upper aerodigestive tract by potential pathogens and their significant decrease after adenotomy indicate the role of pathogenic bacteria in the etiopathogenesis of adenoid hypertrophy. Allergy and tobacco smoke exposure are related to increased colonization by potentially pathogenic bacteria in the upper aerodigestive tract.

KEYWORDS

pathogens – upper airways – adenoid hypertrophy – allergy – smoking

SÚHRN

Uhliarova B., Bugova G., Jesenak M., Pechacova S., Hamarova M., Hajtman A.: Efekt adenotómie, alergie a fajčenia na mikrobiálnu kolonizáciu horného aerodigestívneho traktu u detí

Cieľ: Vyšetriť mikrobiálnu kolonizáciu horného aerodigestívneho traktu a sledovať vplyv adenoidných vegetácií, alergie a expozície cigaretovému dymu na prítomnosť patogénov.
Metodika: Do prospektívnej štúdie bolo zaradených 43 detí s adenoidnými vegetáciami a 17 zdravých detí. Autori určovali mikrobiologický nález v strednom nosovom priechode, nosohltane a podnebných mandliach. Sledovali efekt vykonania adenotómie, prítomnosti atopie a expozície cigaretovému dymu na mikrobiálnu kolonizáciu.
Výsledky: Patogénne baktérie sme izolovali signifikantne častejšie u detí s adenoidnými vegetáciami v porovnaní so zdravými deťmi. Adenotómia viedla k signifikantnému zníženiu kolonizácie horného aerodigestívneho traktu patogénmi. Atopiu sme diagnostikovali u 33 % detí s adenoidnými vegetáciami. U detí s atopiou ako aj u detí, ktoré boli exponované cigaretovému dymu sme detegovali signifikantne vyššie zastúpenie patogénov.
Záver: Infekcia zohráva úlohu v patogenéze adenoidných vegetácií, čomu napovedá zvýšený výskyt patogénnych baktérií a ich pokles po adenotómii. Alergia ako aj expozícia tabakovému dymu sú spojené so zvýšenou incidenciou patogénnych baktérií v horných dýchacích orgánov.

KĽÚČOVÉ SLOVÁ:

patogénne baktérie – horné dýchacie orgány – adenoidné vegetácie – alergia – fajčenie

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INTRODUCTION

The upper aerodigestive tract is the most common point of entry for antigens and pathogens and serves as a first step in interaction between microorganisms and immune system. Lymphoid tissue of the upper aerodigestive tract, collectively defined as the Waldeyer's ring, is equivalent to MALT (*mucosa associated lymphoid tissue*) or GALT

(*gut associated lymphoid tissue*), that is deeply involved in the innate and adaptive immune response [1].
Pharyngeal tonsil hypertrophy, also called adenoid hypertrophy or adenoid vegetation (AV) is detected in approximately one-third of the general paediatric population with maximum of appearance in pre-school children (60%). Adenoid hypertrophy is a common cause of upper-airway obstruction in paediatric patients and can have a significant influence on the health of the child. Children who have hypertrophic adenoids often exhibit nasal obstruction, snoring, sleep apnea, otitis media with effusion and craniofacial abnormalities [2, 3, 4]. The adenoids can serve as a bacterial reservoir that contributes to recurrent and chronic infections in children such as otitis media and rhinosinusitis [1, 5].
The purpose of this study was to analyse the microbial colonization of upper aerodigestive tract in children. The effect of adenotomy, allergy and exposition to cigarette smoke on presence of pathogens was evaluated to see whether these risk factors influence the bacterial colonization of upper aerodigestive tract.

MATERIAL AND METHODS

Design of the study

The prospective study was conducted with 60 children (age 2–12 years) surgically treated at the Department of Otorhinolaryngology, Head and Neck Surgery, Comenius University, Jessenius Faculty of Medicine, University Hospital in Martin, Slovakia. Forty three children enrolled in the study were scheduled to endoscopic adenotomy for adenoid vegetation. Seventeen children admitted for surgical treatment for other reason (e.g. goiter, medial or lateral neck cyst) were eligible as controls if they showed no signs of pathological enlargement of adenoid tissue on nasal endoscopy.
Children with systemic or local antibiotics treatment within the 2 weeks before enrolment, recent respiratory infection, increased level of C-reactive protein and those with recurrent tonsillitis were excluded from the study. Children with other confounding pathologies such as septal deviation, inferior turbinate hypertrophy, rhinosinusitis or hypertrophied palatine tonsils) were also excluded from the study.
Presence of allergy, smoking exposure and bacterial colonization of upper aerodigestive tract were recorded. Patients in control group had no comorbidity and their parents were non-smokers. Differences in bacterial colonization according to the presence of allergy, smoking exposure were evaluated. The effect of these atopy and smoking on bacterial colonization was evaluated only in children with adenoid vegetations (due to the homogenity of the investigated group).
Six months after endoscopic adenotomy, the effect of adenotomy on microbial colonization of upper aerodigestive tract, frequency of infection of upper airways and improvement of nasal symptoms (obturation, snoring, anterior or posterior nasal drainage) were analysed.
The study was approved by the Ethics Committee of Jessenius Medical Faculty, Comenius University in Martin, Slovakia. An informed consent was signed by all parents of participating children.

Allergy

The prevalence of allergy was recorded. Allergy was linked to the questionnaire data about the presence of allergic diseases in personal and family history of the patients, presence of symptoms and objective findings by nasal endoscopy. In addition, allergy was confirmed by positive skin prick test to common inhalant allergens: *Dermatophagoides pteronyssinus, Dermatophagoides farinae*, mixed spring trees (*Betula verrucosa, Corylus avellana, Alnus glutinosa*), mixed grasses (*Avena sattiva, Hordeum vulgare, Secale cereale, Triticum sativa*), wormwood, *Alternaria alternata, Aspergillus fumigatus*, birch, cat and dog dander (Stallergenes, France). The tests were performed as stated by the European Academy of Allergy and Clinical Immunology [6]. A prick test was defined positive if the wheal was ≥ 3 mm in its longest dimension. Children with at least one positive skin prick test to common inhalant allergens were considered as atopic.

Smoking exposure

Only exposure to parental smoking was recorded, because parents were considered to be the closest individuals caring for the child. Parent who smoked 10 or more cigarettes per day during at least the last 5 years was defined as a smoker. Non-smoker was a parent who never smoked or did not smoke in the last 5 years. Parents who smoked more than 0 cigarettes per day and less than 10 cigarettes per day were excluded from the study (even if they met other criteria). The level of exposure when smokers were other than the parents (e.g. grandparent or sibling) and parent was a non-smoker, could not be evaluated and those cases were excluded from the study. Exposure to smoke other than tobacco smoke was not recorded.

Bacteriology

Differences in bacterial colonization of middle nasal meatus, nasopharynx and tonsils were analysed. Middle nasal meatus and nasopharyngeal swab specimens were received under endoscopic control (the place of obtaining the microbial sample was visualised by rhinoendoscop that was inserted into nasal cavity). Material for bacteriological evaluation was obtained by using sterile cotton-wool swabs and transported in Stuart's transport medium to the microbiological laboratory within 2 to 4 hours. The swab was inoculated onto Sheep blood agar (Columbia Bio-Rad, Bratislava, Slovakia), Chocolate agar with bacitracin disc, Mac Conckey agar (Bio-Rad, Bratislava, Slovakia) and placed into a 7% CO_2 incubator at 37 °C. The plates were examined after 18 to 24 hours of incubation. The incubation was further extended to 48 hours to detect the slow-growing microbes. Identification of colonies to genus or species level was based upon typical colony morphology by subculture, Gram stain, standard rapid tests (catalase, pyrrolidonyl aminopeptidase - PYR and oxidase tests), identification by latex agglutination tests and biochemical tests

Statistical analysis

Frequencies of categorical data were tabulated and evaluated with chi-square test using the Yates's correction. For other data, median and interquartile range was calculated and tested with the Kruskal-Wallis or Mann-Whitney tests. The statistical analysis was performed

Table 1. Demographic and clinical data of study population

	Total (n = 60)	AV (n = 43)	Control (n = 17)
Age (yr)	6.2 ± 3.3	5.9 ± 2.7	6.5 ± 3.9
Girls	30 (50%)	20 (47%)	7 (41%)
Boys	30 (50%)	23 (53%)	10 (59%)
Allergy	14 (23%)	14 (33%)	0
SHS	23 (38%)	23 (53%)	0

AV – adenoid vegetation, n – number of patient, SHS – second hand smoke, data are shown as median ± SD

with STATISTICA Cz 10. All conclusions were based on a significance level of $P < 0.05$.

RESULTS

Sixty children (30 boys, 30 girls, age 6.2 ± 3.3 years) were prospectively enrolled in the study. Forty three children (20 girls, 23 boys, age 5.9 ± 2.7 years) enrolled in the study were scheduled to endoscopic adenotomy for adenoid vegetation. Seventeen children (7 girls, 10 boys, age 6.5 ± 3.9 years) were in control group. Allergy was diagnosed in 14 (33 %) children with AV. Among 43 children with AV, 23 (53 %) were exposed to second hand smoke. All children in control group were non-allergic and had non-smoking parents. Demographic and clinical data of the study population are shown in Table 1.

Microorganisms isolated from upper aerodigestive tract are presented in Table 2. Coagulase-negative *Staphylococcus* species, *Corynebacterium* species, viridans *Streptococci* and *Neisseria* species are considered as commensals of upper respiratory tract. Other species of identified bacteria in our study were considered as potential pathogens.

We found significantly more intense colonization by potentially pathogenic bacteria in nasopharynx and tonsils in children with adenoid vegetations compare to controls ($P = 0.024$, $P = 0.041$, respectively) (Figure 1). In children with allergy, potential pathogens were significantly more often isolated from middle nasal meatus, nasopharynx and tonsils compare to non-allergic children ($P = 0.012$, $P = 0.006$, $P = 0.021$, respectively) (Figure 2). There was significantly more intense colonization by potentially pathogenic bacteria in middle nasal meatus and tonsils in children exposed to SHS compare to children with non-smoking parents ($P = 0.015$, $P = 0.039$, respectively). In nasopharynx, there were no significant differences in the presence of potentially pathogenic bacteria between children exposed and non-exposed to tobacco smoke (Figure 3).

Six months after adenotomy, control microbiological investigation of upper aerodigestive tract showed significantly less intense colonization of potential pathogens compare to their occurrence before surgery (Figure 4). Adenotomy decreased frequency of infection of upper airways in 41 (95%) of children and in all children improved symptoms of upper airway obstruction.

DISCUSSION

Chronic nasal obstruction due to adenoid hypertrophy is the most common health problem affecting children and adenotomy is one of the most common surgical procedures performed in this age group [2].

Prolonged antigenic exposure associated with chronic inflammation play an important role in etiopathogenesis of pharyngeal tonsil enlargement. Several studies showed that colonization of nasopharynx by pathogens is significantly higher in children with adenoid hypertrophy suggesting their involvement in chronic adenoiditis [7, 8].

In this study, we investigated the colonization of the nasopharynx in children receiving adenotomy using a traditional culture method. Our data showed that *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae* and *Moraxella catarrhalis* constitute major microorganisms cultured from upper aerodigestive tract. These findings are similar to the previous studies [1, 7, 9]. Healthy children were generally colonized with relatively non-pathogenic microbes. Control microbiological investigation of upper aerodigestive tract six

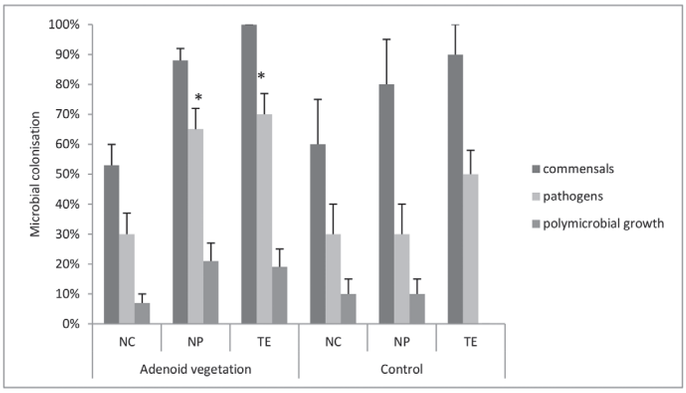

Figure 1. Differences in microbial colonization of upper aerodigestive tract
NC – nasal cavity, NP – nasopharynx, TE – tonsils, polymicrobial growth – the presence of 2 or more potential pathogens, *p < 0.05, data are shown as median ± SD.

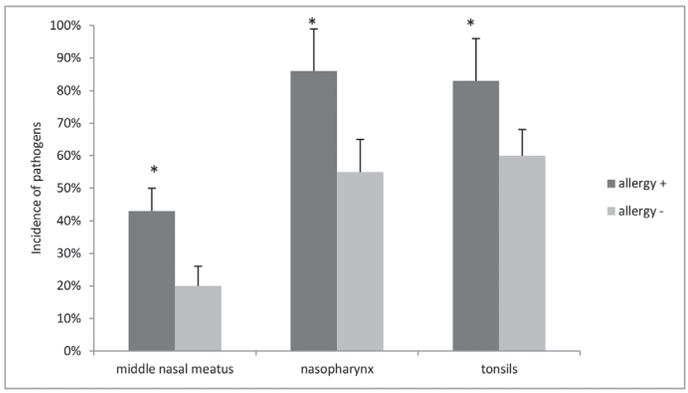

Figure 2. Pathogens in upper aerodigestive tract and presence of allergy
allergy+ – allergy present, allergy- – allergy absent, *p < 0.05, data are shown as median ± SD.

Table 2. Bacterial species isolated from upper aerodigestive tract

	Adenoid vegetation			Control		
Bacteria	**NC**	**NP**	**TE**	**NC**	**NP**	**TE**
Gram-positive						
Coagulase-negative Staphylococcus	14 (33%)	2 (5%)	0	7 (41%)	3 (17%)	0
Corynebacterium species	9 (21%)	4 (9%)	0	5 (29%)	0	0
Viridans Streptococci	1 (2%)	35 (81%)	42 (98%)	0	12 (71%)	15 (88%)
Streptococcus pneumoniae	6 (14%)	5 (12%)	2 (5%)	0	0	0
Streptococcus agalacticae	0	2 (5%)	4 (9%)	0	0	0
Beta-hemolytic Streptococci	0	3 (7%)	4 (9%)	0	0	0
Staphylococcus aureus	2 (5%)	10 (23%)	14 (33%)	3 (17%)	3 (17%)	5 (29%)
Gram-negative						
Neisseria species	0	11 (26%)	21 (49%)	0	7 (41%)	7 (41%)
Haemophilus influenzae	2 (5%)	12 (28%)	11 (26%)	2 (12%)	5 (29%)	3 (17%)
Moraxella catarrhalis	7 (16%)	6 (14%)	3 (7%)	2 (12%)	0	0

NC – nasal cavity, NP – nasopharynx, TE – tonsils

month after adenotomy showed significantly less intense colonization by potential pathogens compare to their occurrence before surgery. These results indicate the role of pathogens in pathogenesis of adenoid hypertrophy. Anaerobic bacteria may also potentially contribute to the etiopathogenesis of adenoid hypertrophy. In this study, the presence of anaerobic bacteria was not assessed.

Bacterial colonisation of upper aerodigestive tract may be also influenced by host factors such as age, immunity, sibling number, crowding, season, use of antibiotics, acute respiratory tract infection, vaccine application, and passive smoking exposure [7, 10, 11]. In our study, the possible effect of allergy and exposure to tobacco smoke on microbial colonisation was evaluated.

Allergy is frequent in children, affecting up to 40% of the general population, and may cause the open-mouth posture. The possible correlation between allergy and adenoid hypertrophy has been investigated by some studies that reported a positive association between the two disorders [12, 13, 14]. In the present study allergy was diagnosed in 33% of children with adenoid hypertrophy. There was significantly higher rate of potentially pathogenic bacteria carriage in allergic compared to non-allergic children. The increased carriage rate in children with allergy was seen in the middle nasal meatus, nasopharynx and also tonsils.

The mechanism by which allergy is associated with the carriage of potentially pathogenic bacteria is not fully understood. One of the possible explanations is the overstimulation of immune system caused by airborne allergens with subsequent enlargement of adenoid lymphatic tissue that serves as a reservoir of pathogens [15]. Further, several studies showed that allergy is associated with changes of respiratory mucosa, such as epithelial damage, goblet cell hyperplasia, alterations of mucociliary transport and smooth muscle hypertrophy [16, 17]. As a consequence, more intense colonization of upper respiratory tract by the pathogenic bacteria might be a result of impaired local defence mechanisms of respiratory mucosa.

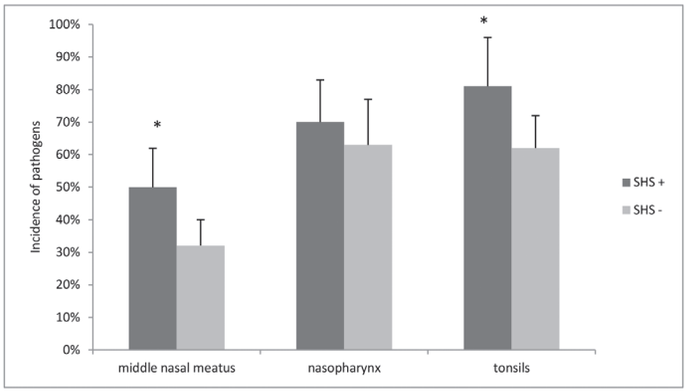

Figure 3. Pathogens in upper aerodigestive tract and tobacco smoke exposure
SHS – second hand smoke, *p < 0.05, data are shown as median ± SD.

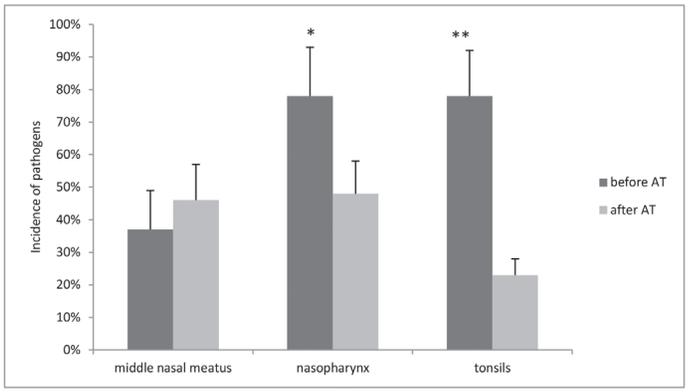

Figure 4. Pathogens in upper aerodigestive tract before and after adenotomy
AT – adenotomy, *p < 0.05, **p < 0.005, data are shown as median ± SD.

Taken together, allergy can be considered as a risk factor for the development of adenoid hypertrophy in children with atopic sensitization to respiratory allergens. Moreover, allergy significantly contributes to the increase respiratory tract infections and chronic stimulation of upper airways mucosal lymphatic tissue. Based on these results we emphasize that all children with adenoid hypertrophy indicated for surgical intervention should be also examined by immunoallergologist. Early diagnosis and proper treatment of allergy lead to improvement of quality of life for these patients, avoiding functional and structural changes of upper and lower airways.

Smoking is another factor that influences the microbial colonization of respiratory tract. Both smoking and exposure to tobacco smoke are associated with upper and lower respiratory tract diseases, such as acute otitis media, asthma, wheezing, cough, bronchitis, pneumonia and impaired pulmonary function [18]. In children, the intensity of exposure to environmental smoking correlates with respiratory infection rates, especially if the parent smokes in the same room as the child [19].

In the present study, we demonstrated that children exposed to smoking by parents had a significantly higher rate of potentially pathogenic bacteria carriage than did children who were not exposed to tobacco smoke. The increased colonization by potentially pathogenic bacteria may be a predisposing factor to recurrent infection of upper airways and also to additional spread of pathogens, resulting in lower respiratory tract infections. Our findings might explain previous reports demonstrating that children exposed to environmental tobacco smoke more frequently experience respiratory infections, such as acute otitis media and pneumonia [19, 20].

Exposure to tobacco smoke causes remodelling of respiratory mucosa with subsequent alterations of mucociliary transport [21, 22]. Furthermore, tobacco smoke also inhibits interleukin 8 and human β-defensin in sinonasal epithelial cell cultures derived from patients with chronic rhinosinusitis [23]. These findings suggest that cigarette smoke may have a suppressive function on innate immunity of upper airways. Taken together, more intense colonization of upper aerodigestive tract by the pathogenic bacteria might be a result of impaired local defence mechanisms of respiratory mucosa. Increased growth of pathogens in upper respiratory tract in children exposed to SHS when compared with children born to non-smoking parents found in our study also confirmed the role of tobacco smoke in pathogenesis of respiratory tract infections in those children.

CONCLUSION

Increased colonization of upper aerodigestive tract by potential pathogens and their significant decrease after adenotomy indicate the role of pathogenic bacteria in the etiopathogenesis of adenoid hypertrophy. Allergy and tobacco smoke exposure are related to increased colonization by pathogenic bacteria in the upper aerodigestive tract. The high prevalence of allergy in children with adenoid hypertrophy emphasize the importance of a complete ENT and immunoallergologic investigations, because early diagnosis and proper treatment lead to improvement of quality of life for these patients, avoiding functional and structural changes of respiratory mucosa.

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Errata

Uvádíme správný titul korespondující autorky u článku "Trend and challenge in mother-to-child transmission of syphilis", který byl publikován v předchozím čísle našeho časopisu (č. 1, s. 24–29):

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