Passive smoker exposure as a risk factor of otitis media with effusion in children

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ABSTRACT

Otitis media with effusion (OME) is a middle ear disorder often found in children and can cause hearing loss, delayed language acquisition, and behavioral disorders. Cigarette smoke exposure is one of the environmental factors that may raise the incidence of OME. This study aimed to identify the prevalence of OME and the risk of cigarette smoke to OME using urinary cotinine level examination, along with the characteristics of smoking history in the family. This cross-sectional study included 398 children aged 0-14 who underwent history taking, questionnaire completion, ear/nose/throat (ENT) examination, and tympanometry. OME patients based on ENT and tympanometry examination underwent urinary cotinine examination using lateral flow chromatographic immunoassay technique. Of the 398 children, five children were diagnosed with OME. The prevalence rate of OME was 1.3%. No OME patients were exposed to cigarette smoke based on urine cotinine levels of >200 ng/mL. However, based on history taking, one of five patients with OME is a passive smoker, with characteristics of active smoking history in the family (the father). In conclusion, there was no proportion of positive urinary cotinine levels in children with OME. This is an open access article under the CC BY-SA license.

1. INTRODUCTION

Otitis media with effusion (OME) is an inflammation of the middle ear without signs of acute infection and tympanic membrane perforations. OME is commonly found in children, with a prevalence of 17.89% in Kuala Lumpur, Malaysia, and 15.5% in Upper Egypt. It is estimated that 80% of children will have at least one OME episode by age 10. OME is often asymptomatic and the diagnosis is usually late and causes complications [1]. Persistent OME may lead to tympanic membrane damage, hearing loss, delayed language acquisition, and behavioral disorders [2]. It can also lead to cognitive dysfunction, academic failure, and quality of life reduction [3], [4]. OME is caused by Eustachian tube dysfunction and other problems related to middle ear ventilation. Several risk factors have been suggested for OME, including cigarette smoke exposure [5].

Passive smoking or secondhand smoke is exposure to tobacco smoke as a result of other people’s active smoking [6]. The proportion of children younger than 15 years old in Indonesia exposed to secondhand smoke at home is 79% [7]. Children are vulnerable to secondhand smoke exposure because they spend most of their time at home and have limited control over their environment [6], [7]. Cigarette smoke exposure harms children and infants because it can increase the risk of respiratory infection, sudden infant death syndrome, and...
more severe asthma and affect their physical development. It has also been identified as a risk factor for other airway diseases, including otitis media [8]. A study by Kiris et al. [9] reported secondhand smoke as one of OME's most significant risk factors. On the other hand, a study by Humaid et al. [10] found no significant correlation between passive smoking exposure and the development of OME. However, those studies assessed cigarette smoke exposure in children by only subjectively asking about their parents’ smoking habits using a questionnaire. Based on the facts above, it is important to find the correlation between secondhand smoke and OME in children using more objective measurements. This study aims to identify the prevalence of OME and the risk of secondhand smoke to OME within the children population.

2. METHOD

This descriptive cross-sectional study conducted a survey of children in East Jakarta and assessed their urine cotinine as a risk factor for cigarette exposure in the OME group. The inclusion criteria include children aged 0-14 who lived in East Jakarta and willingness to participate in the study. The exclusion criteria include uncooperative, smoking, or children with tympanic membranes that could not be evaluated (ear canal stenosis, atresia, and unextractable ear cerumen). The subjects were chosen using a multistage stratified random sampling, stratified from district to village based on population density. From the chosen district, group samples were taken using random sampling by choosing one sub-district out of each district and based on the probability.

A total of 407 children in the district were chosen from the population. However, there were nine children with unextractable ear cerumen and thus excluded from the study. Altogether, 398 children met the criteria. The children underwent history taking, questionnaire completion with their parents’ help, weight and height measurement, ear, nose, and throat (ENT) examination, and tympanometry. Our study used Intercoustics Impedance Audiometer AT-235 for tympanometry examination.

OME was indicated by ENT examination and type B tympanogram. The OME patients underwent urine collection in a non-sterile urine pot made of polystyrene. The cotinine urine was measured using The COT one step cotinine test device® with lateral flow chromatographic immunosassay technique. If cotinine were found to be >200 ng/mL, one line (control) would appear, and then it was considered positive. If the cotinine were found to be <200 ng/mL, two lines (control and test) would appear, which was then considered negative. If no lines appear in the control, it is considered invalid. All the results are documented and verified blindly by two staff of the Otology Division of the Faculty of Medicine, Universitas Indonesia. This study has been approved by the Ethics Committee of the Faculty of Medicine, Universitas Indonesia (No:209/PT02.FK/ETIK). This study also complied with the recommendations of the Declarations of Helsinki and Tokyo. The participants and their parents provided verbal and written informed consent before study participation.

3. RESULTS AND DISCUSSION

All study subjects who met the inclusion and exclusion criteria filled out a questionnaire and were interviewed. Throughout the visit to 364 houses, 398 children were obtained as the study subjects, with 52% of subjects are boys and the median age of the subjects was 8.1 (0.1-14.4) years old. In this study, the age was divided into three age groups; most children are in the 5-9 age group (38.7%). Of these 398 subjects, 50.3% were elementary students as presented in Table 1. Based on parents’ education level and background work, only 14.4% of their fathers and 16.3% of their mothers have higher education as shown in Table 2. Most fathers are working (86.4%), while mothers are non-working or stay-at-home moms (73.4%).

All subjects underwent physical examination, especially on the ear, nose, and throat. They also underwent tympanometry to diagnose OME. From 796 ears examined, most tympanic membranes were intact. However, 10 (1.3%) had perforation. According to the tympanometry examination on 786 ears, type A tympanogram was mostly found (86.4%), followed by type C (4.5%). Type B tympanogram was found to be 0.9% from all ears examined. 63 ears can not be examined using tympanometry due to active infection and membrane perforation on their particular ear. Out of the 398 subjects, five were found to have OME with a prevalence of 1.3%. Restuti et al. [1] found that the prevalence of OME in preschool and elementary children in East Jakarta is 2.28% (46 out of 2016 children). Anggraeni et al. [11] reported that 26 out of 7,005 children in Indonesia were diagnosed with OME.

The comparison of boys and girls with OME in our study was 1.5:1. Incidence for OME, in some studies [1], [12], [13] occurs more in males than females. This difference is probably caused by the Eustachian tube angle, which is more horizontal in males than females [14]. Three out of five OME subjects in our study were in the age group of 5-9 years old and two out of five had bilateral OME. Previous studies [1], [10], [13], [15] also reported higher incidences of OME in younger children. The high prevalence of OME is due to the immaturity of Eustachian tubes in children, caused by three age-related factors:
Eustachian’s tube length, angle, and ability to close. The Eustachian tube dysfunction can not adequately protect the middle ear from nasopharyngeal pressure associated with the contamination of the middle ear by rhinopharyngeal germs [2].

Out of five subjects with OME, only three had complaints; one experienced hearing difficulty in both ears, one felt fullness in the unilateral ear, one experienced tinnitus in both ears, and none had communication problems. All the patients in the OME group have intact tympanic membranes in both ears, with six having a normal tympanic membrane, two having mild retraction, and two having bubbles on the tympanic membrane as presented in Figure 1. It is following a study Anwar et al. [16] that stated physical findings in OME are retracted, bulging, or abnormal coloration of the tympanic membrane, with bubble or air-fluid level findings on the otoscope. In the non-OME group, most of the tympanic membranes were normal (90.7%), followed by mild retraction (7.1%), non-intact (1.3%), bulging (0.5%), severe retraction (0.3%), and bubble (0.1%).

### Table 1. Sociodemographic of the subjects (N=398)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Groups</th>
<th>OME (%)</th>
<th>Non-OME (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Girls</td>
<td>2 (1.0)</td>
<td>189 (99.0)</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>Boys</td>
<td>3 (1.4)</td>
<td>204 (98.6)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0-4 years old</td>
<td>1 (0.9)</td>
<td>108 (99.1)</td>
<td>0.611</td>
</tr>
<tr>
<td></td>
<td>5-9 years old</td>
<td>3 (1.9)</td>
<td>151 (98.1)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10-14 years old</td>
<td>1 (0.7)</td>
<td>134 (99.3)</td>
<td></td>
</tr>
<tr>
<td>Education level</td>
<td>Not-schooled</td>
<td>2 (1.3)</td>
<td>147 (98.7)</td>
<td>0.695</td>
</tr>
<tr>
<td></td>
<td>Elementary school</td>
<td>3 (1.5)</td>
<td>197 (98.5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Junior high school</td>
<td>0 (0)</td>
<td>49 (100.0)</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Parents of study population education level and working status (N=398)

<table>
<thead>
<tr>
<th>Parents' education level &amp; Type of work</th>
<th>OME (%)</th>
<th>Non-OME (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education level</td>
<td>Lower education</td>
<td>4 (1.2)</td>
<td>337 (98.8)</td>
</tr>
<tr>
<td></td>
<td>Higher education</td>
<td>1 (1.8)</td>
<td>56 (98.2)</td>
</tr>
<tr>
<td>Working status</td>
<td>Not working</td>
<td>1 (1.9)</td>
<td>53 (98.1)</td>
</tr>
<tr>
<td></td>
<td>Working</td>
<td>4 (1.2)</td>
<td>340 (98.8)</td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education level</td>
<td>Lower education</td>
<td>3 (0.9)</td>
<td>330 (99.1)</td>
</tr>
<tr>
<td></td>
<td>Higher education</td>
<td>2 (3.1)</td>
<td>63 (96.9)</td>
</tr>
<tr>
<td>Working status</td>
<td>Not working</td>
<td>3 (1.0)</td>
<td>289 (99.0)</td>
</tr>
<tr>
<td></td>
<td>Working</td>
<td>2 (1.9)</td>
<td>104 (98.1)</td>
</tr>
</tbody>
</table>

![Figure 1. Otitis media with effusion frequency distribution regarding ear examination findings](image)

*Passive smoker exposure as a risk factor of otitis media with effusion in children (Riza Rizaldi)*
From all 398 samples, there are 65.3% passive smokers. According to a previous study Putri et al. [17], over 97 million Indonesians, including 43 million children, are exposed to cigarette smoke daily. In addition, the proportion of Indonesian children under 15 years exposed to secondhand smoke at home is 79% [7]. Based on the frequency of OME following smoking history in the house, four out of five subjects with OME were not passive smokers. Only one of the five OME subjects in this study was a passive smoker, with one active smoker in the house, the father. Therefore, the proportion of OME in subjects with passive smokers is 0.4% and the proportion of OME in non-passive smoker subjects is 2.9% as shown in Table 3. Comparison of the proportion with other variables did not show significant results between OME and non-OME for the variables such as smoking family, gender, age group, education level, parents’ education level, and working status.

Cotinine is the best biomarker of secondhand smoke exposure because it is the main metabolite of nicotine. Cotinine is a more stable metabolite with a half-life of approximately 16 hours. It can be detected in bodily fluids, such as saliva, blood, and urine. The urinary cotinine level examination is preferred because it is less invasive and more convenient than the serum cotinine examination. Urinary cotinine levels are associated with secondhand smoke exposure [18], [19]. A previous study Kim et al. [20] reported that cotinine is detected even in unexposed children to secondhand smoke. It can be caused by parental unawareness of possible cigarette smoke exposure to their children and inadequate reporting. The urinary cotinine concentrations are correlated with the number of cigarette packs smoked by their father, the number of smokers in residence, their father’s educational level, smoking rules at the residence, and the number of rooms in their house. However, a study by Susanto et al. [18] concluded that there is no significant correlation between urinary cotinine concentrations and type of cigarette, duration exposure, and the number of smokers in the house. In our study, no OME group subjects had a cotinine urine level of >200 ng/mL, stating that no children aged 0-14 were exposed to cigarette smoke above the normal threshold. In the OME group, only one sample was a passive smoker but showed a cotinine urine level of <200 ng/mL.

Several questionnaire-based studies have been performed to identify cigarette smoke exposure in children as a risk factor for OME. A study by Kiris et al. [9] reported secondhand smoke as one of OME’s most significant risk factors. On the other hand, a study by Humaid et al. [10] found no significant correlation between passive smoking exposure and the development of OME. Another study Gultekin et al. [21] also found that mother’s smoking habits are one of the contributing factors for OME. However, this study [21] found that father’s smoking habits do not significantly correlate with the development of OME. It can be caused by the close relationship between the child and the mother. Previous cotinine-based studies [22], [23] to identify cigarette smoke exposure as a risk factor for OME in children have been performed with different results. A study by Ilicali et al. [22] found a significant difference in urinary cotinine in OME and non-OME groups. Another study by Savitri et al. [23] found no significant correlation between urinary cotinine and OME. This study also found that urinary cotinine in the OME group is lower than in the non-OME group (2.55 and 2.63 ng/mL).

The correlation between cigarette smoke exposure and OME may be caused by the decrease in mucociliary action, local immune function depression, and an increase in the adherence of bacteria to the epithelium after cigarette smoke exposure [5]. In addition, high concentrations of environmental tobacco exposure can also increase the risk of Eustachian tube dysfunction in some pathways, such as direct contact between Eustachian tube mucosa and the smoke because of its proximity to the upper airway, secondary changes due to Eustachian tube obstruction caused by inflammation of nasopharyngeal inlet, and mediators produced that stimulate middle ear disorder [24]–[26]. There are several reasons why the urinary cotinine levels in subjects with OME were found to be negative (<200 ng/mL). First, the subjects were not exposed to the cigarette. This is due to the questionnaire filling that only one of five OME subjects had a history of being passive smokers. Second, cotinine may not provide an accurate estimation when there is a break in smoking or exposure because it reflects smoking exposure over numerous days [27]. Third, the amount of nicotine converted to cotinine varies between 55% and 92% [27], [28]. It is influenced by age, gender, body mass index (BMI) and liver enzymes CYP2A6 [29]. Fourth, there are individual differences in cotinine metabolism and excretion rates [27].

### Table 3. Otitis media with effusion frequency distribution regarding the history of smoking in the house

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Groups</th>
<th>OME n (%)</th>
<th>Non-OME n (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking family</td>
<td>Yes</td>
<td>1 (0.4)</td>
<td>259 (99.6)</td>
<td>0.051</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>4 (2.9)</td>
<td>134 (97.1)</td>
<td></td>
</tr>
</tbody>
</table>

Although the use of cotinine concentrations to assess cigarette smoke exposure has been largely applied, there are no cut-off points that have been generally accepted. The cut-off points of urinary cotinine may differ by ethnic and race [30]. A study by Susanto et al. [18] found urinary cotinine concentrations of ≥17.95 ng/mL (81% sensitivity and 81% specificity) indicate cigarette exposure in children. A study by Kim et al. [20] found urinary cotinine cut-off concentrations of 9.42 ng/mL (53% sensitivity and 57.1% specificity) to differentiate between passive cigarette smoke exposure and non-exposure. Besides, our study could not determine the specific value of the urinary cotinine levels. Thus, it becomes our limitations.

4. CONCLUSION

The prevalence of otitis media with effusion (OME) in children aged 0-14 in East Jakarta was 1.3%. In this study, all subjects had cotinine urine levels of <200 ng/mL. Hence, no proportion of OME in relation to cigarette smoke was found objectively. However, from the interview, one out of five OME group samples were passive smokers. In this study, a characteristic of smoking history in the house on passive smoker OME children were found, with one person maximum of an active smoker, the father. This study might improve the prevention strategy of OME in children. However, further studies are needed to evaluate the specific value of urinary cotinine levels in OME children exposed to secondhand smoke.

REFERENCES

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