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Does passive smoking induce sensorineural hearing loss in children?

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ABSTRACT

Objectives: Smoking plays major role in development of vascular and respiratory serious diseases. It has been reported that negative smoker children are prone for conductive hearing impairment due to repeated attacks of Eustachian tube dysfunction and middle ear effusion. This study aims to identify negative smoking as potential risk factor for development of sensorineural hearing loss.
Study: This study was done between January 2010 and November 2012. 411 children aged 5-11 years (8.2 ± 1.5) participated in this study; they were children attending the Ear, Nose, and Throat clinic of a tertiary care hospital and their siblings. The inclusion criteria were: (i) normal speech and language, (ii) absence of any disease or condition that may cause sensorineural hearing loss, and (iii) normal middle ear function on the day of hearing assessment. They were divided into three groups according to the exposure to second-hand smoke at home; group of "no exposure" whereas no smoker in the family (131 children), group of 'mild exposure" whereas the father was the only smoking parent and smoking was prohibited at home (155 children), and group of "heavy exposure", whereas the mother was smoking, or the father was freely smoking at home and in the presence of his children (125 children). Audiological evaluation in the form of pure tone and speech audiometry and immitancemetry was done for the study group.
Results: Audiological evaluation revealed that the prevalence of hearing loss was 3.8%, 4.5% and 12% in the "no exposure", "mild exposure", and "heavy exposure" groups, respectively. Significant difference was only detected between the high exposure group and the other two groups. All children had minimal sensorineural hearing loss, i.e. threshold of frequencies showing hearing loss was 20 or 25 dB HL. The risk ratios (95% confidence interval) for hearing loss in the study subgroups were 1.18 (0.38, 3.64) for mild exposure group ($p > 0.05$), 3.14 (1.18, 8.3) for heavy exposure group ($p < 0.05$).
Conclusions: Passive smoking in childhood correlates with sensorineural hearing loss, and it is an important risk factor for development of minimal hearing loss. Strict prevention of children exposure to second-hand smoke should be encouraged by every mean.

1. Introduction and rationale

Cigarette smoking has become a common tendency worldwide; in 2003, tobacco was consumed by approximately 1.3 billion of the world's population. It is predicted that 1.5–1.9 billion people will be smokers in 2025 [1]. Smoking plays major role in development of serious vascular and respiratory diseases. Many studies confirmed the relation between hearing loss and smoking [2]. Moreover, it was reported that smoking accelerates age related [3] and noise induced hearing loss [4]. The hazards of smoking are not limited to the smokers; it extends to the surrounding people especially for children and infants. Passive smoking (also called second-hand smoke exposure or environmental tobacco smoke exposure) became a profound public health problem, with more than half of children in the United States exposed [5]. Passive smoking has been associated with lung cancer, ischemic heart disease, sudden infant death syndrome, asthma in children and adults, severe headache, and hearing loss [6].

There is mounting evidence that passive smoking is a risk factor of hearing loss; in a cohort of infants, exposure to cigarette smoke was associated with a 4.9 times increase in the prevalence of hearing deficits [7]. The children of smoking parents are more prone to develop recurrent [8] or persistent [9] otitis media with

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effusion. It was noticed that hearing loss was more prevalent when the mother was the smoking parent, because the mother is more likely to spend time with the child and, if she smokes, the child is more likely to be exposed to the tobacco smoke [10]. Sensorineural hearing loss in smokers and passive smokers was reported in many studies in adults and teenagers [11,12]. This study aims to test the prevalence of sensorineural hearing loss in children experiencing passive smoking.

2. Materials and methods

2.1. Subjects

This study was conducted in a tertiary referral center, between January 2010 and November 2012 in Hadiclinic Hospital, Kuwait. The study group included 411 children (188 males, 223 females), with age range 5-11 years (8.2 ± 1.5). They were children of 290 families attending the ENT clinic; only 89 of these families (131 children) had non smoking parents. Informed consent was obtained from the parents of the children participating in the study, and the study was approved by the Ethics Committee at Hadiclinic Hospital.

2.2. Methods

The entire study group was subjected to detailed history of any condition causing hearing loss, e.g. ototoxic drug intake, family history of hearing loss, head trauma, etc.; otoscopy; immittancemetry; tympanometry and acoustic reflex testing. Pure tone (air and bone conduction) and speech audiometry, speech reception threshold (SRT) and word discrimination scores (WDS) were done using Madsen, Orbiter 922 ver.2 audiometer. Hearing testing was undertaken inside a sound treated booth, with the test environment meeting the specifications detailed in Maximum Permissible Ambient Noise Levels for Audiometric Test Rooms (ANSI S3.1-1999) [13]. Smoking history was ascertained by self-report obtained from the parents.

The inclusion criteria for this study were: (i) normal speech and language development, (ii) absence of any disease or condition that may cause sensorineural hearing loss, and (iii) normal otoscopic examination and middle ear function on the day of hearing assessment.

All the parents of the families participating in the current study replied to a questionnaire concerned with the smoking habits in the family (if any). The questionnaire questions were concerned with identifying the following: (1) awareness of the parents to the hazards of smoking and passive smoking especially on children. (2) Ascertaining parental tobacco consumption, smoking by household members other than parents, e.g. by visitors to the house or child care providers. (3) Does the parents permit smoking in the home in the presence or absence of the children? According to the data obtained by the smoking questionnaire the children in the study group were divided into three groups according to the exposure to second-hand smoke at home: (1) *Group of "no exposure"* whereas no smoker in the family (131 children, 89 families), (2) *Group of 'mild exposure"* whereas the father was the only smoking parent and smoking was prohibited at home, and in the presence of the children (155 children, 120 families), and (3) *Group of "heavy exposure*" whereas the mother was smoking, or the father was freely smoking at home or in the presence of his children (125 children, 81 families).

Hearing loss was identified when pure-tone threshold in any of the tested frequencies was greater than 15 dB hearing level (HL) in the worse ear. The degree of hearing loss was determined according to the pure tone average of 500, 1000 and 2000 Hz. Subjects with hearing loss and pure tone average $\leq$25 were considered minimal hearing loss. In other words in order to determine the presence or absence of hearing loss the hearing threshold of all the frequencies 250 through 8000 Hz were considered, if all the frequencies showed threshold $\leq$15 dB HL then normal hearing was reported. If any frequency had hearing threshold greater than 15 then we would move to the next step, calculating the degree of the hearing loss. The pure tone average only for frequencies 500, 1000 and 2000 was calculated, if it was $\leq$25 dB HL this was considered minimal hearing loss. Other degrees of hearing loss such as mild, moderate or severe would have been considered if the average was 26 dB HL or more [14]. Low frequency hearing loss was considered if the affected frequencies were $\leq$2 kHz. High frequency hearing loss was considered if the affected frequencies were $\leq$3 kHz [11].

3. Results

3.1. Demographic distribution

Table 1 shows the demographic distribution of the study group. One way ANOVA and Chi square test were done, they showed no statistical significant difference as regards age or gender distribution ($p > 0.05$).

3.2. Audiological assessment

According to the inclusion criteria of the present study, the entire study had normal middle ear functions. This was confirmed by the normal otoscopic testing, type (A) tympanogram and intact acoustic reflexes in the entire study group. No air bone gap was detected in any subjects in this study.

Table 2 shows the mean and SD of the air conduction pure tone thresholds, SRT and WDS of both the right and left ears in the study group (822 ears). One way ANOVA test revealed no statistical significant difference as regard hearing threshold in all of the frequencies ($p > 0.05$).

Table 3 shows demographic and audiological features of hearing loss in the different study groups. All of them had minimal hearing loss. Statistical analysis showed insignificant differences in age and gender distribution between children with hearing loss and those with normal hearing, only significant difference was reported in the prevalence of hearing loss in the high exposure group compared with the other 2 groups.

3.3. Risk ratios

The risk ratios (95% confidence interval) for hearing loss in the study subgroups were 1.18 (0.38, 3.64) for mild exposure group ($p > 0.05$), 3.14 (1.18, 8.3) for heavy exposure group ($p < 0.05$).

4. Discussion

Many studies reported a correlation between passive smoking and hearing loss in infants, children, adolescents and adults [7,10,12]. Several possible mechanisms may account for the relationship between smoking and sensorineural hearing loss;

Table 1
Demographic distribution of the study group.

	No exposure	Mild exposure	Heavy exposure
No.	(131/411)	(155/411)	(125/411)
	31.9%	37.7%	30.4%
Age (years), mean ± SD	8.2 ± 1.5	8.2 ± 1.4	8.1 ± 1.5
Females	(74/131)	(83/155)	(66/125)
	56.5%	53.5%	52.8%

No., number and percent of children in each subgroup.