Effect of Lipid Profile Parameters on Noise Induced Hearing Loss

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Abstract

Background: Noise induced hearing loss (NIHL) is an irreversible occupational disease among industrial workers. Recent studies have reported that changes in some metabolic factors such as the serum level of sugar and lipids might have a role in suffering from NIHL among workers exposed to noise. We designed this study to assess the association between lipid profile changes and NIHL occurrence among noise-exposed workers.

Methods: This case-control study has been conducted according to noise-exposed workers registry data in one of the Iranian automobile factories between 2007 and 2017. We classified study workers into the NIHL and control groups. We assessed the impact of lipid profile parameters across the study groups using the independent samples t-test, chi-square, and regression.

Results: The mean serum level of cholesterol was significantly higher in the NIHL group than in workers of the control group (215.27 ± 60.30 vs 204.49 ± 63.69 mg/dL; \( P = 0.041 \)). Moreover, the serum level of HDL was significantly lower in workers in the NIHL group compared with the control group (35.21 ± 6.87 vs 37.43 ± 7.28 mg/dL; \( P < 0.001 \)). Although other lipid profile parameters (LDL, TG, LDL/HDL ratio) were higher among workers of the NIHL group, their differences were not significant.

Conclusion: A cholesterol level lower than 200 mg/dL is known as a protective factor and an HDL level lower than 40 mg/dL is an NIHL risk factor. More attention should be paid to controlling serum levels of cholesterol and HDL.

Keywords: Cholesterol, HDL, LDL, NIHL, Triglyceride, Lipid Profile, Noise Exposure

Introduction

Noise-induced hearing loss (NIHL) is known as a gradually progressive hearing loss that occurs when one is exposed to a high level of noise for a long time at the workplace (1). It is estimated that nearly one-third of workers in European countries are exposed to high levels of noise (2). The National Institute for Occupational Safety and Health (NIOSH) has reported that in the USA, about 5.7 million workers in manufacturing industries are exposed to hazardous noise (3). NIHL is one of the most common forms of hearing loss in the United States with a prevalence of 25% in adults (4). NIHL is an irreversible occupational disease and therefore prevention has a critical role in controlling NIHL among industrial workers. Recent studies have reported that changes in some metabolic factors such as serum levels of sugar and lipids might have a role in suffering from NIHL among workers exposed to noise (5, 6). Unfortunately,

↑What is “already known” in this topic:
The chance of developing noise-induced hearing loss (NIHL) is influenced by a few factors such as genes, diet, and healthy listening habits. Using appropriate hearing protection devices in workers exposed to high noise levels is the most important action to prevent NIHL, however, controlling other factors such as dyslipidemia may also affect the frequency of NIHL.

→What this article adds:
The serum level of cholesterol (>200 mg/dL) in workers is independently associated with noise induced hearing loss. The serum level of HDL (<40 mg/dL) in workers is independently associated with noise induced hearing loss.
most studies in this field have not provided sufficient evidence to create a causative relationship between lipid profile changes and NIHL occurrence. Therefore, more accurate methodological designs, case-control, or cohort studies are required for this purpose.

Local and national occupational health registers have been developed in recent years for recording occupational health variables to create more scientific practical occupational disease protocols and some studies were conducted using these databases (7-9). Audiometry and serum lipid levels have been recorded in Iranian medical occupational health examinations and we can design case-control studies using these data. Accordingly, we designed the present study for the assessment of the association between lipid profile changes and NIHL occurrence among noise-exposed workers.

Methods

The present case control study has been conducted according to recorded data from the annual occupational examination data registry in one of the Iranian automobile factories. We included all data related to male workers in the study workplace between 2007 and 2017. We included male workers with chronic noise exposure at least 85 dB for more than 1 year. Among the workers, those who were smokers, had any pathological ear disorders and acoustic trauma, diabetes, hypertension, a history of using ototoxic drugs in the last 3 months, and secondary jobs with ototoxic chemicals and noise exposure were excluded. Study protocols have been approved by the committee of ethics in Iran University of Medical Sciences and Health Services.

The level of noise exposure was a potential confounder, thus, we calculated the time weighted mean of noise exposure of the study population and matched the exposure level among the study groups. The control group was matched with respect to age, work experience, and the level of noise exposure with the case group. We assessed the audiogram findings of study workers during the study period and defined NIHL cases in our study population since the presence of notch in any of 3 KHz, 4 KHz, and 6 KHz with recovery at 8 KHz in their audiogram results. We classified study workers into the NIHL and control groups and collected demographic characteristics, including age, education, marital status, past medical history, and drug history from the registry data of occupational medicine annual examination and history. We computed the mean serum level of lipid profile (cholesterol, triglyceride, HDL, and LDL) among the study population during the study period to assess the impact of lipid profile changes among the study groups. In the laboratory findings report, we considered serum levels of triglyceride and cholesterol higher than 150 mg/dL and 200 mg/dL and serum levels of LDL higher than 100 mg/dL and HDL lower than 40 mg/dL as lipid profile impaired.

Statistical Analysis

The study data were analyzed using the statistical software SPSS Version. 22.0. Mean and standard deviation were used to report quantitative data, and frequency and percentage were used for qualitative data. For the comparison of quantitative and qualitative variables between the NIHL group and the control group, we used independent t-tests and chi-square tests. We used the odds ratio to assess the effects of lipid profile parameters across study groups.

All statistical tests were considered significant, when $P < 0.05$. Logistic regression model analysis was performed to control the confounding variables. We included NIHL occurrence as a dependent dichotomous variable and age, work experience, and BMI as quantitative variables, and serum levels of TG, cholesterol, LDL, HDL, and LDL/HDL ratio as categorical variables in the regression model. All P values in the final regression model output less than 0.05 were assumed as significant results and related variables were recognized as NIHL independent predictors.

Results

According to study inclusion criteria, data related to 606 workers were used in the statistical analysis. The mean of age and work experience between study workers were 35.26 ± 6.8 years (range: 21-50) and 5.14 ± 3.04 years (range: 1-17), respectively. Among study workers, 564 (93.1%) were married and 108 (17.8%) had secondary jobs. The educational level in half of workers was high school diploma (47.5%). The mean body mass index (BMI) among workers was 23.18 ± 4.06 (17.35-33.9) kg/m². Accordingly, the BMI of 322 (53.14%) workers was in a normal range (BMI < 25). According to findings of audiometry, 218 (35.97%) workers had NIHL and were allocated in the NIHL group and 388 (64.03%) did not have NIHL and belonged to the control group. The mean age among workers in the NIHL and control group did not have a significant difference (37.65 ± 7.65 vs 38.47 ± 7.54; P = 0.784). The mean work experience among workers in the NIHL and control groups did not have a significant difference (6.46 ± 2.89 vs 6.86 ± 3.54; P = 0.122).

Although the mean serum level of triglyceride among the NIHL group was higher than the control group, the noted difference was not significant (165.93 ± 64.89 vs 160.80 ± 71.17 mg/dL; P = 0.379). Average serum level of cholesterol among workers in NIHL group was significantly higher than control group (215.27 ± 60.30 vs 204.49 ± 63.63 mg/dL; P = 0.041). The mean serum level of LDL among the NIHL and control groups did not have a significant difference (108.72 ± 35.10 vs 108.63 ± 33.38 mg/dL; P = 0.977). The mean serum level of HDL among workers in NIHL group was significantly lower than those in the control group (35.21 ± 6.87 vs 37.43 ± 7.28 mg/dL; P < 0.001).

Among the study workers, the prevalence of hypocholesteremia among workers of the NIHL group was significantly higher than workers of the control group (300, 40.1% vs 96, 53.3%; P = 0.001). Workers with hypocholesteremia had a higher chance of NIHL compared with workers of the control group (1.71, C195%: 1.23-2.37). The prevalence of hypertriglyceridemia had no significant difference between workers of the 2 groups (358, 47.8% vs 98, 54.4%; P = 0.113). The prevalence of LDL was similar among workers of the NIHL and control groups (325, 43.4% vs 87, 48.3%; P = 0.228). The prevalence of high HDL (>40mg/dL) was
Table 1: Comparing lipid profile impaired among workers of NIHL and control groups

<table>
<thead>
<tr>
<th>Study variable</th>
<th>Case (NIHL+)</th>
<th>Control (NIHL-)</th>
<th>P-value</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Cholesterol level</td>
<td>449 (59.9%)</td>
<td>84 (46.7%)</td>
<td>0.001</td>
<td>1.71 (1.23-2.37)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>300 (40.1%)</td>
<td>96 (53.3%)</td>
<td>0.113</td>
<td>1.31 (0.94-1.81)</td>
</tr>
<tr>
<td>Serum triglyceride level</td>
<td>391 (52.2%)</td>
<td>98 (54.4%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>358 (47.8%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum LDL level</td>
<td>424 (56.6%)</td>
<td>93 (51.7%)</td>
<td>0.232</td>
<td>1.21 (0.88-1.69)</td>
</tr>
<tr>
<td>Normal</td>
<td>325 (43.4%)</td>
<td>87 (48.3%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>185 (24.7%)</td>
<td>23 (12.8%)</td>
<td>0.001</td>
<td>0.44 (0.28-0.71)</td>
</tr>
<tr>
<td>Serum HDL level</td>
<td>564 (75.3%)</td>
<td>157 (87.2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>280 (37.4%)</td>
<td>51 (28.3%)</td>
<td>0.018</td>
<td>1.51 (1.06-2.16)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>469 (62.6%)</td>
<td>129 (71.7%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Results of logistic regression analysis in our participants

<table>
<thead>
<tr>
<th>Study variable</th>
<th>EXP (β)</th>
<th>Standard Error</th>
<th>P-value</th>
<th>95.0% C.I. for EXP (B) lower</th>
<th>95.0% C.I. for EXP (B) upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>0.998</td>
<td>0.02</td>
<td>0.942</td>
<td>0.96</td>
<td>1.04</td>
</tr>
<tr>
<td>TG&lt;150 mg/dl</td>
<td>1.12</td>
<td>0.22</td>
<td>0.608</td>
<td>0.72</td>
<td>1.71</td>
</tr>
<tr>
<td>Cholesterol&lt;200 mg/dl</td>
<td>0.53</td>
<td>0.24</td>
<td>0.005</td>
<td>0.32</td>
<td>0.82</td>
</tr>
<tr>
<td>LDL&lt;100 mg/dl</td>
<td>1.25</td>
<td>0.21</td>
<td>0.256</td>
<td>0.83</td>
<td>2.08</td>
</tr>
<tr>
<td>HDL&lt;40 mg/dl</td>
<td>2.11</td>
<td>0.28</td>
<td>0.006</td>
<td>1.24</td>
<td>3.67</td>
</tr>
<tr>
<td>LDL/HDL ratio&lt;2.5</td>
<td>1.05</td>
<td>0.23</td>
<td>0.842</td>
<td>0.64</td>
<td>1.73</td>
</tr>
<tr>
<td>Constant</td>
<td>0.07</td>
<td>0.68</td>
<td>0.000</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Discussion

Findings of the present study on the male workers of an Iranian automobile factory showed that among workers with NIHL, the mean serum level of cholesterol was significantly higher than workers in the control group (without NIHL). Moreover, the serum level of HDL was significantly lower in the NIHL group compared with those in the control group. Although other lipid profile parameters (LDL, TG, LDL/HDL ratio) were higher among workers of the NIHL group, their differences were not statistically significant.

In the general population, there are few studies in the literature that found a relationship between hearing loss and high levels of blood lipids. For instance, Axelsson and Lindgren in their study, examined hearing loss among 78 people with cholesterol levels >270 mg/dL and 75 people with serum cholesterol levels < 216 mg/dL and reported that higher levels of serum cholesterol was associated with hearing loss (10). In another study, Fuortes et al reported that hearing loss among 665 university worker was associated with high serum cholesterol levels (11). Contrary to the above findings, Chang et al reported a weak association between high levels of serum triglycerides and hearing loss, nonetheless, in their study there was no correlation between high cholesterol levels and hearing loss among 4071 participants who underwent health checkups (12). We can therefore conclude that the impact of lipid profile on hearing loss among the general population is a controversial topic and needs to be further studied to confirm or reject this relationship.

There is a hypothesis on the pathophysiology of high lipid profile parameters in hearing loss. Dyslipidemia can affect the microcirculation in the organ of Corti, which can lead to hearing loss. Since the organ of Corti has a high blood supply, hearing loss is associated with reduced local blood flow and the formation of free radicals. It seems that we had a multifactorial phenomenon as hearing loss and some of causing factors might overlap their impacts and decrease their effects in their interactions (13).

In occupational settings, in a study by Doosti et al, workers with hypertriglyceridemia who were exposed to noise, had more chance having NIHL compared with exposed workers with normal triglyceride levels (14). Demir et al conducted their study on 456 noise-exposed (≥85 Db) and control unexposed workers to assess the relationship between noise exposure and serum lipid levels. They found that total cholesterol, LDL-cholesterol, triglycerides, and cholesterol/HDL ratios increased with workplace noise exposure, but after adjusting for the effect, the relationship was not significant (15). There are some differences between above studies and our findings. We think that some epidemiolog-
Lipid Profile & Hearing Loss

The authors declare that they have no competing interests.

References