ORIGINAL ARTICLE

Assessment of the Relationship between NIHL and Blood Biochemical Tests

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ABSTRACT

Noise-induced hearing loss (NIHL) is the most common complication of long-term noise exposure, though there are some other complications due to exposure to noise. Metabolic complications of the noise exposure are being evaluated in recent studies. We aimed to assess the relationship between NIHL and lipid profile, and fasting blood sugar (FBS). In this cross-sectional study, 462 workers with exposure to noise were randomly selected. Considering the presence of NIHL they were divided into two groups. FBS, cholesterol, LDL, HDL, TG, ALT, AST, ALP and Cr levels were measured in these workers and all variables were compared between two groups. We found a significant relationship between FBS, TG, cholesterol and LDL levels in two groups (p<0.001 for all variables). There was not a significant difference in Cr and liver enzymes levels between two groups. Noise exposure could be a cardiovascular risk factor, which can cause considerable metabolic disorders among those who suffer from NIHL.

Keywords: Noise, NIHL, Lipid profile, FBS, Hepatic enzymes

INTRODUCTION

Noise is one of the most pervasive and important physical hazards at workplaces [1]. One of the most important effects of noise on health is noise-induced hearing loss (NIHL), a common occupational disease of adult population [1, 2]. Patients exposed to noise for a long duration have hearing loss that is especially maximal at 3-6 kHz and this hearing loss slightly improves at high frequencies, but it is not affected at low frequencies [1]. NIHL is always sensorineural, and typically bilateral. Its first sign is a notching of the audiogram at 3000, 4000, or 6000 Hz, with recovery at 8000 Hz [3].

The American National Institute of Occupational Safety and Health (NIOSH) views hearing loss as one of the top priority areas for research in the 21st century [4]. It is now well recognized that exposure to intense noise not only causes hearing loss in man but also produces other extra-auditory effects [1, 5-12]. A sound level above 80 dB has been considered to affect functional ability, biochemicals, enzymes, immunological system and histology in the animals and human [6].

Many studies have shown a relationship between NIHL and the cardiovascular, endocrine, metabolic, gastrointestinal and neurological systems [7-11]. The cardiovascular system is the most involved, whereas few specific studies have been performed concerning the relationship between noise and myocardial disease. There is some evidence that noise is a significant factor in the induction of arterial hypertension [8, 12] and coronary disease [13]. Some studies have shown a relationship between NIHL and lipid profile [7, 14-17].

On the contrary, some studies have not shown any relationship between NIHL and cardiovascular risk.
factors [17–20]. Vaughan showed the effect of noise on hearing loss among diabetics [19]. Hypertriglyceridemia was related to NIHL (OR = 1.281; 95% CI, 1.088-1.507) but not as for hypercholesterolemia [15].

Souto Souzo found a positive association between occupational noise exposure and hypertension [12]. A higher level of LDL, triglyceride, uric acid and blood pressure among those exposed to communal or industrial noise comparing those without exposure is shown [7]. The studies about the effect of noise on serum creatinine and liver enzymes could not show a significant relationship between these variables and noise [20, 21].

The sensitivity of humans to noise is different, so in a given level of noise some people may suffer from NIHL and some of them not. Thus the sensitivity to other possible causes of noise may be different as well. We aimed to assess whether there was any difference in fasting blood sugar, lipid profile, creatinine and liver enzyme levels between the noise-exposed workers with and without NIHL.

**MATERIALS AND METHODS**

In this cross-sectional study, from 574 workers with exposure to noise over 85 dBA (8 hour time-weighted average) from two tile and ceramic factories (Mojtame Kashi, and Ehsan factories, Meybod, Yazd) were randomly selected. Workers older than 50 years old or with any history of exposure to ototoxic agents and drugs, acoustic trauma, central nervous system (CNS) infection in childhood, conductive hearing loss and unilateral hearing loss were excluded from the study. At last, 462 individuals remained in the study. Then audiometry was performed for all subjects (using clinical audiometer: AC40, Interacoustic, Denmark, headphone: TDH39). Audiometry was performed by an expert audiologist (blinded to the study) in an acoustic chamber, meeting standards of ANSI 2004 [22].

The subjects were divided into two groups including with and without NIHL. We defined NIHL as a sensorineural, bilateral hearing loss which at least involves 3000, 4000, and 6000 Hz and the hearing threshold at least at one of these frequencies is higher than 20 dB [2]. According to this definition, 201 subjects entered the case group and 261 subjects were considered as the control group. Then 10 ml blood was sampled from each subject and fasting blood sugar (FBS), total cholesterol (Chol), triglyceride (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), creatinine, alanine aminotransferase (ALT), asparagine aminotransferase (AST), and alkaline phosphates (ALP) were measured. Then these measures were compared between two groups.

We considered abnormal FBS, TG, Chol, LDL, AST, ALT, ALP, and creatinine as blood level higher than 110 mg/dL, 190 mg/dL, 240 mg/dL, 160 mg/dL, 36 U/L, 33 U/L, 306 U/L, and 1.2 mg/dL, respectively [26].

Then using SPSS (ver. 17), we analyzed data by Chi square, and t test. Level of significance was set at 0.05. An informal consent was filled out for each subject.

**Table 1. Descriptive statistics of the subjects**

<table>
<thead>
<tr>
<th>Variable</th>
<th>N*</th>
<th>Mean</th>
<th>SD*</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>201</td>
<td>36.41</td>
<td>5.619</td>
<td>0.394</td>
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<tr>
<td>Control</td>
<td>261</td>
<td>35.86</td>
<td>8.274</td>
<td></td>
</tr>
<tr>
<td>Employment Duration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>201</td>
<td>12.44</td>
<td>5.758</td>
<td>0.384</td>
</tr>
<tr>
<td>Control</td>
<td>261</td>
<td>11.94</td>
<td>6.665</td>
<td></td>
</tr>
</tbody>
</table>


**Table 2. The comparison of blood biochemical tests between case and control groups**

<table>
<thead>
<tr>
<th>Blood biochemical tests</th>
<th>N</th>
<th>Mean</th>
<th>SD*</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS* (mg/dL)</td>
<td>Case</td>
<td>201</td>
<td>119.46</td>
<td>69.846</td>
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<tr>
<td></td>
<td>Control</td>
<td>261</td>
<td>101.11</td>
<td>24.801</td>
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<tr>
<td>Chol* (mg/dL)</td>
<td>Case</td>
<td>201</td>
<td>243.98</td>
<td>59.543</td>
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<tr>
<td></td>
<td>Control</td>
<td>261</td>
<td>198.15</td>
<td>39.598</td>
</tr>
<tr>
<td>LDL* (mg/dL)</td>
<td>Case</td>
<td>166</td>
<td>139.701</td>
<td>50.176</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>256</td>
<td>112.48</td>
<td>32.517</td>
</tr>
<tr>
<td>HDL* (mg/dL)</td>
<td>Case</td>
<td>200</td>
<td>51.12</td>
<td>12.456</td>
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<tr>
<td></td>
<td>Control</td>
<td>260</td>
<td>55.38</td>
<td>9.549</td>
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<tr>
<td>TG* (mg/dL)</td>
<td>Case</td>
<td>201</td>
<td>256.21</td>
<td>150.825</td>
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<td></td>
<td>Control</td>
<td>261</td>
<td>155.04</td>
<td>89.069</td>
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<tr>
<td>ALP* (U/L)</td>
<td>Case</td>
<td>196</td>
<td>198.76</td>
<td>63.198</td>
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<td></td>
<td>Control</td>
<td>249</td>
<td>204.73</td>
<td>65.205</td>
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<tr>
<td>ALT* (U/L)</td>
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<td>33.68</td>
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<td>Control</td>
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<tr>
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<td>Control</td>
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<td>Creatinine</td>
<td>Case</td>
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<td>0.987</td>
<td>0.2392</td>
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<tr>
<td></td>
<td>Control</td>
<td>250</td>
<td>0.957</td>
<td>0.2231</td>
</tr>
</tbody>
</table>

* SD: standard deviation, FBS: fasting blood sugar, Chol: total cholesterol, TG: triglyceride, LDL: low-density lipoprotein, HDL: high-density lipoprotein, ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALP: alkaline phosphates
**Results**

Totally 462 subjects exposed to noise level higher than 85 dB entered the study. From 462 subjects 201 individual suffered from NIHL (case group) and 261 individuals were free from NIHL (control group). Table 1 compares age and employment duration between both groups.

Univariate analysis showed that mean FBS, Chol, LDL and TG were significantly higher in case group (p <0.001 for all variables). Table 2 compares different blood biochemical tests between two groups. We compared the frequency of abnormal blood biochemical tests between two groups and Table 3 shows this comparison.

Mean age was 38.36 (± 6.34) and 35.60 (± 5.42) year in those with and without abnormal FBS and the difference was statistically significant (p<0.001). There was not any significant age difference for other biochemical tests.

**Discussion**

Despite various studies investigating the relationship between noise and health effects other than NIHL, including the increase in the frequency of cardiovascular risk factors, answer to this question is not clear yet.

Some studies have shown an association between noise or NIHL and some biochemical tests. These effects are mediated through neuro-endocrine systems, such as increased activity of the sympathetic nervous system and increased levels of hormones, such as cortisol [1, 5, 26, 27].

In this study we compared the level of some blood biochemical tests such as lipid profile and FBS in workers exposed to noise with and without NIHL.

NIHL risk in our study had significant relationship with harmful blood lipids (total cholesterol, LDL and TG) and fasting blood glucose, although multivariate analysis could not show this relationship for cholesterol and FBS.

The relationship between NIHL and blood sugar was also observed in Vaughan study. This study showed a significant relationship between diabetes and high-frequency hearing loss depending to patients age [21].

Our study showed a significant relationship between NIHL and TG, Chol, and LDL levels which was consistent with some other studies [7, 14-17], although our result was in contrast to the results of Gold [29] and Jalistaghi [20]. We could not find any statistically significant relationship between NIHL and liver enzymes, which was consistent with Sorhen study [24]. There was not any relationship between creatinine level and NIHL in our study which was consistent with Mocci [23].

Gold compared the means of certain cardiovascular and biochemical measures in subjects with NIHL with those in subjects exposed to similar occupational noise, but demonstrating normal hearing. Their results showed that no significant differences were found between two groups in terms of the distribution of subjects for the indices measured, which was completely inconsistent with our study [29].

These results can show the harmful effects of noise on harmful lipids and blood sugar. Although some other factors such as nutrition, exercise and lifestyle can affect these biochemical indices in the body.

As it was mentioned there is some controversy about the effect of noise on laboratory tests, and the relationship between NIHL and these tests. So we recommend performing long-term studies, to follow the workers for a time period from the beginning of exposure to loud noise for blood biochemical tests to get more accurate results.

Our study had some limitations. We could not recheck the results of the laboratory tests, because the workers were not available after the first test. All subjects were male so we could not assess the effect of gender.

**Conclusion**

Our study shows a significant association between noise-induced hearing loss and abnormal glucose, LDL and TG levels, which are all cardiovascular risk factors; although there are some confounding factors which may affect biochemical tests.

**Acknowledgment**

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**References**


