

Role of Hypercholesterolemia in Development of Tinnitus

Dr. Paras Singhal¹; Dr. Shatakshi Dubey^{2*}; Dr. Ashray Jain

¹Resident Doctor, Jain ENT Hospital, Jaipur

² Junior Resident, Department of ENT, Index Medical College Hospital and Research Centre, Indore

³Resident Doctor, Jain ENT Hospital, Jaipur

Corresponding Author: Dr Shatakshi Dubey*

Publication Date: 2026/07/04

Abstract:

➤ Background

One of the most common aural symptoms is the condition of tinnitus where a person hears a sound despite there being no external stimulus. New data seems to indicate that metabolic and vascular causes especially hypercholesterolemia might lead to cochlear dysfunction and the onset of tinnitus.

➤ Purpose

The study aims to assess the correlation between hypercholesterolemia and tinnitus and the metabolic and vascular risk factors associated with the circumstance in patients attending a tertiary care facility.

➤ Methods

The study was a prospective case control study with 240 participants to represent 120 patients with tinnitus and 120 age and gender-matched controls without tinnitus. All of the participants received a detailed clinical examination, audiological examination, and estimation of the fasting serum lipid profile. The lipid parameters that were measured were total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides. The statistical analysis was done using descriptive and inferential statistics through the application of logistic regression in finding out the independent predictors of tinnitus.

➤ Findings

Hypercholesterolemia was highly reported in tinnitus patients than in controls (52.5% vs 28.3, $p = 0.001$). High LDL, low HDL and increased triglyceride level were also greatly related to tinnitus. Hypercholesterolemia (OR = 2.79), high LDL level (OR = 2.21), low HDL (OR = 1.76), hypertension (OR = 1.68), and smoking (OR = 1.82) were found to be independent predictors using logistic regression analysis. Among tinnitus patients sensorineural hearing loss was more prevailing.

➤ Conclusion

The research indicates that hypercholesterolemia is strongly related to tinnitus, which justifies the role of the vascular and metabolic factors in the pathogenesis of tinnitus. Early identification and treatment of dyslipidemia through developing a routine lipid profile and routine evaluation can help in better clinical outcomes in patients with tinnitus.

Keywords: Tinnitus, Hypercholesterolemia, Lipid Profile, Sensorineural Hearing Loss, Cochlear Microcirculation.

How to Cite: Dr. Paras Singhal; Dr. Shatakshi Dubey; Dr. Ashray Jain (2026) Role of Hypercholesterolemia in Development of Tinnitus. *International Journal of Innovative Science and Research Technology*, 11(6), 2421-2427.
<https://doi.org/10.38124/ijisrt/26jun1710>

I. INTRODUCTION

Tinnitus is the term used to define the sound heard in the ear with no outside sound and often referred to as the sound of ringing, buzzing, hissing, or whistling in the ear [1-3]. It is one of the commonest otological complaints that is being presented in clinical practice and is seen in almost 15-20% of the general population, and it is more prevalent among the elderly age group [4]. Even though tinnitus as an entity is not a disease, it can be seen to be indicative of underlying hearing and systemic pathology, such as sensorineural hearing loss, acoustic trauma, exposure to ototoxic drugs, and vascular abnormalities of the inner ear [5]. The condition may seriously affect the quality of life by disrupting sleep and concentration levels as well as emotional stability. [6,7].

Some of the most typical conditions associated with age are age-related hearing loss or presbycusis, which normally occurs after the sixth decade of life [8]. Another etiological factor acknowledged to be causing cochlear damage and development of tinnitus is prolonged exposure to high-intensity noise due to occupational or recreational exposures [9-11]. Moreover, the reversible factors including the impaction of cerum can also disrupt the transmission of sound and cause irritation of the tympanum membrane, therefore, triggering the symptoms of tinnitus [12]. Possible pathogenesis of tinnitus has also been associated with various systemic conditions like Meniere disease, head and neck trauma, hypertension, and use of medications such as antibiotics, diuretics and salicylates amongst others [13,14].

Hypercholesterolemia has been one of the systemic factors that have been put forward as a possible cause of audio dysfunction. It is thought that high serum cholesterol and triglyceride may affect the cochlear microcirculation, which leads to elevation of blood viscosity, development of microthrombus, and changed vasomotor control in the inner ear [15,16]. They can also cause changes in the vascular patterns that interfere with oxygen and nutrient supply to cochlear structures causing metabolic stress and dysfunction of sensory hair cells, which can consequently cause perception of phantom auditory sensations.

Hypercholesterolemia is a risk factor of cardiovascular disease, and dietary therapies, including low-fat and low-cholesterol diets, have also been linked to better lipid phenotype and potential reduction in tinnitus symptoms. [17]. Nonetheless, even with the increased attention devoted to the vascular and metabolic factors of the aural disorder, the exact connection between hypercholesterolemia and tinnitus is not properly delineated yet.

Thus, it is clinically important to study how hypercholesterolemia contributes to the development of tinnitus. A better idea of this relationship would possibly allow identifying people at risk early and implement the prevention and treatment strategies to enhance auditory performance and overall vascular conditions.

II. MATERIAL AND METHODS

The study was a prospective case control study that was done in the Department of Otorhinolaryngology in a tertiary care teaching hospital to establish the possible contribution of hypercholesterolemia to the development of tinnitus. After receiving the consent of the Institutional Ethics Committee, the research was conducted within a specific period of the study.

The cases were enrolled consecutively as patients came to the outpatient department with complaints of tinnitus. The number of age and gender-matched controls with no tinnitus who visited the hospital because of non-otological complaints were also equal. All participants were carefully assessed in terms of clinical and biochemical examination. All the participants gave informed consent in writing and the study was carried out in accordance to the ethical principles of the Declaration of Helsinki.

A. Inclusion Criteria

➤ Cases:

- Patients aged ≥ 18 years
- Presence of subjective, non-pulsatile tinnitus

➤ Controls:

- Individuals aged ≥ 18 years
- No history of tinnitus

➤ Exclusion criteria

- History of head or neck trauma
- Presence of pulsatile or objective tinnitus
- Known otological diseases such as chronic otitis media or Meniere's disease
- History of prolonged occupational or recreational noise exposure
- Use of known ototoxic medications
- Presence of major psychiatric or neurological disorders
- Patients with identifiable causes of sensorineural hearing loss

III. METHODOLOGY

All participants were provided with a detailed clinical history focusing on the duration, occurrence of lateral tinnitus as well as its quality and were also asked to highlight the presence of other symptoms including hearing loss, vertigo, and aural fullness. Data on systemic diseases, the use of medication, lifestyle, and occupational noise exposure was recorded. Each study participant was thoroughly examined in terms of Otorhinolaryngology with the use of otoscopic examination. All cases and controls indicated by pure tone audiometry (PTA).

The estimation of serum lipid profile was done by fasting venous blood samples taken under aseptic conditions in all participants. The biochemical parameters were evaluated as a total cholesterol, low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol

(HDL), and serum triglycerides. The total cholesterol of 200mg/dl was considered to be hypercholesterolemia according to the standard reference values.

➤ Statistical Analysis

Statistical Package of the Social Sciences (SPSS) software was used in entering and analyzing the data. Demographic and clinical variables were summarized by using descriptive statistics, which included mean, standard deviation, frequency and percentage. The independent sample t-test was used to compare cases and controls with respect to continuous variables and the chi-square test was used to compare cases and controls with respect to categorical variables. Odds ratio with 95% confidence interval was

calculated to determine the strength of association between hypercholesterolemia and tinnitus. A p-value <0.05 was considered statistically significant.

IV. RESULTS

A total of 240 participants were included in this prospective case control study, comprising 120 patients with tinnitus (cases) and 120 age- and gender-matched individuals without tinnitus (controls). The mean age of cases was 47.8 ± 12.6 years, while that of controls was 46.3 ± 11.9 years, with no statistically significant difference between the groups ($p = 0.38$). The demographic characteristics of study participants are summarized in Table 1.

Table 1. Demographic Characteristics of Study Participants

Variable	Cases (n=120)	Controls (n=120)	p-value
Age group (years)			0.62
18–40	36 (30.0%)	40 (33.3%)	
41–60	55 (45.8%)	52 (43.3%)	
>60	29 (24.2%)	28 (23.4%)	
Gender			0.71
Male	74 (61.7%)	71 (59.2%)	
Female	46 (38.3%)	49 (40.8%)	

Hypercholesterolemia (total cholesterol ≥ 200 mg/dL) was observed in 63 patients (52.5%) in the tinnitus group compared to 34 patients (28.3%) in the control group, demonstrating a statistically significant association ($p = 0.001$). Elevated LDL, low HDL, and raised triglyceride levels were also significantly more prevalent among tinnitus patients (Table 2).

Table 2. Comparison of Lipid Profile Between Cases and Controls

Lipid parameter	Cases (n=120)	Controls (n=120)	p-value
Hypercholesterolemia	63 (52.5%)	34 (28.3%)	0.001*
Elevated LDL	69 (57.5%)	40 (33.3%)	0.002*
Low HDL	43 (35.8%)	25 (20.8%)	0.010*
Elevated triglycerides	54 (45.0%)	29 (24.2%)	0.003*

*Statistically significant

Among tinnitus patients, unilateral tinnitus was reported in 80 patients (66.7%), while bilateral tinnitus was present in 40 patients (33.3%). Fourtyfour patients (36.7) were noted to be associated with hearing deficiency, 31 patients (25.8) with vertigo, and 27 patients (22.5) with aural fullness (Table 3).

Table 3. Clinical Characteristics of Tinnitus Patients

Clinical variable	Number (n=120)	Percentage
Unilateral tinnitus	80	66.7%
Bilateral tinnitus	40	33.3%
Hearing impairment	44	36.7%
Vertigo	31	25.8%
Aural fullness	27	22.5%

Additional evaluation showed that hypercholesterolemic patients were far more likely to have bilateral tinnitus, pre-existing symptoms, and developed hearing loss than the patients with normal cholesterol (Table 4) did.

Table 4. Association of Hypercholesterolemia with Tinnitus Characteristics

Variable	Hypercholesterolemia (n=63)	Normal cholesterol (n=57)	p-value
Bilateral tinnitus	28 (44.4%)	12 (21.1%)	0.008*
Duration >6 months	45 (71.4%)	26 (45.6%)	0.004*
Hearing loss present	30 (47.6%)	14 (24.6%)	0.010*

*Statistically significant

The analysis of risk factors showed that Hypercholesterolemia was much more common in tinnitus cases than controls (52.5% vs 28.3, $p = 0.001$), which means that tinnitus is closely metabolically correlated with Hypercholesterolemia. Other outcomes that were significantly related to tinnitus were hypertension (31.7% vs 20.0, $p = 0.04$) and smoking (36.7% vs 24.2, $p = 0.03$), which indicated the role of vascular and lifestyle-related risks. Even though the patients with tinnitus had a higher prevalence of diabetes mellitus (21.7% vs 15.0% $p = 0.18$) and noise exposure (25.0% vs 17.5% $p = 0.16$), the differences were not statistically significant. These results highlight the leading role of hypercholesterolemia and vascular risk factors in the formation of tinnitus. (Table 5)

Table 5. Risk Factors Associated with Tinnitus

Risk factor	Cases (n=120)	Controls (n=120)	p-value
Hypercholesterolemia	63 (52.5%)	34 (28.3%)	0.001*
Hypertension	38 (31.7%)	24 (20.0%)	0.04*
Diabetes mellitus	26 (21.7%)	18 (15.0%)	0.18
Smoking	44 (36.7%)	29 (24.2%)	0.03*
Noise exposure	30 (25.0%)	21 (17.5%)	0.16

An aural test showed that sensorineural hearing loss was found in higher percentages amongst tinnitus patients than controls (36.7% vs 17.5, $p = 0.008$). Cases of tinnitus had more cases of mild to moderate hearing loss (Table 6).

Table 6. Pure Tone Audiometry Findings and Association with Tinnitus

PTA finding	Cases (n=120)	Controls (n=120)	p-value
Type of hearing loss			0.008*
SNHL	44 (36.7%)	21 (17.5%)	
CHL	12 (10.0%)	9 (7.5%)	
Mixed HL	4 (3.3%)	3 (2.5%)	
Normal	60 (50.0%)	87 (72.5%)	
Severity			0.041*
Mild	26 (21.7%)	15 (12.5%)	
Moderate	18 (15.0%)	10 (8.3%)	
Severe	6 (5.0%)	2 (1.7%)	

The logistic regression analysis revealed that hypercholesterolemia was a potent independent predictor of tinnitus, and individuals with the condition were at a risk of having tinnitus almost 3 times higher (OR = 2.79, 95% CI: 1.62479, $p = 0.001$). High LDL levels had significant changes with tinnitus (OR = 2.21, 95% CI: 1.323.69, $p = 0.003$) and lower levels of HDL were also associated with tinnitus to a modest but significant level (OR = 1.76, 95% CI: 1.023.02, $p = 0.039$). Smoking (OR = 1.82, 95% CI: 1.0731) and hypertension (OR = 1.68, 95% CI: 1.0132) were also found to be significant predictors of the vascular and lifestyle factors. The results indicate the independent role of lipid abnormalities and vascular risk factors in the pathogenesis of tinnitus. (Table 7)

Table 7. Multivariate Logistic Regression Model for Predictors of Tinnitus

Predictor	B	SE	p-value	OR	95% CI Lower	95% CI Upper
Hypercholesterolemia	1.02	0.29	0.001*	2.79	1.62	4.79
Elevated LDL	0.79	0.26	0.003*	2.21	1.32	3.69
Low HDL	0.57	0.28	0.039*	1.76	1.02	3.02
Hypertension	0.52	0.26	0.045*	1.68	1.01	2.82
Smoking	0.60	0.27	0.028*	1.82	1.07	3.10
Vertigo	0.88	0.31	0.004*	2.41	1.31	4.41

Pseudo $R^2 = 0.26$; Model accuracy = 71%

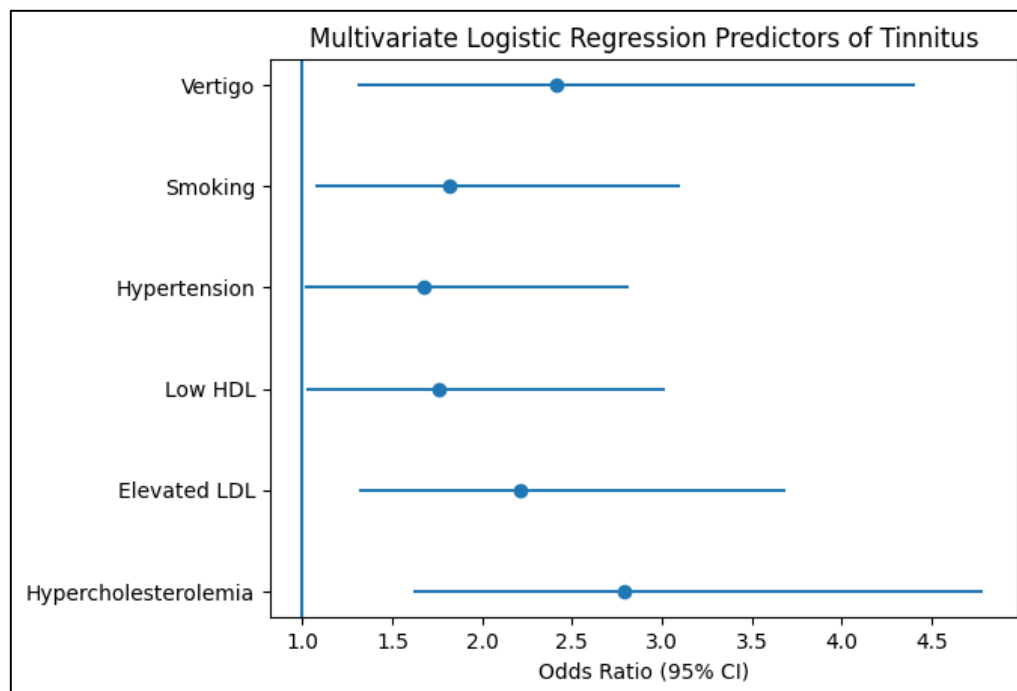


Fig 1. Multivariate Logistic Regression Predictors of Tinnitus

These findings demonstrate that hypercholesterolemia, vascular risk factors, and tinnitus are closely related to each other and a complex vascular-metabolic mechanism occurs during tinnitus pathogenesis.

V. DISCUSSION

One of the auditory symptoms is tinnitus, which is the ability to hear some sound in the absence of external acoustic stimuli, also known as a ringing, buzzing, hissing or whistling sensation [18-20]. It is a multifactorial etiological disorder and it is frequently associated with systemic metabolic and vascular disorders. Most recently, the role of lipid abnormalities in the pathophysiology of tinnitus is of increasing interest due to the possibility of lipid abnormalities to influence cochlear microcirculation and auditory activity [21,22].

The existing prospective case control study demonstrated the fact that the tinnitus had significant associations with hypercholesterolemia. The tinnitus patients had better chances of having high amounts of total cholesterol, LDL, and triglyceride and low amounts of HDL. These findings validate the hypothesis that the cochlear ischemia might involve lipid abnormalities in the following ways: dysfunction of the endothelium, high blood viscosity, and microvascular impairment of the inner ear. Such vasculature alterations would result in vascular dysfunction of oxygenation and metabolic homeostasis of the cochlear hair cells that would prefer the development of tinnitus.

The results of the current study resemble the findings of Musleh et al. (2022), which identified that patients who were high in lipids were at risk of tinnitus nearly twice as many as individuals who were normal lipid levels [1]. No less important, Erdogan et al. (2020) said that tinnitus and

dyslipidemia were associated with each other and that lipid abnormalities in the etiopathogenesis of the auditory dysfunction may not be neglected [2]. The results obtained also validate the hypothesis that inner ear perfusion and auditory processing might be affected by atherosclerotic changes induced by hyperlipidemia.

Previous studies have also discussed how the metabolic factors influence the auditory symptoms. Kaźmierczak et al. (2001) concluded that the metabolic disturbances patients of tinnitus and vertigo were experiencing included hyperlipidemia and abnormal glucose metabolism that suggested that the symptoms were systemically produced by the metabolism [22]. In addition, Spencer et al. (1973) were the first to introduce the connection between hyperlipoproteinemia and inner ear disease since it turned out to be a cause of sensorineural hearing loss and vestibular impairment [23]. This was subsequently confirmed by Evans et al. (2006) who reported that sustained dyslipidemia may have adverse consequences on auditory processes particularly because of elevated levels of triglycerides in the body as well as impaired cochlear vascularity [24].

Interventional evidence also supports the use of lipid regulation in the treatment of tinnitus. According to Satbas et al., (2007), the large percentage of patients with hyperlipidemia exhibiting significant improvement or even remission of their symptoms of tinnitus were noted in both lipid-lowering treatment and dietary intervention [25]. Similarly, as put across by Pulec et al. (1997), the application of hypolipidemic dietary interventions was found to increase the efficiency of the lipid profile as well as the level of tinnitus, and this pointed to a reversible vascular pathology of tinnitus in the selected group of patients [26].

On the other hand, other researchers have come up with conflicting findings. Shirazi et al. (2011) have also not found any statistically significant relationship between dyslipidemia and the prevalence of tinnitus but hypercholesterolemia was highest in the affected people [27]. The same case, Lee et al. have discovered that the differences in lipid profile in elderly individuals with hearing impairment were little and statistically non-significant [28]. These variations can be attributed to the variance in the study design, population, sample size and diagnostic criteria.

The current study that verified the role of vascular risk factors in auditory dysfunction also reported hypertension and smoking to be significant predictors of tinnitus. They correlate their findings with the epidemiological research by Shargorodsky et al. who discovered higher rates of tinnitus among individuals, with the presence of cardiovascular predisposing factors, and those exposed to environmental noise [29,30].

Overall, the findings of the present paper could be commented upon in the framework of the growing amount of evidence that hypercholesterolemia and the lipid dysregulations associated with it are the pathogenesis factors of tinnitus. The results indicate how crucial it is to implement the metabolic evaluation of the patient who developed the mentioned tinnitus in detail and underline how potentially helpful the application of lipid-lowering measures can be. However, the current research has some drawbacks. The sample size was rather small and limited to a single tertiary care center and this may be a weakness in terms of generalizability. The nature of the cross-section makes it impossible to make causal inferences. The longitudinal follow-up and objective data of the severity of tinnitus were not addressed which could have influenced the determination of the time relations and the results of treatment.

Larger longitudinal cohort designs are required to establish the causal relationship of specific lipid fractions on the intensity of tinnitus and examine the appropriateness of certain metabolic interventions in the treatment of tinnitus.

VI. CONCLUSION

The findings indicate the important correlation between hypercholesterolemia and tinnitus, which suggests that the metabolic and vascular disturbances might be one of the key factors affecting the onset and further evolution of auditory impairment. The disruption of cochlear microcirculation and disrupted neural signaling appear to be caused by the changes in lipid profile and, in particular, the rise in the concentration of cholesterol and LDL. Recognition of the discussed modifiable risk factors gives an opportunity to act in time during the early intervention through the specific regulation of the metabolism and lifestyle modification. Incorporating lipid testing into the routine evaluation of tinnitus may not only be helpful in determining the possible contributors at the system level, but may also be used to introduce the more personal and multifaceted treatment of the issue to improve the patient outcomes and quality of life.

REFERENCES

- [1]. Musleh A, Alshehri S, Qobty A. Hyperlipidemia and its relation with tinnitus: Cross-sectional approach. *Niger J Clin Pract* 2022;25:1046-9.
- [2]. Erdogdu S. What is the prevalence of dyslipidemia in patients with idiopathic tinnitus?. *Int J Res Med Sci* 2020;8:2983-7
- [3]. Papadakis MA, McPhee SJ. *Current Medical Diagnosis and Treatment* 2019. New York: McGraw-Hill; 2019.
- [4]. McCormack A, Edmondson-Jones M, Somerset S, Hall D. A systematic review of the reporting of tinnitus prevalence and severity. *Hear Res.* 2016;337:70-9.
- [5]. Lutman ME, editor. *Hearing Science and Hearing Disorders*. London: Academic Press; 2014.
- [6]. Henry JA, Zaugg TL, Schechter MA. Clinical guide for audiologic tinnitus management I. *Am J Audiol.* 2005;14:21-48.
- [7]. Hoare DJ, Kowalkowski VL, Kang S, Hall DA. Systematic review and meta-analyses of randomized controlled trials examining tinnitus management. *Laryngoscope.* 2011;121:1555-64.
- [8]. Sindhusake D, Golding M, Newall P, Rubin G, Jakobsen K, Mitchell P. Risk factors for tinnitus in a population of older adults: The Blue Mountains Hearing Study. *Ear Hear.* 2003;24:501-7.
- [9]. Hoffman HJ, Reed GW. Epidemiology of tinnitus. In: Snow JB Jr, editor. *Tinnitus: Theory and Management*. Hamilton: BC Decker; 2004. p. 16-41.
- [10]. Axelsson A, Prasher D. Tinnitus induced by occupational and leisure noise. *Noise Health.* 2000;2:47-54.
- [11]. Guest H, Munro KJ, Prendergast G, Howe S, Plack CJ. Tinnitus with a normal audiogram: Relation to noise exposure but no evidence for cochlear synaptopathy. *Hear Res.* 2017;344:265-74.
- [12]. El-Shunnar SK, Hoare DJ, Smith S, Gander PE, Kang S, Fackrell K, et al. Primary care for tinnitus: Practice and opinion among GPs in England. *J Eval Clin Pract.* 2011;17:684-92.
- [13]. Gopinath B, McMahon CM, Rochtchina E, Karpa MJ, Mitchell P. Risk factors and impacts of incident tinnitus in older adults. *Ann Epidemiol.* 2010;20:129-35.
- [14]. Baguley D, McFerran D, Hall D. Tinnitus. *Lancet.* 2013;382:1600-7.
- [15]. Mohammed AA. Lipid profile among patients with sudden sensorineural hearing loss. *Indian J Otolaryngol Head Neck Surg.* 2014;66:425-8.
- [16]. Hameed MK, Sheikh ZA, Ahmed A, Najam A. Atorvastatin in the management of tinnitus with hyperlipidemia. *J Coll Physicians Surg Pak.* 2014;24:927-30.
- [17]. Sutbas A, Yetiser S, Satar B, Akcam T, Karahatay S, Saglam K. Low-cholesterol diet and antilipid therapy in managing tinnitus and hearing loss in patients with noise-induced hearing loss and hyperlipidemia. *Int Tinnitus J.* 2007;13(2):143-149.

- [18]. Weisz N, Hartmann T, Dohrmann K, Schlee W, Norena A. High-frequency tinnitus without hearing loss does not mean absence of deafferentation. *Hear Res* 2006;222:108-14.
- [19]. Henry JA, Griest S, Zaugg TL, Thielman E, Kaelin C, Galvez G, et al. Tinnitus and hearing survey: A screening tool to differentiate bothersome tinnitus from hearing difficulties. *Am J Audiol* 2015;24:66-77.
- [20]. Seidman MD, Standring RT, Dornhoffer JL. Tinnitus: Current understanding and contemporary management. *Curr Opin Otolaryngol Head Neck Surg* 2010;18:363-8.
- [21]. Itabe H. Oxidative modification of LDL: Its pathological role in atherosclerosis. *Clin Rev Allergy Immunol* 2009;37:4-11.
- [22]. Kaźmierczak H, Doroszeńska G. Metabolic disorders in vertigo, tinnitus, and hearing loss. *Int Tinnitus J* 2001;7:54-8.
- [23]. Spencer JT. Hyperlipoproteinemia in the etiology of inner ear disease. *Laryngoscope*. 1973;83:639-78.
- [24]. Evans MB, Tonini R, Shope CD, Oghalai JS, Jerger JF, Insull W Jr, et al. Dyslipidemia and auditory function. *Otol Neurotol*. 2006;27(5):609-14.
- [25]. Sutbas A, Yetiser S, Satar B, Akcam T, Karahatay S, Saglam K. Low-cholesterol diet and anti-lipid therapy in managing tinnitus and hearing loss in patients with noise-induced hearing loss and hyperlipidemia. *Int Tinnitus J*. 2007;13(2):143-9.
- [26]. Pulec JL, Pulec MB, Mendoza I. Progressive sensorineural hearing loss, subjective tinnitus, and vertigo caused by elevated blood lipids. *Ear Nose Throat J*. 1997;76(10):716-20.
- [27]. Shirazi M, Farhadi M, Jaleesi M, Kamrava SK, Heshmatzadeh-Behzadi A, Arami B. Prevalence of dyslipidemia among Iranian patients with idiopathic tinnitus. *J Res Med Sci*. 2011;16(7):890-6.
- [28]. Lee FS, Matthews LJ, Mills JH, Dubno JR, Adkins WY. Analysis of blood chemistry and hearing levels in a sample of older persons. *Ear and hearing*. 1998 Jun 1;19(3):180-90.
- [29]. Alberti KG, Eckel RH, Grundy SM. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and Int Association for the Study of Obesity. *Circulation*. 2009;120(16):1640-5.
- [30]. Shargorodsky J, Curhan GC, Farwell WR. Prevalence and Characteristics of Tinnitus among US Adults. *Am J Med* 2010;123:711-8.