Lipid Profile among Patients with Sudden Sensorineural Hearing Loss

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Abstract Associations between hearing and blood lipids have been the focus of scientific inquiry for more than 50 years. The aim of the present study is to evaluate the association between hyperlipidaemia among patients presented with sudden sensorineural hearing loss compared to normal controls. A case control study concerned with 22 patients presented with sudden sensorineural hearing loss who underwent lipid profile evaluation. The lipid profile of these patients was compared with corresponding results of 55 age matched persons (volunteers) with normal hearing. These patients were collected from the Out Patient Department of ENT at Al-Jamhory Teaching Hospital, Mosul/Iraq and private clinic of the author for the period from February 2011 to July 2013. The average age of patients was 44.7 years with a range of 26–65 years. The peak age incidence was in the 5th decade of life. The study included 11 male patients (50 %) and 11 females (50 %). Meanwhile, the average age of the control group was 41.7 years with 25 (45.5 %) males and 30 (54.5 %) females. Statistical analysis showed that there was significant difference between the means of lipid profile and blood sugar of the patients and the control group apart from HDL where there was no significant difference. In conclusion, hyperlipidemia seems to be significantly associated with the occurrence of sudden sensorineural hearing loss according to this study.

Keywords Sudden sensorineural hearing loss · Lipid profile · Hyperlipidaemia · Dyslipidaemia

Introduction

Wilson et al. defined idiopathic sudden sensorineural hearing loss (SSHL) as sensorineural hearing loss greater than 30 dB over at least three contiguous frequencies occurring over a period of 3 days [1]. In recent years there has been a significant increase in the diagnosis of SSHL in western, countries with an incidence of 20 of 100,000 people affected every year. No clear causes for this disease have been found thus far, but cochlear ischemia has been hypothesized in patients in whom an infectious episode or acoustic neurinoma have been excluded [2].

The pathogenesis of sudden hearing loss (SHL) has not been elucidated as yet. Insufficient perfusions of the cochlea due to an increased blood viscosity, microthrombosis, or altered vasomotion are assumed. Hypercholesterolemia and hyperfibrinogenemia are frequently observed in patients with SSHL [3].

Many investigators who have analyzed the possible correlation between hearing loss and high serum cholesterol levels have found that hearing appears to be influenced by high blood lipids [4]. Vascular events, immunological processes and viral infections have to be considered as pathomechanisms for most cases of SHL. Heparin-induced extracorporeal low-density lipoprotein (LDL) precipitation (HELP) apheresis which acutely and drastically reduces LDL, fibrinogen and lipoprotein (a) has been reported in the treatment of patients suffering from SHL. It was proved to be superior or at least equal to the more expensive standard treatment with prednisolone, dextranes and pentoxifylline [5, 6].

Our study was designed to evaluate the association between hyperlipidaemia among patients presented with SSHL compared to normal controls Table 1.

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Table 1 Lipid profile among patients and control groups

Lipid profile	Patients <i>n</i> = 22 Mean ± SD	Control <i>n</i> = 55 Mean ± SD	<i>P</i> value ^a
Age (Years)	44.7 ± 11.3	41.7 ± 11.1	0.070
Sex			
Males	M = 11	M = 25	0.718 ^b
Females	F = 11	F = 30	
Total cholesterol (mg/dl)	190.5 ± 43.2	145.1 ± 31.5	0.000
Triglyceride (mg/dl)	124.5 ± 52.5	73.6 ± 27.7	0.000
HDL (mg/dl)	48.3 ± 8.75	49.7 ± 8.77	0.574
LDL (mg/dl)	118.6 ± 36.0	81.8 ± 34.2	0.000
Atherogenic index	3.9 ± 1.12	2.9 ± 1.06	0.000
Blood sugar (mg/dl)	104.7 ± 34.9	84.7 ± 8.36	0.000

^a Independent *t*-test for two means was used^b Chi square test was used for categorical variables

Patients and Methods

This case control study is concerned with 22 patients presented with SSHL (group I) who underwent lipid profile evaluation. Lipid profile was compared with the corresponding results of 55 age matched persons (volunteers) with normal hearing (Group II). These patients were collected from the Out Patient Department of ENT at Al-Jamhory Teaching Hospital, Mosul/IRAQ and the private clinic of the author for the period from February 2011 to July 2013.

The inclusion criterion was sensorineural hearing loss greater than 30 dB over at least three contiguous frequencies occurring over a period of 3 days. Patients with recent history of upper respiratory tract infection were excluded from this study.

Detailed history was taken and full ENT examination was performed including otoscopic examination. All patients had Rinne and Weber tuning fork tests using 512 Hz fork to confirm sensorineural hearing loss.

Audiological evaluation was done for patients and control group in a sound attenuated booth using Amplivox 260 diagnostic audiometer (Made in England). Pure tone audiometry was determined at frequencies of 250, 500, 1000, 2000, 4000 and 8000 Hz for air and 250, 500, 1000, 2000 and 4000 Hz for bone. Sensorineural hearing loss was diagnosed if both air and bone conduction thresholds were superimposed with no air-bone gap. Hearing level was determined by pure tone average for air conduction at frequencies 500, 1000 and 2000 Hz. Hearing was labeled as abnormal if the hearing threshold was 26 dB or more at two or more test frequencies. All audiometries were performed by one audiometrist whom was kept blind to the lipid status of the participants.

Magnetic resonance imaging was done for all patients to exclude cerebellopontine angle tumours. Moreover,

complete blood picture, fasting blood sugar and lipid profile were performed for patients.

Five milliliters of venous blood samples were collected from patients and control group after at least 12 h fasting for evaluation of serum lipid profile. Then this blood was put in plain tubes and allowed to clot in water bath at 37 °C followed by centrifugation of blood at 3000 rpm for 10 min. Serum was aspirated and stored at −20 °C until required for measurement of total cholesterol (T-chol), serum triglyceride (TG) and high-density lipoprotein (HDL) levels. LDL levels were calculated by an indirect method using the Friedewald formula.

$$LDL = Total\ cholesterol - \left(\frac{HDL - Triglyceride}{2.22} \right)$$

On the other hand, Atherogenic index was calculated by the following equation:

$$Atherogenic\ index = \frac{Total\ cholesterol}{HDL}$$

Exclusion criteria

- History of recent upper respiratory tract infection.
- Noise induced hearing loss.
- Patients using ototoxic drugs.
- Patients with ear discharge.
- Hearing loss due to other causes like otitis media, Meniere's disease, otosclerosis, presbycusis.
- Patients with medical diseases as diabetes mellitus, ischaemic, cerebrovascular or haematological diseases.

All continuous variables are presented as the mean ± SD and compared using independent *t* test. Categorical data are shown as percentages and compared using the Chi square test. A multivariate non-conditional logistic regression analysis is conducted to study the effect of hyperlipidemia on sensorineural hearing loss. All analyses were conducted

using SPSS version 11.5 for Windows (SPSS Inc., Chicago, IL USA) and *P* values of less than 0.05 were considered to indicate statistical significance.

Results

The study included 22 patients presented with SSHL (Group I) with a mean age of 44.7 years and a range of 17–65 years. There were 11 male patients (50 %) and 11 females (50 %). The peak age incidence was in the 5th decade of life (Fig. 1).

SHL affected right ear in 10 (45.5 %) patients, left ear in 11 (50 %) patients whereas one patient (4.5 %) suffered from bilateral hearing loss. This patient proved to have encephalitis and died after 3 days. The mean hearing level of the affected ear was 60.5 dB.

The control group (Group II) included 55 persons (Volunteers) with normal hearing. The mean age of them was 41.7 years with 25 (45.5 %) males and 30 (54.5 %) females.

Statistical analysis using Chi square test revealed that there was no significant difference in age and sex between patients (Group I) and control group (Group II). Moreover, independent *t* test was used for comparison of lipid profile means between the above two groups. The latter test showed that there was significant difference between the means of lipid profile and blood sugar between the patients and the control group apart from HDL where there was no significant difference.

Discussion

Although the pathogenesis of SHL is not as yet known, the clinical picture and the frequent association with vascular risk factors make an ischaemic event likely [5].

Fig. 1 Age distribution of the patients among the sample of study

The mean age of our patients was 44.7 years with almost equal sex distribution. Similarly, Maru and Jain [7] concluded that atherosclerotic disease of the labyrinthine vessels usually affects patients above 40 years of age. It occurs bilaterally. Commonly both the cochlear and vestibular components are involved. Raised serum total cholesterol, L.D.L. cholesterol levels and hypertension, are more contributory as risk factors in its development. Moreover, Oiticica J and Bittar RSM [8] in studying the prevalence of metabolic disorders among patients with SSHL in Brazil noted that the mean age of their patients was 46.5 years and that 43.9 % were males and 56.1 % were females.

Our results revealed that there was significant difference between the means of lipid profile and blood sugar of the patients and the control group apart from HDL where there was no significant difference.

On comparison, Suckfull et al. [3] concluded that hyperfibrinogenemia and hypercholesterolemia may contribute to the clinical event of SHL. Their study showed for the first time that acute and drastic removal of plasma fibrinogen and low density lipoproteins can be an effective clinical tool in the treatment of patients with SHL. Similarly, Marcucci et al. [2] data suggested that hypercholesterolemia, hyperhomocysteinemia, elevated PAI-1 levels and anticardiolipin antibodies are associated with idiopathic sudden sensorineural hearing loss ISSHL, so indirectly supporting the hypothesis of a vascular occlusion in the pathogenesis of the disease. Moreover, Thakur J S et al. [9] found that LDL were significantly associated with many waveforms in hyperlipidaemic patients. Thus, LDL may be important in auditory dysfunction.

On the other hand, Axelsson and Lindgren [4] results indicated an increased risk of acquiring high frequency sensorineural hearing loss for people who work in noisy environments and have high serum cholesterol levels. Moreover, Sutbas et al. [10] in studying the effect of low cholesterol diet and antilipid therapy in managing tinnitus and hearing loss in patients with noise-induced hearing loss and hyperlipidemia found that the incidence of hyperlipidemia is high among patients with noise-induced hearing loss. Significant improvement by way of lowered tinnitus intensity and hearing thresholds can be achieved after lowering the serum cholesterol levels.

Oiticica J, Bittar RSM [8] concluded that hyperglycemia and thyroid disorders are much more frequent in patients with sudden deafness than in the general population of Brazil and should be considered as important associated risk factors for sudden deafness. Furthermore, there was an increase on the prevalence of hypercholesterolemia in the sensorineural hearing loss SHL patient sample compared to the historical reference value for the Brazilian population, whereas, no difference was observed regarding LDL cholesterol fraction or triglyceride.

Furthermore, Gopinath et al. [11] in a longitudinal study, assessed associations between age-related hearing loss and dietary intake of cholesterol, as well as the use of cholesterol-lowering drugs, and reported that high dietary intake of cholesterol, but not blood total cholesterol, was associated with increased likelihood of hearing loss. Whereas treatment with statins and consumption of monounsaturated fats may have a beneficial influence.

On the contrary Ullrich, Aurbach and Drobik [12] findings indicated that both hyperlipidemia and atherogenic risk factors are not of major pathological importance in sudden hearing loss SSHL. Moreover, Kazmierczak H and Doroszewska G [13] concluded that disturbances of glucose metabolism as diabetes mellitus and hyperinsulinemia may be responsible for inner ear diseases, whereas the role of disturbances of lipid metabolism remains vague. Furthermore, Anbari et al. [14] found that dyslipidemia seems to have no association with sensorineural hearing loss in 5–18 years old children according to their study.

Simpson AN, Matthews LJ and Dubno JR [15] in a cross-sectional sample of 837 subjects found modest associations between triglycerides and all PTAs. Weak associations were observed between the ratio of total cholesterol and HDL and narrow PTA, broad PTA, and high-frequency PTA. However, when assessing changes in hearing and lipids over time in a longitudinal analysis, no significant associations between hearing and lipids remained. Thus, the association is either spurious or mediated by other factors, which are yet to be identified. Recent reports of the effects of diet and lipid-lowering therapy, as well as smoking, suggest that the exploration of the association between hearing and dietary factors and environmental factors may prove to be more fruitful than a continuing focus on lipid levels.

Sutbas et al. [10] cited that the exact pathological mechanism for the hyperlipidaemia-induced hearing loss remained obscure. Increased blood viscosity and atherosclerosis of the cochlear vessels reduce the blood perfusion of the cochlea and promote hearing impairment. Vascular mechanisms are not solely responsible for the auditory dysfunction. Lipidosis of the inner ear has been postulated by Nguyen and Brownell as an alternate mechanism. The latter authors showed that the lateral wall of outer hair cells from Guinea pig cochlea incorporates water-soluble cholesterol. This uptake of cholesterol is accompanied by an increased stiffness of the cells which may impair the cells' electromotile response.

Conclusion

All variants of blood lipids were significantly higher among patients with sudden sensorineural hearing loss than the control group apart from HDL. Thus, hyperlipidemia

seems to be significantly associated with the occurrence of sudden sensorineural hearing loss according to this study.

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