LIPID PROFILE IN SUDDEN SENSORINEURAL HEARING LOSS- A PROSPECTIVE STUDY

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ABSTRACT

BACKGROUND

To study the correlation between hyperlipidaemia among patients presented with sudden sensorineural hearing loss compared to normal controls.

MATERIALS AND METHODS

30 adults aged 22 to 58 years with sudden sensorineural hearing loss were included in the prospective study and was compared with serum lipids level. Relevant history was obtained. Examination and investigations were conducted.

RESULTS

Statistical analysis showed that there was significant difference between the means of lipid profile of the patients and the control group.

CONCLUSION

The study shows significant relationship between hyperlipidaemia and sudden sensorineural hearing loss, but more studies are required to establish a pathological connection between lipid levels and SSNHL.

KEYWORDS

Sudden Sensorineural Hearing Loss, Lipid Profile, Hyperlipidaemia.

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BACKGROUND

Sudden Sensorineural Hearing Loss (SSNHL) is defined as hearing impairment of more than a 30 dB decrease, occurring over a 72-hour period on three consecutive frequencies of pure tone audiometry.[1] Its incidence is estimated at 5 - 20/10,000 individuals per year.[2,3] In recent years, there has been a significant increase in the diagnosis of SSNHL. Although, various potential causes have been reported including viral infections and immunologic diseases,[2,4–6] most cases of SSNHL are idiopathic. Recently, cochlear ischaemia has been hypothesised in patients in whom an infectious episode or acoustic neuroma have been excluded.[2] Insufficient perfusions of the cochlea due to an increased blood viscosity, microthrombosis or altered vasomotion are assumed. Hypercholesterolaemia and hyperfibrinogenaemia are frequently observed in patients with SSNHL.[7]

It is no wonder that vascular theory has been advocated as the main pathogenesis behind SSNHL because SSNHL, Cardiovascular Disease (CVD) and stroke are more likely to present in a similar fashion (ex: abruptly onset) in older age. However, other studies have reported that the vascular risk factors could not be responsible for auditory damage.[6,8] Thus, the association between SSNHL and vascular risk factors is still controversial.

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

MATERIALS AND METHODS

This prospective study is concerned with 30 patients presented with SSNHL (Group I), who underwent lipid profile evaluation. Lipid profile was compared with the corresponding results of 30 age matched persons (controls) with normal hearing (Group II) who attended Outpatient Department of ENT at Kempegowda Institute of Medical Sciences, Bangalore for symptoms other than hearing loss from January 2016 to January 2017.

The inclusion criteria was sensorineural hearing loss greater than 30 dB over at least three contiguous frequencies occurring over a period of 3 days. Detailed history was taken and full ENT examination was performed including otoscopic examination. Audiological evaluation was done for patients and control group in a sound attenuated room. 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

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was labelled as abnormal if the hearing threshold was 26 dB or more at two or more test frequencies. All audiometries were performed by one audiometrist, who was kept blind to the lipid status of the participants.

Exclusion criteria included- 1) Mixed hearing loss due to middle ear pathology, 2) Prior history of sudden deafness or previous ear surgery, 3) Fluctuating hearing loss such as Meniere disease, 4) Noise induced hearing loss, 5) Patients using ototoxic drugs, 6) 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 hyperlipidaemia on sensorineural hearing loss. P values of less than 0.05 were considered to indicate statistical significance.

RESULTS

The study included 30 patients presented with SSNHL (Group I) with a mean age of 42.7 years and a range of 22 - 58 years. There were 15 male patients (50%) and 15 females (50%). The peak age incidence was in the 5th decade of life. The mean hearing level of the affected ear was 60.5 dB.

The control group (Group II) included 30 persons (controls) with normal hearing. The mean age of them was 40.7 years with 15 (50%) males and 15 (50%) 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.

Sl. No.	Lipid Profile	Patients n=30	Controls n=30	P Value
1.	**Age**	42.7 ± 11.3	4.7 ± 11.1	0.070
2.	**Sex**			
	Male Female	M = 17 F = 13	M = 14 F = 16	0.714
3.	**Total cholesterol (mg/dL)**	192.5 ± 43.2	142.1 ± 31.5	0.0001
4.	**Triglyceride (mg/dL)**	122.5 ± 52.5	67.6 ± 27.7	0.0001
5.	**HDL**	47.2 ± 8.75	42.7 ± 8.77	0.0541
6.	**LDL**	120.6 ± 36.0	76.1 ± 34.2	0.0001

Table 1

DISCUSSION

The results of our study revealed that lipid profiles, such as elevated TC and TG levels are significantly associated with the prevalence of SSNHL and its prognosis. Because the cochlea is very vulnerable to ischaemic injury and lacks collateral circulation besides the cochlear artery (supplied by the labyrinthine artery), blood viscosity elevations due to hyperlipidaemia can be attributed to produce disturbance in the cochlear microcirculation. Thus, blood lipid level has been suggested as a main risk factor of SSNHL as well as CVD.[2,6,9] However, many studies of the association between SSNHL and lipid profiles are inconclusive with often contradictory results.[9,10,11,12] The mean age of our patients was 42.7 years with almost equal sex distribution. Similarly, Maru and Jain[13] 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, LDL cholesterol levels and hypertension are more contributory as risk factors in its development. Moreover, Oiticica J and Bittar RSM[14] 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 of the patients and the control group.

On comparison, Suckfull et al[7] concluded that hyperfibrinogenaemia and hypercholesterolaemia 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[15] data suggested that hypercholesterolaemia, hyperhomocysteinaemia, 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[16] 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[17] 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[18] 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 hyperlipidaemia found that the incidence of hyperlipidaemia 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 and Bittar RSM concluded that hyperglycaemia 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 in the prevalence of hypercholesterolaemia 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[19] 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[8] findings indicated that both hyperlipidaemia and atherogenic risk factors are not of major pathological importance in sudden hearing loss (SSHL). Moreover, Kazmierczak H and Doroszeweska G[20] concluded that disturbances of glucose metabolism as diabetes mellitus and hyperinsulinaemia may be responsible for inner ear diseases, whereas the role of disturbances of lipid metabolism remains vague. Furthermore, Anbari et al[21] found that dyslipidaemia 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[22] 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 overtime 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 suggests 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[18] 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, hyperlipidaemia seems to be significantly associated with the occurrence of sudden sensorineural hearing loss according to this study, but conclusive decision cannot be made owing to small sample size. Hence, we recommend further studies involving large sample studies in the future.

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