



Original Research Article

Status of Lipid Profile and Uric Acid in Sudden Sensorineural Hearing Loss

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Abstract

Background: Sudden Sensory Neural Hearing Loss (SSNHL) is loss of 30dB HL over three contiguous audiometric frequencies within 3 days and the incidence is 5-20 per 1,00,000 population occurring mostly among adults in fifties and sixties. The cause is idiopathic but recently been attributed to disturbance in cochlear microcirculation secondary to dyslipidaemia.

Aim and Objective: The aim of our study is to establish a relationship between sudden sensory neural hearing loss (SSNHL) and vascular risk factors like serum lipid and uric acid.

Material & Methods: This is a case control study of 40 cases of SSNHL of age group 26-65 years attending the outpatient Department (OPD) of E.N.T, VIMSAR, Burla between 1st September to 30th November, 2017. After patient history, physical examinations and audiological examination, blood parameters like lipid profile and uric acid level were analysed. Levels of total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL-CH), very low density lipoprotein (VLDL-CH), high density lipoprotein (HDL-CH) and uric acid (UA) of these patients then compared with an age and sex matched control group of 40 patients treated in the same OPD for E.N.T diseases other than hearing loss during the same period.

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

Conclusion: This study indicates that metabolic disturbances of serum lipids and uric acid may be potential risk factors for SSNHL.

Keywords: Sudden sensory neural hearing loss(SSNHL), dyslipidaemia, uric acid.

Introduction

Sudden SNHL has been defined as sensory neural hearing loss of 30dB over at least in 3 contiguous audiometric frequencies occurring within 3 days.¹

The incidence of SSNHL has been estimated to

range from 5-20 cases per 1,00,000 in general population and occurs most commonly among adults in 50s or 60s^{2,3} accounting for 1% of all cases of sensory neural hearing loss.⁴ SSNHL is assumed to be a multi-factorial disorder and

etiology can be identified in about 10-15% of all cases which include microcirculation disturbances, infection, autoimmune disorders. But in most instances (approximately 70% of all cases) SSNHL is idiopathic in origin.⁵ As there seems to be similarities between cochlear microcirculation disturbance and cardio or cerebrovascular embolism, in recent years many studies have been conducted to evaluate the role of various cardiovascular risk factors and blood vessel embolism in SSNHL.^{6,7} There is some evidence that dyslipidaemia may be a risk factor for SSNHL and has been hypothesised to contribute to the initiation of an inflammatory or stressful response in the inner ear leading to SSNHL.⁸ There is also evidence that lipid lowering therapy (low density lipoprotein cholesterol [LDL-CH] apheresis) can be more effective than traditional treatment for patients with SSNHL who have elevated serum LDL-CH concentrations.⁸ But there is still no consensus regarding dyslipidaemia as a cause of SSNHL as there are other studies that rule out such an association.^{9,10} Hence additional studies are needed to ascertain the relationship or rule out the same, between serum lipids and SSNHL.

Based on the lack of definitive information regarding association of dyslipidaemia and SSNHL, we designed the study to evaluate the association between hyperlipidaemia among patients presented with SSNHL compared to normal controls in our setup.

We combined the study with association of hyperuricemia in SSNHL as many patients visiting the hospital are in elderly age group having arthritis problem and raised uric acid level. Studies show that direct penetration of uric acid into outer hair cells will cause stiffness in outer hair cell, hence motility disorders and electrical response.¹¹ Also uric acid sets up inflammatory procedure leading to endothelial dysfunctions in striae vascularis (indirect mechanism) and functional disorder of endocochlear potential.^{12,13}

Material and Methods

This case control study was conducted in the Department of E.N.T in association with Department of Biochemistry, VIMSAR, Burla, during a period of 3 months from 1st September to 30th November, 2017.

The study protocol was reviewed and approved by the Ethical Committee of VIMSAR, Burla.

Subjects were 40 clinically diagnosed SSNHL patients attending the outpatient Department of E.N.T. The inclusion criteria were sensory neural hearing loss greater than 30dB over at least three contiguous frequencies occurring over a period of 3 days.

After detailed history and physical examination, degree of hearing loss was determined using pure tone audiometry in a sound attenuated chamber. The audiologist was kept blind to the status of participants as case or control.

Exclusion criteria

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

5ml of venous blood samples were collected from all patients after 12 hours of fasting for evaluation of lipid profile and uric acid. The sample was allowed to clot, then centrifuged at 3000rpm for 10 mins. Then the test was carried out with the supernatant fluid. Measurement of total cholesterol (TC), triglyceride (TG), and high density lipoprotein (HDL) were done in semi auto analyser by enzymatic method. Serum LDL-CH was calculated by standard WHO approved Friedewald's formula

i.e., $LDL = TC - (HDL + VLDL)$, where $VLDL = TG/5$.

Estimation of uric acid was done in semi auto analyser by uricase method. Statistical analysis was done using SPSS version 18. Unpaired t-test was used for comparison

between the study and control population. Statistical significance was set at p value less than or equal to 0.05.

Results

Table 1: Biochemical parameters of the study groups

Parameters	Cases(n=40)	Controls(n=40)	t value	P value	95% confidence interval of the difference	
					Lower	Upper
AGE	45.28±10.83	44.75±10.18	0.223	0.824	-4.154	5.204
TG(mg/dl)	172.72±20.302	139.88±20.124	7.268	0.000	23.852	41.848
TC(mg/dl)	183.10±4.886	173.92±13.99	3.16	0.002	3.395	14.955
HDL(mg/dl)	42.60±5.39	47.75±6.34	-3.91	0.000	-7.771	-2.529
VLDL(mg/dl)	34.52±4.05	27.98±4.04	7.24	0.000	4.749	8.351
LDL(mg/dl)	105.98±13.72	98.20±15.13	2.40	0.018	1.316	14.205
Uric acid (mg/dl)	6.72±0.52	5.70±0.75	7.06	0.000	0.740	1.320

Fig 1: Sex distribution in the cases.

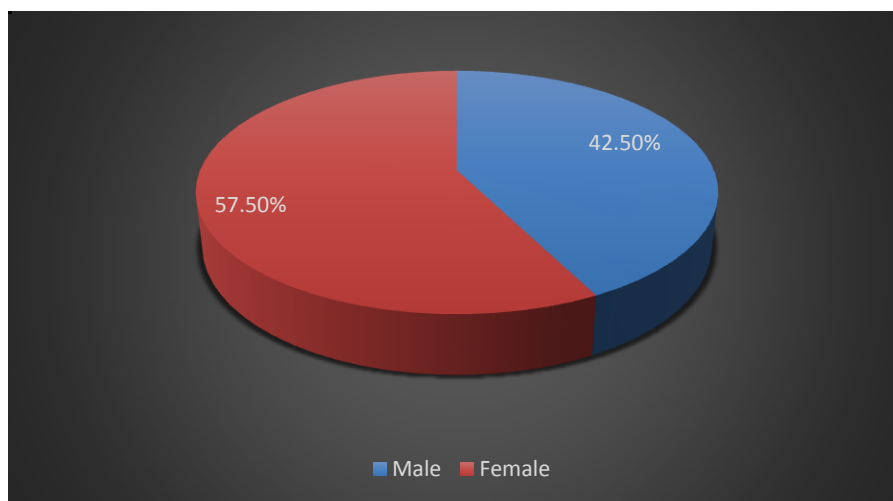
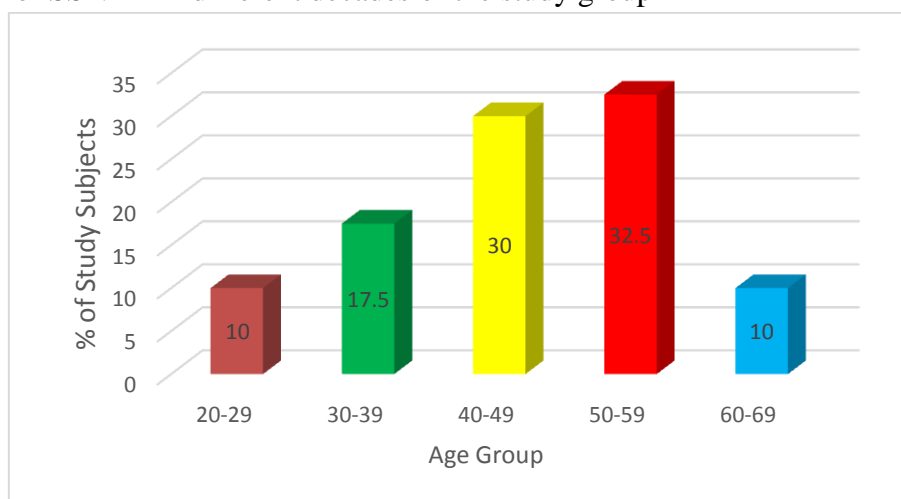


Fig 2: Distribution of SSNHL in different decades of the study group



The study included 40 patients with SSNHL with a mean age of 45.28 years ranging from 25-65 years. There were 17 males (43%) and 23 females

(57%). [Fig;1] The peak age incidence was in the 6th decade of life. [Fig;2].

Table 1; The mean age of control group was 44.75 years. Calculated value of TC, TG and LDL for cases and control were 183.10 ± 4.886 , 172.72 ± 20.302 , 105.98 ± 13.72 and 173.92 ± 13.99 , 139.88 ± 20.124 , 98.20 ± 15.13 respectively, $p \leq 0.05$. HDL levels is higher in control group (47.75 ± 6.34) with $p < 0.05$. Similarly uric acid level was found to be higher in case (6.72 ± 0.52) comparison to control (5.70 ± 0.75). For all the parameters statistically significant difference was found between the cases and control ($p \leq 0.05$).

Discussion

The pathogenesis of SSNHL remains unknown. Numerous conditions produce SSNHL including infectious disease, trauma, circulatory disturbance, ototoxicity and neoplasm. The disturbance of cochlear perfusion seems to have a prominent effect on the development of SSNHL and association of vascular risk factors make such ischemic events more likely.¹⁴ Gender, regional differences, seasons and smoking have no effect in the development of the disease.^{15,16}

The mean age of our patients were 45.28 years. Maru and Jain¹⁷ concluded that atherosclerotic diseases of labyrinthine vessels usually affects patients above 40 years of age. Raised serum total cholesterol, LDL cholesterol levels and hypertension are more contributory risk factors in its development. Similarly study of prevalence of metabolic disorders among patients with SSNHL in Brazil by Oiticica J and Bittar RSM¹⁸ also noted mean age of patients to be 46.5 years and 43.9% were males and 56.1% were females. Our study also shows that females are more affected than males (57% in females compared to 43% in males).

Our study revealed that there was significant difference between the means of lipid profile and uric acid level of the patients and control groups. As one of the risk factors of atherosclerosis, hypercholesterolemia contributes to vascular occlusion. Similar effects in cochlear microcirculation, like ischemia reperfusion injury

from hypercholesterolemia have been suspected. Marcussi et al⁶ concluded that hypercholesterolemia was an independent risk factor for SSNHL through his study on the relationship between SSNHL and risk factors for cardiovascular thrombosis. LU Yuan-yuan's research¹⁹ showed that total cholesterol, TG and lipoprotein An in SSNHL group were higher than the control group. On the other hand Axelsson and Lindgren²⁰ indicated an increased risk of acquiring high frequency SNHL for people who work in noisy environment and have high serum cholesterol level. Also, Sutbas et al²¹ in his study of effect of low cholesterol diet and anti-lipid therapy in managing tinnitus and hearing loss found incidence of hyperlipidaemia to be among patients with noise induced hearing loss. Suckfull et al²⁷ concluded that hyperfibrinogenemia and hypercholesterolemia may contribute to the clinical events of SNHL. Their study showed that acute and drastic removal of plasma fibrinogen and LDL is effective in the treatment of patients with SNHL. Mohindroo and Thakur JS et al²⁸ found that LDL were significantly associated with many waveforms in hyperlipidaemic patients, hence LDL may be important in auditory dysfunction.

Our study shows that the HDL level in SSNHL patients were lower than control group. HDL-C/H is a type of cholesterol carried by HDL and indirectly reflects HDL level. HDL eliminates extra cholesterol from blood and organs by counter transport thus slowing the process of atherosclerosis.²⁵ HDL can also reverse endothelial dysfunction, inhibit LDL oxidation, stimulate endothelial cell proliferation and prostacyclin generation.²⁶ Lowered HDL level diminishes these benefits and increases the risk of thrombosis.

On the contrary Ullrich, Aurbach and Drobik²² indicated that both hyperlipidaemia and atherogenic risk factors are not major pathological importance in SSNHL. Also, Kazmierczak H and Doroszewska G²³ concluded that disturbance of glucose metabolism and hyperinsulinemia may be

responsible for inner ear diseases, whereas role of disturbance of lipid metabolism remains vague. Likewise, Claudia Rudack et al²⁴ found correlation between HDL-CH and LDL-CH and SSNHL.

Our study shows higher blood uric acid level in SSNHL group compared to control group. Increased blood uric acid level may lead to UA deposit in vascular intima, resulting in local damage and inflammation that accelerates the formation of thrombosis.²⁹ But on contrary, some researchers insist that UA is the most important antioxidant in blood that can prevent cells from tyrosine residue nitration and prevent extracellular superoxide dismutase from degradation³⁰ thus executing protective effect on heart and cerebral vascular condition.

In their study Sutbas et al²¹ have cited that vascular mechanism is not solely responsible for auditory dysfunction. Lipoidosis of inner ear (uptake of cholesterol by lateral wall of outer hair cells shown in guinea pig) as studied by Nguyen and Brownell increases the stiffness of cells and impairs the electro motile response.³¹

Conclusion

Our study indicates a relationship between occurrence of SSNHL and presence of hyperlipidaemia and high serum uric acid level. But further studies are needed to establish the relationship.

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Conflicts of Interest: There are no conflicts of interest.

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