"LIPID PROFILE IN PATIENTS WITH SENSORI NEURAL HEARING LOSS"- A ONE YEAR HOSPITAL-BASED OBSERVATIONAL STUDY IN KLES Dr.PRABHAKAR KORE HOSPITAL.

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BELAGAVI, KARNATAKA

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KAHER

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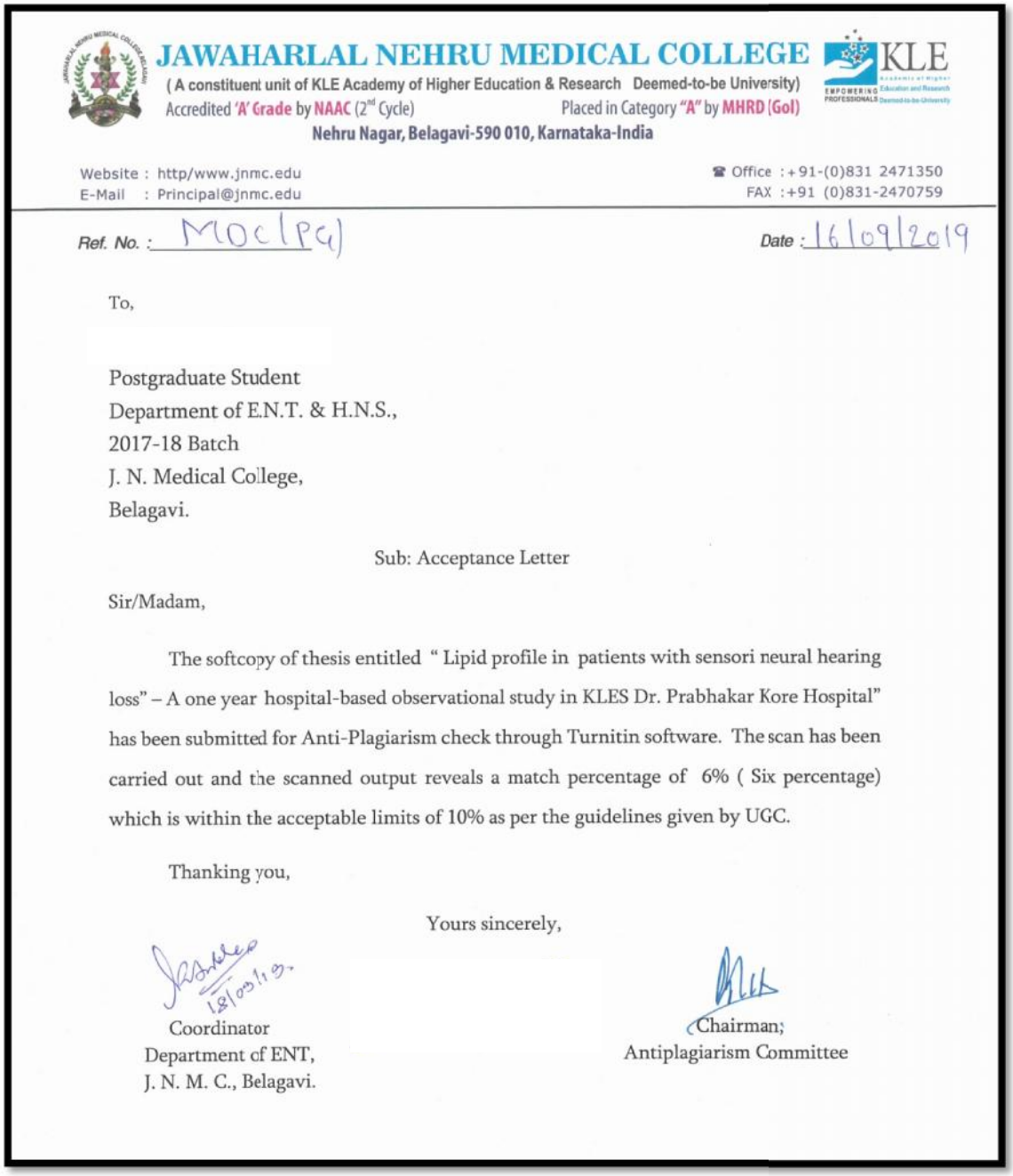

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Sir/Madam,

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ABBREVIATIONS USED

ABR	: Auditory brain stem response
dB	: Decibel, unit of sound intensity
et al	: *et alii* (Latin; 'and others')
HDL	: High density lipoprotein
IDL	: Intermediate density lipoprotein
i.e	: *id est* (Latin; 'that is')
KHz	: Kilo hertz, unit of frequency
LCAT	: Lecithin acyl cholesterol transferase
LDL	: Low density lipoprotein
LPL	: Lipoprotein lipase
NIHL	: Noise induced hearing loss
HLOSS	: Hearing loss.
PTA	: Pure tone audiometry
SNHL	: Sensorineural hearing loss
T.C	: Total Cholesterol
TRIGLYDS	: Triglycerides
VLDL	: Very low density lipoprotein
WHO	: World Health Organisation

ABSTRACT

Background:

The negative influence of hypercholesterolemia on the blood vessels condition and following degeneration lesions of the organs is well known. There have been several studies which previously established a relation betwixt raised lipid levels and hearing disorders, but till date the theory remains questionable.

Objective:

To study association among various degrees of sensorineural hearing loss and serum levels of total cholesterol, triglycerides, HDL (high density lipoprotein) and LDL (low density lipoprotein)

Methods:

In all, 58 cases of sensorineural hearing loss during the study period were selected and investigated by pure tone audiometry for assessing the hearing loss and their blood investigated for lipid levels. Sensorineural hearing loss was classified as mild, moderate, moderately severe, severe and profound based on the pure tone average of the worse hearing ear.

Result:

Serum levels of High Density Cholesterol and Triglycerides were having a negative correlation with hearing loss (-1.46 and -1.52). Their p values were 0.1488 and 0.1329. Similarly, the Low Density Cholesterol and Total Cholesterol levels had a minimal positive correlation with the hearing loss (0.99 and 0.46), and their p values are 0.3260 and 0.6435 each.

Conclusion:

The serum levels of low density lipoprotein directly correlate with the severity of sensorineural hearing loss whereas the serum levels of high density lipoprotein negatively correlate with the severity of sensorineural hearing loss in both sexes, but not significantly.

Key words:

Sensorineural hearing loss, Serum lipids, Total Cholesterol, Triglycerides, Low density lipoproteins, high density lipoproteins.

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INTRODUCTION

Hearing loss is a frequent observation in the general population with many etiologies. The higher incidence of hearing loss among patients with hyperproteinemia has long been established and is constantly being verified by new studies.

The normal physiological mechanisms of the human body face new challenges everyday due to the modern life style. There is an adverse impact of the modern, sedentary lifestyle which we lead. Along with that, an excessive body weight and a diet rich in total fatty acids and saturated fats has adverse effects. This, with a genetic background, confers a susceptibility to increased circulating lipids. The negative influence of hypercholesterolemia on the blood vessels' condition and following degenerative lesions of the organs is well known.[1]

There are different mechanisms by which hyperlipidemia leads to SNHL and these include atherosclerosis, lipidosis, hypoxia, microvascular disease, embolic episode, metabolic conditions, hyper viscosity, hereditary, hypertension and also ageing.[2]

In recent years, cochlear ischemia has been hypothesized in patients of sensorineural hearing loss, but excluding such patients who were affected by an infectious episode or acoustic neuroma.[3] There have been several studies which previously demonstrated a relation between rise in serum lipids and hearing disorders, but till date the point remains controversial.

Estimation of serum lipids and their correlation with multiple variants and levels of SNHL may provide an insight into this relationship. Suzuki K et al[3] in their study on male population found that hearing levels at 2000Hz and 4000Hz for group with higher HDL cholesterol values were characteristically better than those in lower HDL cholesterol groups.

This study was designed to emphasize the need for early detection of hyperlipidemia in SNHL patients who were otherwise not under the care of physicians. And in doing so, a metabolic abnormality which apparently leads to high risks for coronary artery disease, hearing loss, vestibular dysfunction and other illnesses may be better controlled.

OBJECTIVE OF THE STUDY

To study the association between degree of sensorineural hearing loss and serum levels of total cholesterol, triglycerides, HDL (high density lipoprotein) and LDL (low density lipoprotein)

REVIEW OF LITERATURE

Anil.H.T and Shazia[4] in 2016 conducted a prospective comparative study on 100 adults aged 30-60 years, with SNHL and various degree of hearing loss was compared with serum lipids levels. The study shows significant relationship between serum lipid and various degrees of sensori neural hearing loss. Increase in total cholesterol, triglycerides and LDL, were associated with worsening of hearing levels.

According to the study conducted by Ali A. Muttalib Mohammed[1] in 2013, it was concluded that hyperlipidemia had a significantly strong association with the occurrence of sudden SNHL.

A study conducted by Roshan R Jalisatgi[2] in 2009 in B M PATIL Medical college and Hospital, Bijapur on 64 patients with SNHL showed that serum levels of LDL directly correlate with the severity of Sensori neural hearing loss whereas HDL levels correlate negatively.

Sutbas A et al[5] in 2007 investigated the role of a low cholesterol diet and antihyperlipidemic therapy to alleviate the severity of tinnitus and also possibly promote hearing gain after therapy in patients with acoustic trauma. The study consisted of 42 hyperlipidemic patients with subjective tinnitus and noise induced hearing loss, who was put on a low cholesterol diet or antihyperlipidemic therapy. These patients were followed for 24 months, and assigned fewer than two groups as “responsive” and “unresponsive” respectively.

Significant results were obtained when self rated tinnitus severity results and the difference between average air conduction thresholds at high frequencies after the treatment in the two age groups were compared, and were found to be significant.

They concluded that lowered tinnitus intensity and higher frequencies in average hearing thresholds can be achieved after lowering the serum lipid level.

According to a study conducted by Martin Villares C et al[6] in 2005, on 180 patients of more than 65yr of age with bilateral hearing loss, 71% of patients had hypercholesterolemia that had worse hearing loss than patients with normal serum lipid levels.

Namyslowski G et al[7] in 2003 conducted a study to estimate the influence of lipid balance disorders on small blood vessels of the brain and inner ear in the patients with hypercholesterolemia and hypertriglyceridemia on the basis of audiometric, transient-evoked otoacoustic emissions TEOAE and ABR evaluations. They had observed no statistically significant correlation between cholesterol serum levels and amplitudes of TEOAE.

Erdem T et al[8] in 2003 conducted a prospective study on the existence of subclinical auditory dysfunction related to Hyperlipoproteinemia and Diabetes Mellitus by transient-evoked (TEOAEs) and distortion-product otoacoustic emissions (DPOAEs) in patients with hearing levels better than 30 dB compared with the control group. The mean DPOAE amplitudes of the hypertriglyceridemic and Diabetes Mellitus groups at 4 kHz were higher than those of the control group. They concluded that an otoacoustic emission helps in early prediction of the prospective effects of Hyperlipoproteinemia and Diabetes Mellitus on the auditory system.

Karlidağ T et al[9] in 2002 evaluated the effects of hyperlipidemia on hearing function. The study included 274 hyperlipidemic patients with sixty healthy subjects and their hearing thresholds were analyzed using pure-tone audiometry.

Marked increase in hearing levels were observed in both sexes at 8000 Hz in 3 lipid groups, at 6000 Hz in both sexes in the VLDL group. But at 2000 Hz, the increase was found in the males of total cholesterol and VLDL groups. When evaluation was done by including both male and female patients together, the biggest difference was identified in the VLDL group at 5 different frequencies. They concluded that a raise in lipid levels could have a significant role in leading to SNHL.

Satar B et al[10] in 2001 undertook a study to determine the holdings of raised cholesterol levels on the cochlea of guinea pigs. It was also done to find out the root of pathological changes. 20 guinea pigs on a normal diet were compared with 24 animals on cholesterol diet. The hearing acuity of the animals was compared to values before and to values after the diets with the help of an auditory brainstem response. The cochlea was examined in detail under light microscopy first and later under transmission electron microscopy.

It was found that the cholesterol group showed severe edema within strial marginal layer. Also, the outer hair cells demonstrated a slightly lesser edema. This was found to be in terms with evidence obtained from auditory responses of brainstem. It revealed modifications in various degrees of hearing sensitivity. Hence concluded that hypercholesterolemia is the lone cause for auditory dysfunction whenever dietary cholesterol for these guinea pigs was maintained at a raised level for a comparatively longer time.

Kojima Y et al[11] in 2001 conducted a study on 12 patients who had unilateral sudden hearing loss and hyperlipidemia. Also included were patients who had greater than 1 month of hearing loss. Evaluation is done by calculating air conduction thresholds of the patients at 125 to 8,000 Hz with help of pure tone audiometry. A

therapy for raised lipid levels was given. Also, dietary changes were incorporated along with giving antilipemic agents. The hearing levels were measured both before and after therapy.

It was found that, the mean hearing levels had significantly improved at each of the frequencies 125, 250, 500, and 2,000 Hz after administration of therapy. The results suggested that with a proper treatment for hyperlipidemia, there is a tendency for the hearing levels to improve in all patients having chronic-phase sudden deafness and related raise in lipid levels.

Suzuki K et al[3] in 2000 undertook a study to estimate influence of serum lipid levels on auditory function. They also researched the relation among hearing levels of better-hearing ear with serum concentrations of 1-total cholesterol, 2-triglyceride, and 3-high-density lipoprotein cholesterol. They concluded that a low level of HDL cholesterol is related to hearing loss and arteriopathy may exhibit a role in such auditory dysfunctions.

Jones NS et al[12] in 2000 tested a null hypothesis that the distribution of blood lipid levels is the same between a SNHL group and control population. This study led to the rejection of the proposed Null hypothesis, as hearing thresholds were found to be significantly better in those with raised cholesterol levels.

Jones NS et al[13] in 1999 did a prospective case-controlled study on 85 patients who presented consecutively with a sensorineural hearing loss of unknown etiology to observe if there was a prevalence of hyperlipidaemia between the group with an idiopathic sensorineural hearing loss and a control population and whether any difference existed. No consistent association was found.

Lee FS et al[14] in 1998 did a study along with another ongoing study of presbyacusis. They investigated relationship between different serum bio-chemical levels and hearing thresholds. They found that hearing levels of female patients with raised LDL/HDL ratios were better with 5 dB difference, as compared to those with lower LDL/HDL ratios. This suggested that blood chemical measures that are principally within normal bounds had very lower prediction value for pure-tone thresholds among older patients.

Ohhira S et al[15] in 1998 in their study to examine the influence of hyperlipidemia and smoking on age-related changes in caloric response and pure-tone hearing found a significant difference in slow phase velocity of the caloric nystagmus only, but not in the hearing level. This suggests that age-related changes in the caloric response may be promoted by atherosclerosis, unlike presbyacusis.

ANATOMY OF INNER EAR

Temporal bone forms a significant portion of base of skull extending from lateral calvaria at the level of external auditory canal to almost the centre of the skull as it articulates with basi-sphenoid. Cranial nerves V to XII course about or through the temporal bone; the middle cranial fossa contents lie on its superior surface and the posterior cranial fossa contents lie on its posterior surface. The internal carotid artery travels through it, and the internal jugular vein originates within the temporal bone through the jugular foramina.

The temporal bone consists of four separate bones:

- Petrous
- Tympanic
- Squamous
- Styloid process.

The inner ear or the labyrinth lies inside the petrous part of the temporal bone. The auditory and vestibular components of the inner ear are known as the labyrinth because of its serpinginous and convoluted course. Labyrinth is further divided into 1-a bony part, 2-a membranous part. The membranous part contains 1-sensory epithelium of cochlea and 2-all vestibular structures. It be found within the cavities enclosed by bony part.

Bony labyrinth is a derivation from inner periosteal layer belonging to otic capsule. Later, in adult life, it subsists of a thin and dense bony shell enclosing all these structures i.e vestibule, cochlea and semicircular canals.

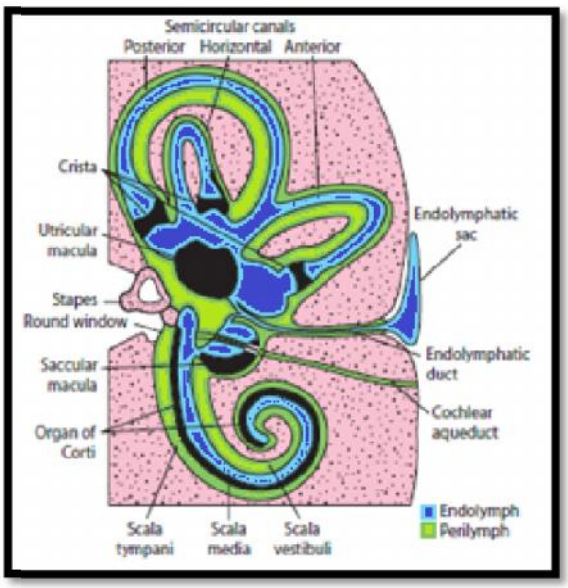


Figure 1: Diagrammatic representation of the human inner ear which shows various compartments.

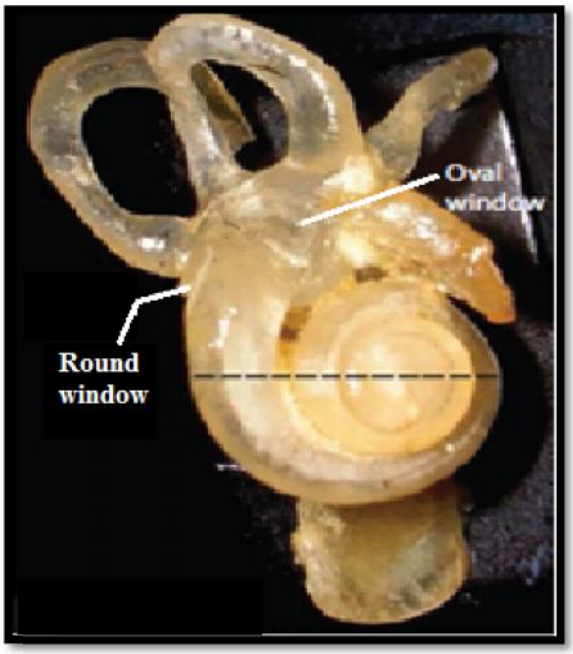


Figure 2: Human inner ear- a plastic model of the membranous labyrinth. Scale bar= 5mm. the dotted line shows plane of section shown in next diagram.

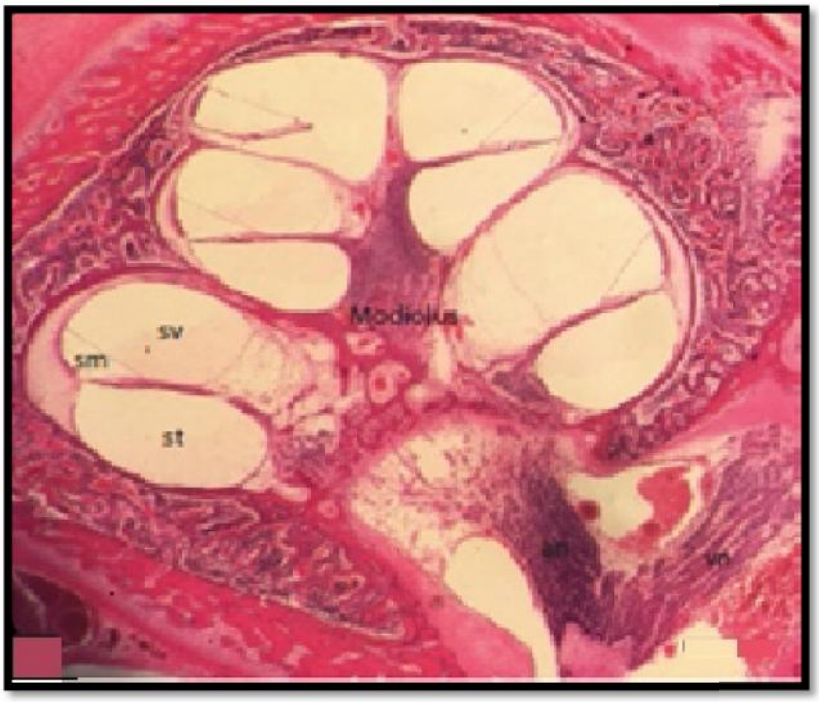


Figure 3: Section through the human temoral bone at approximately the mid modiolar level of the cochlea. sm= st= scala media; sv= scala vestibule; an= auditory nerve; vn= vestibular nerve. Scale bar= 1.5mm.

THE COCHLEA

The bony cochlea appears externally like the shell of a snail and lies in front of the vestibule. It is a coiled tube, with the inner lumens being separated by a dense, thin bony wall. The shell has approximately two and a half turns. The cochlear coils turn about the modiolus. It is a central cone arising from the portion of the cochlear nerve. It is directed forwards and laterally. It tapers from a broad base to the slender apex. Basal coil forms bulge of the promontory below the apex. It faces forwards and laterally pointing to upper part of tympanic cavity's medial wall. A thin shelf of bone arises from the modiolus, and winds up spirally inside the lumen of cochlea. This is called "the bony spiral lamina".

Coming to membranous part of spiral lamina, it continues from the margin of bony spiral lamina till extrinsic wall of cochlea. It divides every coil into 2 major proportions, 1- upper scala vestibule, 2- below it, the scala tympani.

The Helicotrema forms a communication in the perilymph spaces at apex of cochlea where scala vestibuli joins scala tympani. This part of the otic capsule overlying this apical region is known as cupola of the cochlea. At this point the interscalar septum makes an abrupt vertical turn to end in the enchondral bone of the overlying cupola. This abrupt vertical turn of the interscalar septum makes a 900 angle with the hamulus of the osseous spiral lamina.

The hamulus circumscribes a semicircle, where its inner edge is immediately adjacent to the helicotrema and outer free edge attaches to basilar membrane. As the spiral ligament and basilar membrane reaches the end of hamulus they continue to complete at the apex ultimately ending on the oblique and vertical portion of the interscalar septum.

This then creates an oblique channel known as helicotrema. CSF may mix with the perilymph through two routes; the cochlear aqueduct (direct) and the modiolus (serpinginous), beginning in the fundus of internal auditory meatus and ultimately entering the organ of corti.

The scala vestibuli opens into the vestibule at the base of the cochlea as fenestra vestibule, and stapes foot plate closes lateral wall of vestibule. Resembling a blind end tube, the scala tympani have at its base a fenestra cochlea i.e., round window. It is covered with a "secondary tympanic membrane". Also, one small opening emerges from basal edge of scala tympani to the cochlear aqueduct.

The modiolus is made up of many tiny canals. They spread out and enter bony spiral lamina. Those canals present on outer extremes, relate fibers from basal portions of cochlea. Those canals which are central carry fibers to and from the apical regions. Such canals have a dilatation so as to hold "the bipolar ganglion cells", a portion of the spiral ganglion. The assemblage of all dilated spaces has a name "spiral canal of the modiolus".

Cochlear duct (Scala media):

The cochlear duct is made up of spirally aligned tube. It lies over the superior surface of "spiral lamina". It also lies across external wall of bony canal in cochlea. It has an average length of 34mm and width approximately 80mm. It is a wedge shaped compartment lying between the scala tympani and the scala vestibuli. The vestibular (Reissner's) membrane separates it from the scala vestibuli and basilar membrane from the scala tympani. The external part of "bony spiral lamina" along with membranous lamina forms the floor.

The floor of the cochlear duct:

The bony spiral lamina forms the inner part of the floor, which divides into two ridges horizontally. The ridge above is called spiral limbus. The tectorial membrane emerges from this ridge. The ridge below gives rise to membranous part of spiral lamina. This ridge contains acoustic nerve fibers running through and reaching "the organ of corti".

On outer side of the membranous part of spiral lamina, it has a "flattened epithelium of scala tympani", the middle layer is fibrous and the organ of corti is present on its upper surface. The inner and outer sulcus separates it from the spiral limbus the lateral wall respectively.

The organ of corti is ridge like framework. It contains auditory sensory cells with a complex array of other supporting cells. They are arranged in 2 distinct groups; 1-inner hair cells, 2-outer hair cells. The inner hair cells are present in a single row, but occasionally extra hair cells may be present. The outer hair cells are present in three, four or five irregular rows.

These cells are so named as the free surface of each hair cell is covered with bristle like clumps of hair.

Every hair cell comprises a body, facing inward to organ of corti; the *cuticular plate*, and a thickened outer surface from which arises a cluster of hair or stereocilia. These stereocilia resemble microvilli containing an inner core of molecules of actin.

As the length of duct goes, height of the longest present stereocilia progressively increases in an ascending fashion with distance from the base. The

number of stereocilia also decreases in the passage from the base to the apex, forasmuch as the length increases, but not in a linear fashion.

The fetus and newborn contain about 3,500 inner hair cells and about 13,000 outer hair cells. Total sum of hair cells shows variation with length of cochlea. The shorter cochleae have few inner cells and outer cells. It is observed from cytocochleogram that as the age of a person advances, a generalised reduction in hair cells is observed. These variations are more pronounced in outer hair cell population.

The pillar cells, Dieters's cells and Hensen's cells are a set of specialized and highly differentiated cells which support the hair cells.The tectorial membrane attached to spiral limbus acts like an acellular gel matrix. It comprises of fibrillar strands. It is acutely sensitive to distortions and shrinkage.

The lateral wall - cochlear duct:

It includes 3 distinct zones, from above downwards:

- Stria vascularis.
- Spiral prominence
- A transitional zone in between.

The bulk of lateral wall is formed by stria vascularis and consists of three cell layers: the basal cell layer, the marginal cells and the intermediate cells. Marginal cells affront the endolymph. In between are the intermediate cells arising from layer of basal cell.

The roof - cochlear duct (i.e. "Reissner's membrane"):

"Reissner's membrane" / the floor, is a fine sheath. It extends from bony part of lamina to tip of lateral wall. The outer surface of endolymphatic sac is made up of classic squamous epithelial cells containing microvilli. They are joined to each other by tight cellular junctions. There is fine basement membrane splitting these cells from the ones present in upper part of scala vestibuli.

Such cells lining scala media are unied together by tight intercellular junctions. These productively separate the endolymph and the perilymph. It helps in maintaining usual ionic contents of this endolymph.

Blood Supply:

The organ of corti and other structures of cochlear duct get their blood supply by vessels within the stria vascularis and also the spiral vessels underlying the basilar membrane and the spiral limbus. The main cochlear artery enters through the modiolus along with the VIII nerve fibers. Arterioles divide at the level of spiral lamina, where one group of vessels proceeds to a position underlying the basilar membrane. The second arteriolar system travels across the wall of scala vestibuli within the periosteal lining to the region of spiral ligament. At this point the arterioles break up to form three capillary networks along the lateral wall of peri-otic labyrinth.

The first group of vessels supplies the region of spiral ligament just above the insertion of Reissner's membrane. The second group of vessels forms a highly anastomosed capillary bed of stria vascularis and the third set supplies the vessels of spiral prominence.

The vein of the cochlear and vestibular aqueducts drains the inner ear. The cochlea is primarily drained by anterior and posterior spiral veins. The anterior spiral vein drains the more supero-lateral, or more anterior cochlea such as scala vestibuli and osseous spiral lamina. The posterior vein drains the infero-medial aspect of cochlea; such as spiral ganglion, scala media and scala tympani. These veins enter a common modiolar vein which enters the cochlear aqueduct, and forming a tributary to inferior petrosal sinus.

It is to be noted that neither the organ of corti nor the corti lymphatic space has its own blood supply. The neuroepithelium of organ of corti thus receives the oxygen and nutrients indirectly from either the vessels of lateral wall of the cochlear duct or the spiral vessels underlying the basilar membrane.

Innervation:

The organ of corti is innervated by two types of nerve fibres. Efferent fibres of the olivocochlear bundles conduct nerve impulse from the brain to hair cells. Afferent fibres of auditory portion of VIII cranial nerve conduct nerve impulse from the hair cells to brain. [16,17]

APPLIED PHYSIOLOGY:

In man hearing becomes the vital basis for acquisition of speech and language. The mechanism of hearing can be broadly divided into:

- Mechanical conduction of sound.
- Transduction of mechanical energy into electrical impulses (by sensory system of cochlea)
- Conduction of electrical impulses to the cerebral cortex (neural pathways).

Physiology of sound conduction:

The pinna assembles a sound signal coming from surrounding environment. It traverses from external auditory canal, goes on to strike the tympanic membrane. The tympanic membrane vibrations are transmitted through the ossicular chain to the cochlea. Then, Impedance matching mechanism of middle ear comes to action:

When sound through air conduction travels to cochlear fluids, sound energy loss that occurs is compensated by impedance matching or transformer action of the middle ear, which is accomplished by;

- Ossicular-level chain ratio is 1.3:1. It is between handle of malleus, long process of incus.
- The ratio of “area of tympanic membrane” and “the oval window”. Effective area ratio is 14:1.
- Phase differential between oval and round window.

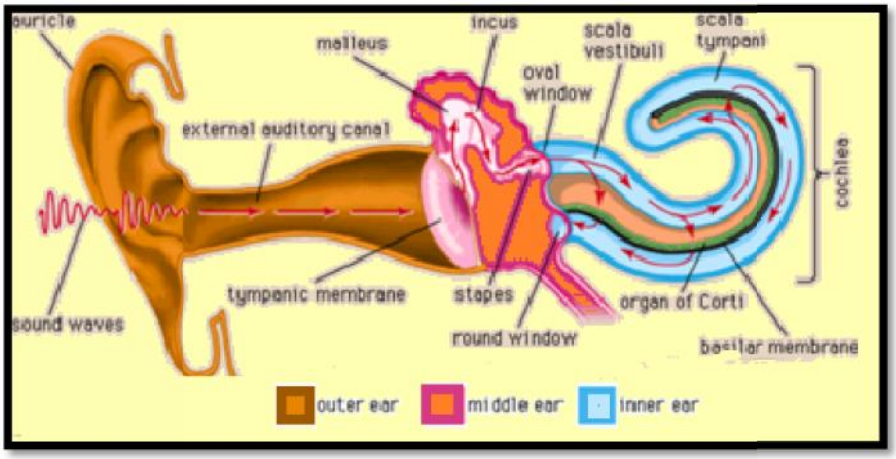


Figure 4: Schematic flow of sound energy

Inner ear:

The two essential physiological processes taking place in Cochlea are transmission and transduction. Transmission accounts for "the transfer of acoustic energy from the oval window to the hair cells". The transduction is "a process by which this transmitted sound energy pattern is converted into action potentials". It takes place at the level of organ of Corti, present in the auditory nerve.

Vibrations of the stapes produce a flow of perilymph. It goes up the scala vestibuli, through the helicotrema and then down the scala tympani to the round window membrane. With vibrations, movement of basilar membrane occurs, which sets up a shearing force between the tectorial membrane and hair cells.

The basal turn of cochlea represents higher frequencies and towards the apex lower frequencies are represented. The distortion produced in hair cells gives rise to cochlear micro phonics. It triggers the neural impulses. Auditory nerve action potential is "the algebraic sum of the neural discharges in the whole of the cochlear nerve". Every single nerve fiber has an optimum stimulus frequency, corresponding for which threshold levels are lowest.

Transduction by hair cells:

Each individual stereo-cilia present on apical surface of hair cells are "mechanically rigid". They have cross links between each other and hence movement is there as one unit- i.e. stiff bundle. Thence, whenever a bundle moves, "different rows of stereocilia could be expected to glide in relation to each other".

There are delicate attachments from the tips of shorter stereocilia running high on hair cells. These join neighboring, taller stereocilia falling on adjacent row. When

such stereocilia are deflected along direction of tallest stereocilia, all those links get stretched, hence opening ion channels within the cell membrane.

When the stereocilia are deflected in the opposite direction, the tension is taken off the links and the channels close. This hypothesis is consistent with the present electro-physiological evidence from hair cells.

When ion channels on stereocilia open, such ions move inside or out. This depends on electrical charge and the chemical gradients maintained. It is hypothesized that these ion channels are huge and non-selective; so for example, Na^+, K^+ and Ca^{++} all enter with nearly same potency.

Under the generally accepted position, the apical surface of the hair cells is facing towards the endolymph. They possess a greater positive potential (±80mV) and K^+ ion concentration is also higher. Within the cell, we find a negative intracellular potential. It is -70mV for outer hair cells, –45mV for inner hair cells. These potentials later combine and give 125mV for inner cells and 150mV for outer cells of potential drop cross the gradient.

When ion channels open, this big potential gradient drives K^+ from the endolymph into the cell, thus making cells more positive on the inner side. The cells will become more negative when the channels are completely shut off. K^+ ion carries most of transducer current. Sometimes it is also taken by Ca^{++}.

The whole process gets its energy from stria vascularis. It stores energy within the ‘battery’ of endolymph, with the help of ion pumping. This is termed as “the ‘battery’ theory or ‘resistance modulation’ theory of Davis (1965)”.

Neural pathways:

The Hair cells get their nerve supply from bipolar cells existing in the spiral ganglion. A cochlear nerve is formed by joining the central axons of these cells. This nerve relates to central and dorsal cochlear nuclei. "Further, both crossed fibres of the nerve and uncrossed fibers travel to the superior olivary complex, lateral leminiscus, inferior colliculus and medial geniculate body and culminate in the auditory cortex" of the temporal lobe.[16,17]

THEORIES OF HEARING:

1. **Travelling wave theory of Von Bekesy:-**

According to this theory, a sound wave reaches maximum amplitude on a particular place on the basilar membrane, depending on its frequency and stimulates that segment. If the basilar membrane is observed for vibrations, a travelling wave is seen to start at the base of the cochlea and progress towards the helicotrema. There is an increase in amplitude to a region of maximum displacement, the position of which depends over the frequency. Basal turn of cochlea presents for higher frequencies and progresses towards the apex for lower tones.

2. **Helmholtz's resonance place theory:-**

This theory suggested that the frequency analysis by the ear was due to the fact that each pitch would cause resonant vibration of its own at a particular place on the basilar membrane. But later it was disproved as basilar membrane cannot act as a resonator.

3. **Rutherford's telephone theory:**

This theory suggests that "pitch perception is based on the rate of firing of individual nerve fibers".

4. **Wever's volley theory:-**

This theory postulates that high frequencies are perceived by a place alone in the basal turn of cochlea, and low frequencies (below 1000) stimulate nerve action potentials at a rate equal to the stimulus frequency. But the intermediate frequencies are presented in the auditory nerve by asynchronous discharge in groups of neurons whose combined activity represents the frequency of the stimulus.

TRANSPORT OF LIPIDS AND LIPOPROTEIN MOLECULES

THE STRUCTURE OF LIPOPROTEINS:

Lipoproteins are spherical fragments. They are made of many lipids and protein molecules. Lipoproteins are small in size visible only through electron microscopy. Cholesterol, triglycerides and phospholipids are the major lipids found in lipoproteins. The core of the lipoproteins is formed by triglycerides and esterified form of cholesterol also called as cholesteryl esters. These are nonpolar-lipids and hence are not soluble in aqueousmediums.

The surface of the particles is covered by 1-phospholipids, 2- small quantity of free/ unesterified cholesterol. They are amphipathic, i.e. soluble in both lipid medium and aqueous environments. These act as an interface amidst plasma and core components.

Also, the surface of lipoproteins is occupied by apolipoproteins, a family of lipoproteins which play a crucial role in the regulation of lipid transport in the body and lipoprotein metabolism.

The classification of lipoproteins, based on their densities is done into 5 major classes:

- Chylomicrons
- Very low density lipoproteins (VLDL)
- Intermediate density lipoproteins (IDL)
- Low density lipoproteins (LDL)
- High density lipoproteins (HDL).

APOLIPOPROTEINS:

The apolipoproteins (apos) provide structural stability to the lipoproteins and determine the metabolic fate of the particles upon which they reside. They are found in two forms - apo B 100 and apo B 48. “B100 is the major apolipoprotein in VLDL, IDL and LDL, comprising about 30, 60 and 95% of the protein in these lipoproteins, respectively”. Apo B48 is essential for the assembly and secretion of chylomicrons.

The apolipoproteins belonging to C series are manufactured in our liver. These apos are a part of all plasma lipoproteins and trace amounts found in LDL. Each individual apo C has a different metabolic role. Overall in general, apo Cs suppress the removal of 1-plasma chylomicrons 2-VLDL remnants by the process in liver. Apo CII deficiency in individuals leads to severe hypertriglyceridemia.

Apo E is synthesized mainly in hepatocytes, and is found to be present in 1-chylomicrons, 2-IDL, 3-VLDL and 4-HDL. It deals with uptake of such lipoproteins in our liver. When there is total absence of apo E, the plasma concentration increases

of chylomicrons and all VLDL remnants. Thence it causes early stage of atherosclerosis.

OTHERS:

The role of VLDL in atherogenesis is not much studied. The major reason is that "an inverse relationship between the elevated levels of triglyceride-rich lipoprotein and reduced levels of the antiatherogenic HDL cholesterol" exists. The risk of getting atherosclerosis from raised triglyceride levels and raised levels of VLDL could be determined by knowing levels of cholesteryl ester-enriched remnants.of VLDL [18]

LIPOPROTEIN CLASSIFICATION

Lipoproteins are substances formed by the combination of proteins with hydrophobic lipids, in order to transport them to various organs.These lipoproteins contain a core of hydrophobic lipids (Triglycerides and cholesteryl esters) surrounded by hydrophilic lipids (phospholipids; unesterified cholesterol) and proteins that interact with body fluids.

The plasma lipoproteins are divided into five major classes based on their relative densities.

Lipoprotein	Density g/ml	Size nm	Electrophoretic mobility	Major apo lipoproteins	Other Apolipoproteins
Chylomicrons	0.93	75-1200	Origin	Apo B-48	A-1,A-IV,C- I,C-II, C-III
Chylomicrons	0.930-1.006	30-80	Slow pre-β	Apo B-48	E,A-I,A-IV,C-I, CII,C-III
VLDL	0.930-1.006	30-80	Pre- β	Apo B-100	E,A-I,A-II,A-V,C-I, C-II,C-III
IDL	1.006-1.019	25-35	Slow pre-β	Apo B-100	E,C-I,C-II,C-III
LDL	1.019-1.063	18-25	β	Apo B-100	
HDL	1.063-1.210	5-12	Alpha	Apo A-I	A-II,A-IV, E,C-III

Table- 1: Lipoprotein Classes[18]

Sl No:	Apolipoproteins	Molecular mass, Da	Lipoproteins	Metabolic functions
1.	Apo AI	28,016	HDL, Chylomicrons	Structural component of HDL; LCAT activator.
2.	Apo AII	17,414	HDL, Chylomicrons	unknown
3.	Apo AIV	46,465	HDL, Chylomicrons	Unknown: Possibly facilitates transfer of other apos between HDL and Chylomicrons.
4.	Apo B48	264,000	Chylomicrons	Necessary for assembly and secretion of chylomicrons from the small intestine.
5.	Apo B100	540,000	VLDL, IDL, LDL.	Necessary for assembly and secretion of VLDL from the liver; structural protein of VLDL, IDL, LDL; Ligand for LDL receptor.
6.	Apo CI	6630	Chylomicrons, VLDL, IDL, HDL	May inhibit hepatic uptake of chylomicrons and VLDL remnants.
7.	Apo CII	8900	Chylomicrons, VLDL, IDL, HDL	Activator of lipoprotein lipase.
8.	Apo CIII	8800	Chylomicrons, VLDL, IDL, HDL	Inhibitor of lipoprotein lipase; May inhibit hepatic uptake of chylomicrons and VLDL remnants.
9.	Apo E	34,145	Chylomicrons, VLDL, IDL, HDL	Ligand for binding of several lipoproteins to the LDL receptor, to LRP and possibly to a separate hepatic apo E receptor.

Table- 2: Characteristics of the major Apolipoproteins[18]

NOTE: "HDL=high-density lipoprotein; LCAT= Lecithin: cholesterol acyltransferace; VLDL=Very low density lipoprotein; IDL=Intermediate-density lipoprotein; LDL=Low-density lipoprotein; LRP=LDL receptor-related protein".

ULTRASTRUCTURAL CHANGES IN COCHLEA DUE TO HYPERCHOLESTEROLEMIA

The cochlea receives its blood supply majorly from one end artery. It is contemplated to have high sensitivity to even small pathological changes in the vessels. It is widely known fact that the raised cholesterol levels cause arteriosclerotic changes inside blood vessels. This leads to a partial obstruction in the vessel and then, end-organ hypoxia. Further, theories have been proposed that such arterio-sclerotic changes occurring in cochlear vessels can lead to some loss of hearing[19].

Oxidative LDL is another entity believed to play an important role in process associated with formation of atherosclerosis in vessels. One recent hypothesis explained that once can find development of fatty streaks filled with foam cells inside the arterial walls. These streaks are believed to set-up atherosclerosis. The study suggested that "oxidative modification of LDL in the arterial wall is critical to the process"[20].

"Uptake of oxidized LDL via recognition of the modified apolipoprotein B at the macrophage scavenger receptor leads to the formation of lipid laden foam cells in vitro". These cells resemble those present in fatty streaks which are the precursors of atheroma[21]. The foam cells secrete 1- monocyte-chemotactic protein-1 and 2-macrophage colony stimulating factor. This can further lead to recruitment and with-holding of lipid-laden macrophage cells. Later on, this aggregates to form what is

known as the fatty streak[22]. A study shows that HDL blocks oxidative modification of Low Density Lipoproteins in vitro[23], as well as blocks it in vivo[24].

The stria contains capillaries which are very thickly filled with red blood corpuscles. Hence the hematocrit value in such blood vessels become much raised as compared to capillary beds present in different parts. As a result, due to such high levels of hematocrit, the viscosity of the blood tends to be proportionately raised. Cinegraphic studies have proved that blood corpuscles move at lower speed through these strial capillaries in comparison with any of the vessels belonging to spiral ligament or the spiral prominence[25]. The greater the "high shear blood viscosity", the lesser will be the sensorineural thresholds.

If red blood cells were less elastic than normal, it could lead to poorer flow through the nutrient capillaries of the stria vascularis. Hence, ischemia of the inner and outer hair cells sets in with loss of sensitivity and resolving power[26]. It is due to this special character of blood flow which is seen through strial capillary bed, that any condition which has an influence on blood viscosity or over the physical properties of red blood corpuscles is likely to even affect efficacy of oxygen supply and nutrient delivery to strial tissue.

Hildesheimer et al[27] in 1982 reflected that "a reduction in peripheral circulation, secondary to increased blood viscosity was the cause of decreased cochlear action potentials in their work with guinea pigs".

Another theory says that free cholesterol in our blood is exchangeable in vivo. It takes place between erythrocyte membrane and the serum lipoprotein[28]. Thus, whenever serum levels of cholesterol are raised, red blood corpuscles imbibe increased volumes of cholesterol in their membranes. This increases the size of red

blood corpuscles and even alters their shape[29]. Furthermore, agglomeration of cholesterol in the red corpuscular membrane affects activities of all membrane-bound enzymes[30], which include Na- K ATPase. Erythrocytes are dependent on these for functioning normally.

Hypercholesterolemia-induced decline in Na-K ATPase activity leads to changes in cell membrane permeability. It cuts down rate of oxygen shift from cells[31]. Cholesterol absorption in the red corpuscles also increases viscosity of its cell membranes, along with a subsequent diminution in their deformability[32]. When the deformability of red cells is reduced in association with small vessel disease, it could be the cause of raised threshold levels. Such changes influence the rheological properties of blood. They make it harder for red blood corpuscles to travel via the microvasculature. Consequently, the inner ear cells show altered metabolic activity in view of high metabolic requirements due to the presence of hypercholesterolemia and decreased oxygen availability.

Another analogous explanation could be given, i.e. in hypercholesterolemia, platelets get sensitized to epinephrine, leading to thrombosis.[33] A study done by Tami et al[34] concluded no change was observed in hearing acuity of experimental animals who were given a pure cholesterol rich diet for 3 consecutive weeks. But still the study personnel believed that if it all hearing loss occurs; it may depend on any vascular disease arising due to long-term side effects of raised cholesterol levels.

On the contrary, Pillsbury[35] in his study proposed his views that one cannot find reduction in cochlear flow of blood among Wistar rats who were receiving an atherogenic type of diet. But, he also accepted that raised cholesterol levels had a

tendency to precipitate noise-induced hearing loss. He mentioned that such an effect most probably depends on the vascular changes.

Another mechanism which explains some amount of loss of hearing is the glycogen aggregation which takes place in stria vascularis and all outer hair cells. This could be due to variation in blood supply of cochlea and strial transportation. The above said mechanism also inclines on alterations in blood viscosity in raised cholesterol levels.[36]

A general observation is that intercellular edema can be observed in "strial marginal layer" and the outer hair cells exhibit intracellular edema. It could be a good reason for auditory defunction. The changes occurring in the blood vessel wall and a decline in the blood flow are most likely to be the reason for edema in the stria vascularis. This is akin to arteriosclerosis observed in coronary arteries[11].

An identical observation made by Sikora et al[37] who studied aortas of dead chinchillas who were fed with a diet rich in cholesterol. The corresponding edema which developed in outer cells could be the result of a response to metabolic stress and/or malnourishment.

The earliest response of a cell or tissue exposure to stress is edema. It is also the earliest sign of tissue damage or cellular damage. A well known fact is our body directs more amount of glucose to glycogenesis pathway whenever there is abundant of cholesterol. As proposed by Gratton et al[36], there could be a possibility that glycogen could accumulate in some of the motile cells. However, theories have it that glycogen should actually accumulated in liver attributing to its activity in the pathway of glycogen metabolism. This is opposite to what they have found out in study.

Keeping all these things in consideration, the pathophysiological mechanism which is made of minute changes in "cochlear vasculature", along with possibly the glycogen accumulation is by and large held responsible for producing auditory dysfunction.

With respect to its metabolic function, basal turn of cochlea is much actively functioning when compared to cochlear apical turn. More so over, glycogen distribution is more to cochlear apical turn than to its basal turn[38]. P-creatinine levels decrease more speedily in basal turn as compared to cochlear apical turn during episodes of ischemia[39]. Hence, the basal turn is more liable to damage than the apical turn.

It was also found that any alterations were usually situated in basal turn, which is in accord to the common belief. Just in the same way, Saito et al[40] gave a report about the protrusions occuring in the stria vascularis. They mostly were positioned in basal turn.

Another subject with regard to raised cholesterol levels is that primary alteration takes place in stria vascularis or common hair cells. When apparently visualized under light microscopy and the TEM, changes in stria is graver than which is occurring in outer hair cell. This would probably show that whatever pathological changes, have first taken place in stria vascularis. Later spread to outer hair cells.

Likewise, a study by Saito et al[40] goes in same lines, noting that variations detected by the scanning electron microscope were far more obvious in stria vascularis rather than the organ of Corti.

METHODOLOGY

Study Design– An observational study.

Study Period– January 01, 2018 to December 31, 2018

Source of Data– Patients aged between 18 and 60 years with sensorineural hearing loss attending ENT & HNS outpatient department in KLES Dr Prabhakar Kore Hospital, Belagavi during the study period.

Sample Size– 58 cases.

Ethical Clearance– Obtained from the Institutional Ethical Committee

Inclusion Criteria –

Patients between the age group of 18-60 years with sensorineural hearing loss who are attending the ENT & HNS department in KLES Dr. Prabhakar Kore Hospital, after excluding certain patients mentioned under exclusion criteria by history, clinical examination and relevant investigations.

Exclusion Criteria –

1. Patients with chronic suppurative otitis media.
2. Patients with sensorineural hearing loss with a known cause like Meniere's disease, labyrinthitis, acoustic neuroma, temporal bone fracture, syphilis, meningitis and noise induced hearing loss.
3. All patients having diabetes mellitus.

4. Patients with chronic alcoholism, myxedema, nephrotic syndrome, and those on drugs affecting hearing and lipid levels like statins, oral contraceptives, beta-blockers.

Methodology –

- Informed consent was taken for the study.
- The patients' detailed history was obtained for duration of hearing loss, duration and nature of any previous treatment.
- All patients were clinically examined including general physical examination, detailed ENT examination and otoscopic examination of the ear. Tuning fork tests (Rinne's Test, Weber's Test, and Absolute Bone Conduction) were performed.
- Pure tone audiometry was performed on all patients to evaluate hearing profile.
- The fasting serum lipid profile estimation done by using ERBA commercial kit available for:

HDL

LDL

Triglycerides

Total cholesterol.

- Triglycerides, total cholesterol, HDL will be the measured parameters; LDL will be the calculated parameter.
- 5ml of venous blood was collected from patients after an over-night fast. This sample was evaluated for serum lipid profile. The blood was instilled in plain tubes, left to clot in water bath at 37ºC. Later it was followed by

centrifugation. It was at 3000 rpm for a minimum of 10 min. The separated serum was aspirated. It was stored at -20ºC. It was used for measurement of total cholesterol (TC), high-density lipoprotein (HDL) levels, and serum triglycerides. LDL levels were later calculated using an indirect method with the Friedewald's formula.

$$LDL = Total\ Cholesterol - \left(HDL + \frac{TRIGLYCERIDE}{5} \right)$$

Pure Tone Audiometry

The assessment of hearing was done on an outpatient basis with the help of pure tone audiometry. This test was performed by using the MAICO MA-53 apparatus (as shown in Pic.1). Here, the subject is made to sit in a soundproofed room, and wear headphones such that the designated cup falls over the designated ear. Testing is done separately for each ear.

Figure- 5 - Pure Tone Audiometry in progress

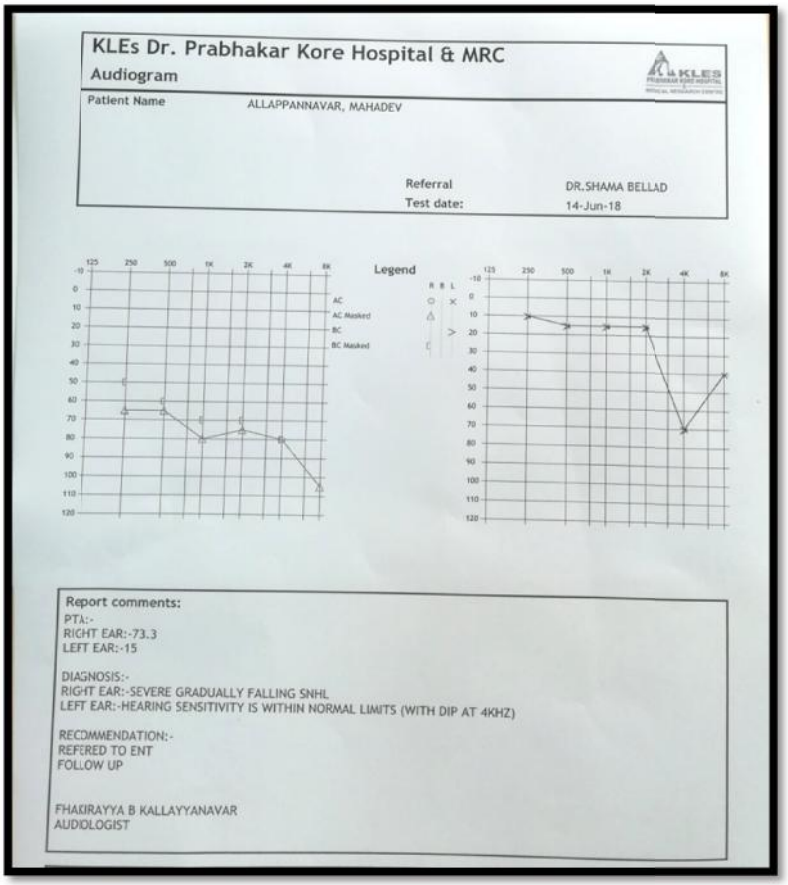
Pure tones (sounds of a single frequency) are presented to each ear in order of increasing frequency and the lowest perceived intensity for each tested frequency is recorded. This lowest perceived intensity is called the threshold for that frequency. The average increase in the thresholds of speech frequencies gives the magnitude of hearing loss in decibels (dB). Air conduction is tested by microphones in the external auditory canal and bone conduction is tested by placing a specialized microphone over the mastoid process in the post auricular region. The rationale is that impairment in hearing will reduce the perception of low intensity sounds (i.e., sounds of less loudness).

A graphic representation of thresholds for increasing frequencies of pure tones is made, and the resulting plot gives an indication of the type of hearing loss. The audiogram shows absent or negligible air bone gap for a sensorineural hearing loss and the air conduction as well as the bone conduction curves show elevated hearing thresholds.

Interpretation of Audiogram

In this study the degree of sensorineural hearing loss is based on the WHO classification and classified as:

Degree of hearing loss		Hearing threshold (Pure tone average)
Mild		26-40 dB
Moderate		41-55dB
Moderately Severe		56-70dB
Severe		71-90dB
Profound		> 91dB


KLEs Dr. Prabhakar Kore Hospital & MRC

Audiogram

Patient Name ALLAPPANNAVAR, MAHADEV

Referral DR.SHAMA BELLAD

Test date: 14-Jun-18

Legend

AC

AC Masked

BC

BC Masked

Report comments:

PTA:-

RIGHT EAR:-73.3

LEFT EAR:-15

DIAGNOSIS:-

RIGHT EAR:-SEVERE GRADUALLY FALLING SNHL

LEFT EAR:-HEARING SENSITIVITY IS WITHIN NORMAL LIMITS (WITH DIP AT 4KHZ)

RECOMMENDATION:-

REFERED TO ENT

FOLLOW UP

FHAKIRAYYA B KALLAYYANAVAR

AUDIOLOGIST

Figure- 6: PTA report.

STATISTICAL METHODS USED:

Data was analyzed using following statistical methods

1. Diagrammatic representation.
2. Mean ± Standard deviation.
3. Chi square test.
4. Kolmogorov Smirnov test for normalcy of the parameters.
5. Karl Pearson's correlation coefficient method to check the correlation.

RESULTS

In all, 58 patients aged between 18 to 60 years with sensorineural hearing loss attending ENT & HNS outpatient department in KLES Dr. Prabhakar Kore Hospital from January 2018 to December 2018 were studied. All observations recorded in the study are described under the following headings.

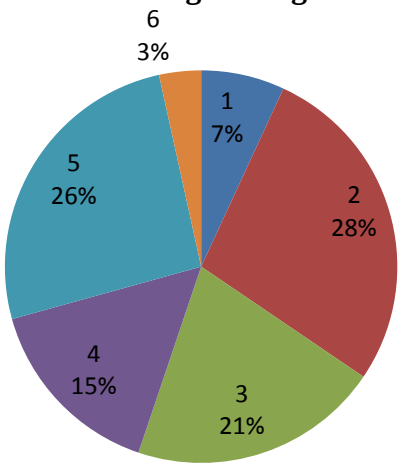
1. Percentage of degree of sensorineural hearing loss:

Out of 58 cases, 4 (7%) patients were having minimal hearing loss, 16 (28%) patients were having mild SNHL, 12 (21%) patients were having moderate SNHL, 9 (15%) patients were having moderately severe SNHL, 15 (26%) were having severe SNHL and 2 (3%) patients were having profound SNHL.

Table 3: Percentage of degree of SNHL

Total no. of cases	**58(100%)**
Minimal SNHL	4 (7%)
Mild SNHL	16 (28%)
Moderate SNHL	12 (21%)
Moderately severe SNHL	9 (15%)
Severe SNHL	15 (26%)
Profound SNHL	2 (3%)

Figure- 7: Percentage of degree of SNHL



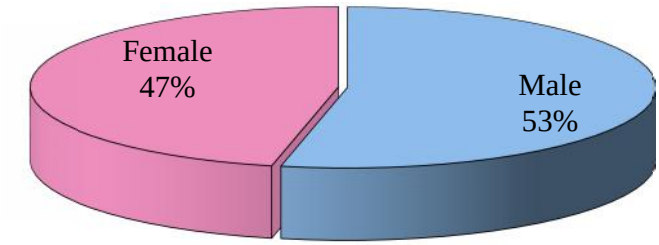
2. Sex distribution

Out of a total 58 cases, 31 (53.45%) were male and 27 (46.55%) were female. The sex distribution is depicted below.

Table – 4.

Gender	No of patients	% of patients
Male	31	53.45
Female	27	46.55
Total	58	100.00

Figure- 8 :Gender wise distribution of patients



3. Age wise distribution of patients.

The patients were distributed according to their age into categories. Out of the 58 patients, the groups were formed as <=30 years, which had 12 patients (20.69%), 31-40 years had 12 patients (20.69%), 41-50 years had 11 patients (18.97%), 51-60 years group had 23 patients (39.66%).

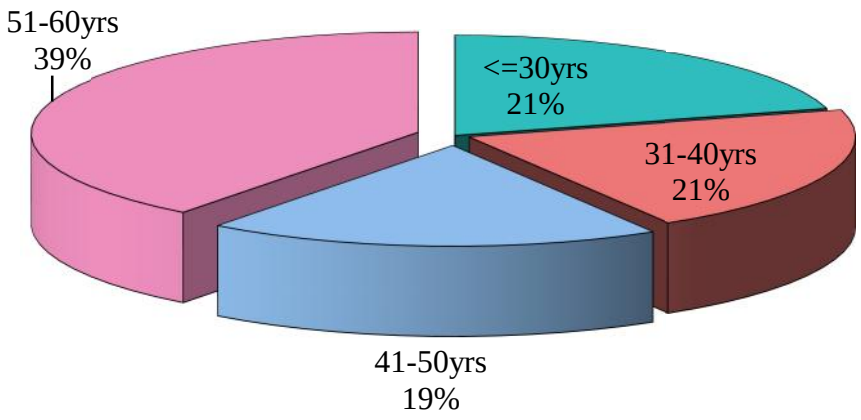
The Mean Deviation is 43.03 and Standard Deviation is 12.40. The data is depicted below.

Table-5

Age groups	No of patients	% of patients
<=30yrs	12	20.69
31-40yrs	12	20.69
41-50yrs	11	18.97
51-60yrs	23	39.66
Total	58	100.00
Mean age	43.03	
SD age	12.40	

Note: SD= Standard Deviation.

Figure- 9: Age groups wise distribution of patients



4. Distribution of males and females by age groups.

The distribution according to the age groups show that, 10 males and 2 females are in ages <=30yrs. 6 males and 6 females are between 31-40yrs, 3 males and 8 females are between 41-50yrs, 12 males and 11 females are between 51-60yrs.

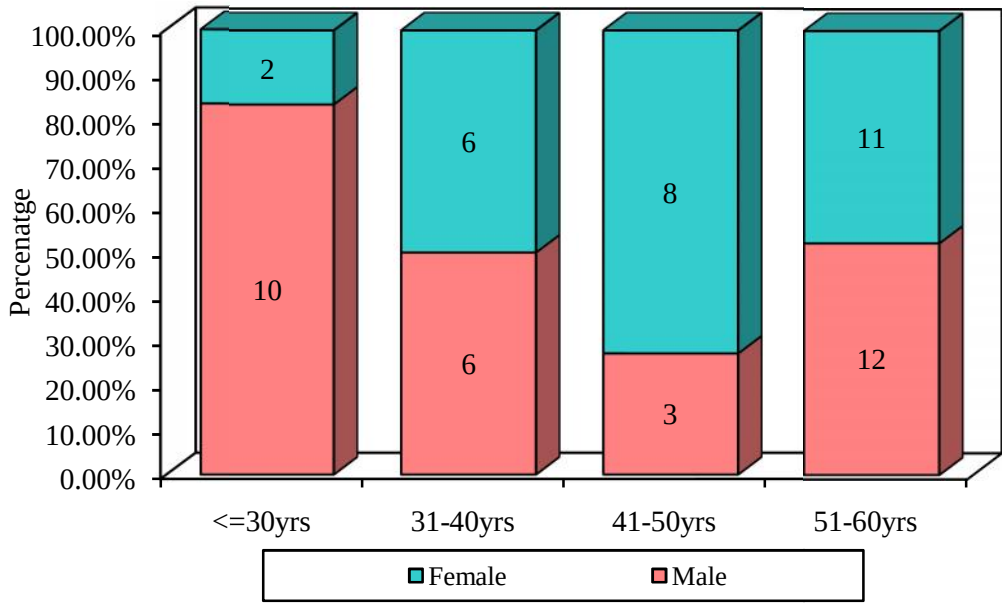
The Chi Square statistical value is 7.409 and the p value is 0.0601.

Since the p value is more than 0.05, it shows that a direct relationship exists between the age and severity of hearing loss.

Table- 6: Distribution of males and females by age groups

Age groups	Male	%	Female	%	Total	%
<=30yrs	10	83.33	2	16.67	12	20.69
31-40yrs	6	50.00	6	50.00	12	20.69
41-50yrs	3	27.27	8	72.73	11	18.97
51-60yrs	12	52.17	11	47.83	23	39.66
Total	31	53.45	27	46.55	58	100.00
Chi-square= 7.409 2 P = 0.0601						

Figure- 10: Distribution of male and females by age groups



5. Normality of blood parameters and hearing loss scores at right and left ear by Kolmogorov Smirnov test.

Table -7: Normality of lipid parameters and hearing loss

Parameters	Z-value	p-value
HDL	0.8320	0.4930
LDL	0.8970	0.3970
Triglycrides	0.8940	0.4010
Total Cholesterol	0.8440	0.4740
Degree of Hearing Loss (Right)	0.8980	0.3960
Degree of Hearing Loss (Left)	0.8800	0.4210

The blood parameters like the HDL, LDL, triglycerides, total cholesterol and hearing loss scores at right and left side follow a normal distribution; therefore, the parametric tests were applied by Karl Pearson's correlation coefficient method.

6. Correlation between age in yrs with blood parameters.

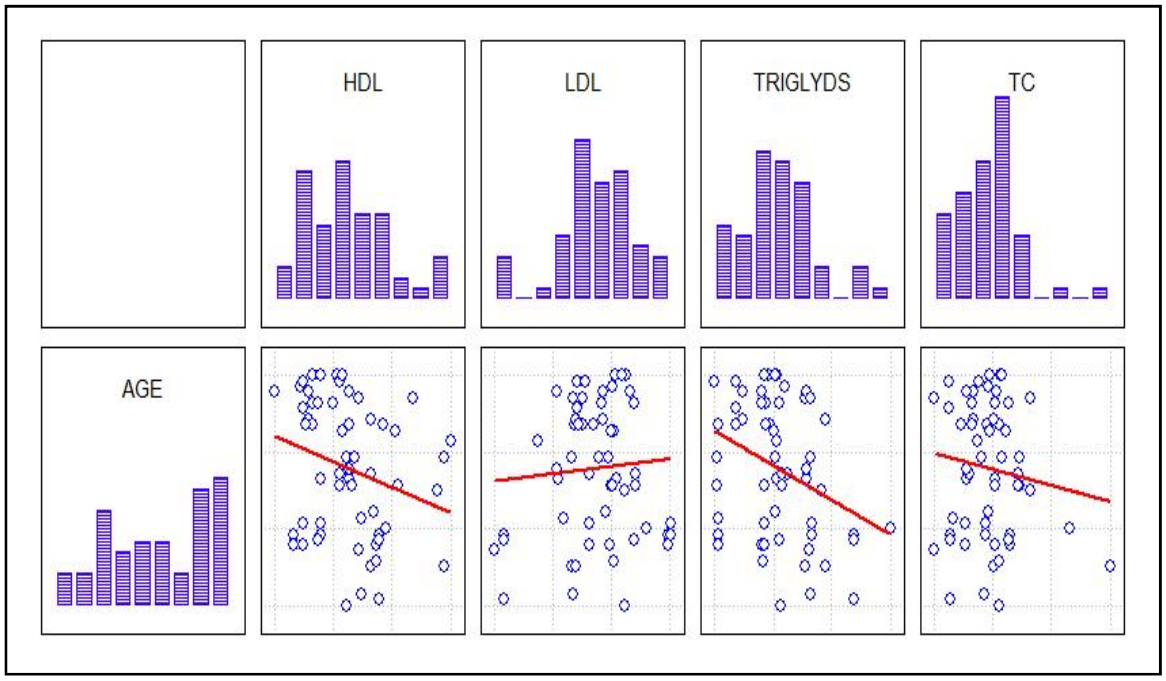
The 58 patients were categorized into 4 groups according to their ages, and the correlation between age and lipid profile was done. It was found that, HDL and triglycerides had a strong negative correlation (-2.02 and -2.67) with advancing age, and it was also significant. The p values were 0.047 for HDL and 0.0099 for triglycerides.

Total cholesterol also showed a negative correlation with advancing age (-0.925), with p value of 0.35. On the other hand, LDL showed a positive correlation with age (+0.548) with a p value of 0.58.

Table- 8

Blood parameters	Correlation between age in yrs with lipid parameters		
	r(X,Y)	t-value	p-value
HDL	-0.2611	-2.0244	0.0477*
LDL	0.0730	0.5481	0.5858
Triglycerides	-0.3361	-2.6704	0.0099*
Total Cholesterol	-0.1228	-0.9259	0.3585

Figure- 11: Scatter plot of correlation between age in yrs with blood parameters



7. Correlation between hearing loss with blood parameters by Karl Pearson's correlation coefficient method.

All the 58 patients were categorized into 5 groups. minimal SNHL seen in 4 patients, mild SNHL= 16 patients, moderate SNHL =12 patients, moderately severe SNHL = 9 patients, severe SNHL = 15 patients and profound hearing loss was seen in 2 patients.

Next, the degree of hearing loss was compared with lipid profile parameters of the patients. It was found that High Density Cholesterol and Triglycerides were having a negative correlation with increasing degree of hearing loss (-1.46 and -1.52). Their p values were 0.1488 and 0.1329. Similarly, the Low Density Cholesterol and total cholesterol levels had a minimal positive correlation with the increasing degree of hearing loss (0.99 and 0.46), and their p values are 0.3260 and 0.6435 each.

Table- 9

Blood parameters	**Correlation with hearing loss.**		
	r(X,Y)	t-value	p-value
HDL	-0.1920	-1.4639	0.1488
LDL	0.1313	0.9909	0.3260
Triglycerides	-0.1997	-1.5248	0.1329
Total Cholesterol	0.0621	0.4653	0.6435

Figure- 12: Scatter plot of correlation between hearing loss with blood parameters

8. Comparison of male and female patients with blood lipid parameters by independent t test.

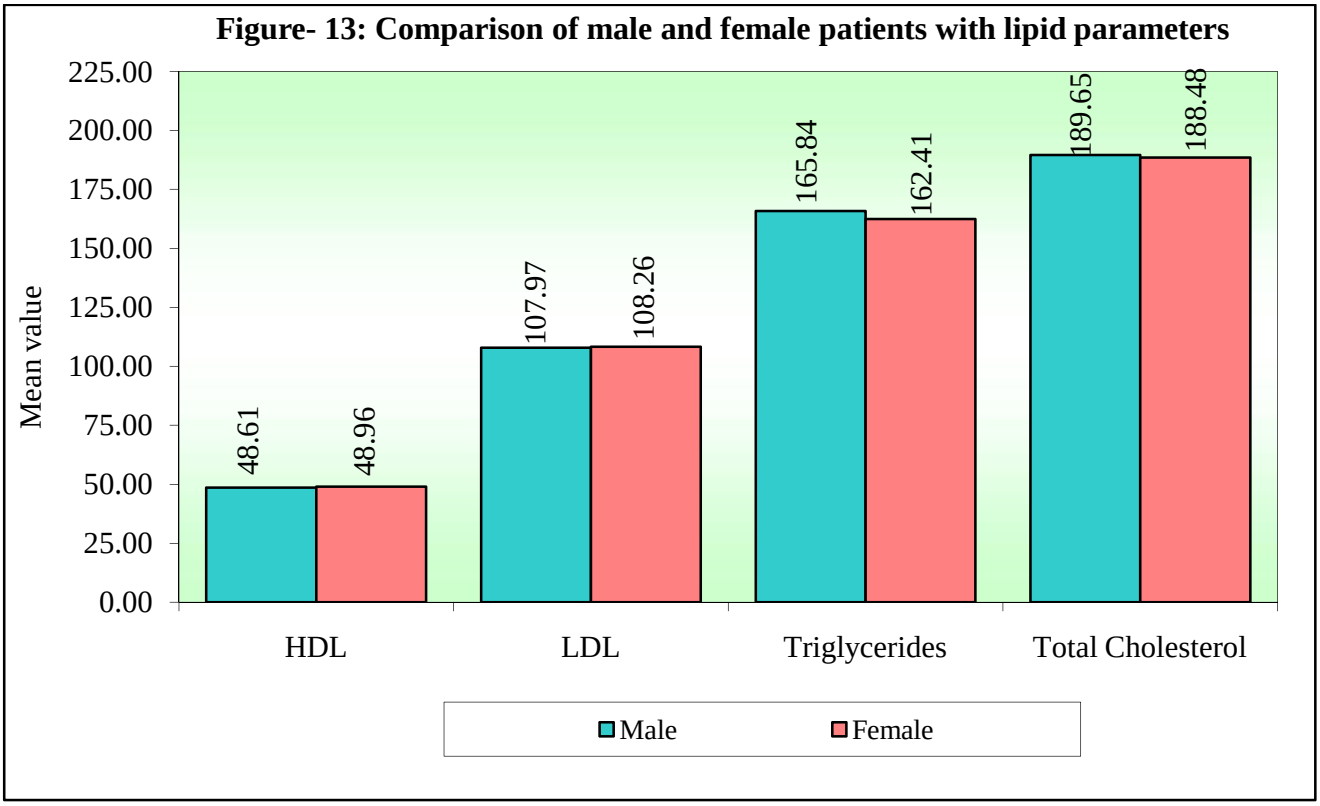
Statistical analyses were carried out using Karl Pearson's correlation coefficient method. The mean values of HDL, LDL, triglycerides and total cholesterol among the males and females were found to be similar.

The p values were all above 0.05, and hence statistically not significant.

Table -10: Comparison of sex ratio with lipid parameters

Variable	Gender	Mean	SD	SE	t-value	P-value
HDL	Male	48.61	13.08	2.35	-0.1000	0.9207
	Female	48.96	13.55	2.61		
LDL	Male	107.97	32.99	5.93	-0.0366	0.9709
	Female	108.26	26.70	5.14		
Triglycerides	Male	165.84	53.59	9.62	0.2575	0.7977
	Female	162.41	46.96	9.04		
Total Cholesterol	Male	189.65	35.59	6.39	0.1448	0.8854
	Female	188.48	23.34	4.49		

Figure- 13: Comparison of male and female patients with lipid parameters



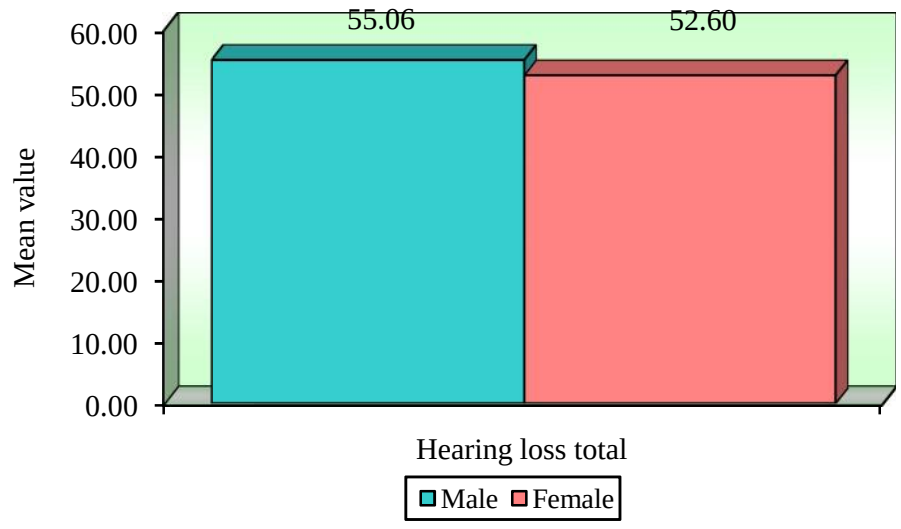
9. Comparison of male and female patients with hearing loss scores by independent t-test.

Statistical analyses were carried out using Karl Pearson's correlation coefficient method. The mean values of hearing loss in males and females were found to be similar. The p value is 0.6, which is above 0.05, and hence statistically not significant.

Table –11.

Variable	Gender	Mean	SD	SE	t-value	P-value
Hearing loss	Male	55.06	21.36	3.84	0.4244	0.6729
	Female	52.60	22.79	4.39		

Figure- 14: Comparison of sex ratio with hearing loss scores



DISCUSSION

The results of this study show that there are significant alterations in the lipid profiles among different degrees of sensorineural hearing loss.

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.

Sex distribution: comparison with present study.

Table- 12.

Studies	Males	Females	male: female ratio
Lee FS et al[14]	128	89	1.44:1
Suzuki K et al[3]	607	317	1.91:1
Present study	31	27	1.15:1

The sex distribution among various studies has been depicted here. Lee FS et al[14] in their study had a total of 128 males and 89 females with a ratio of 1.44:1. Similarly, Suzuki K et al studied 607 males and 317 females with a ratio of 1.91:1.

In the present study, 31 males and 27 females were studied, and the male: female ratio is 1.15:1.

Age distribution and hearing loss:

According to Scott Brown 8th edition[17], the prevalence of age associated hearing loss in a population is tabulated:

Table- 13: Age distribution and hearing loss

Age group (years)	Prevalence (%)
18–30	2
31–40	5
41–50	10
51–60	17
61–70	30
71–80	53

In this present study, it is found that a positive correlation exists between advancing age and hearing loss, with the p values of 0.99 and 0.06 for each ear.

Table- 13.i.

Variables	Correlation between age in yrs with		
	r(X,Y)	t-value	p-value
Hearing loss in right side	-0.0001	-0.0011	0.9991
Hearing loss in left side	0.2452	1.8931	0.0635

Total Cholesterol:

Comparison of Total Cholesterol in the whole study group:

Table- 14.

Studies	Mean Total Cholesterol(mg/dl)
Lee FS et al[14]	211.83±20
Jones NS et al[13]	225.77
Present study	189.1± 23.34

The mean Total Cholesterol values among various groups of SNHL in the above studies done by Lee FS et al[14] and Jones NS et al[13] were not statistically significant.

In our present study there was no statistical significance of the mean TC values among various degree of SNHL in accordance with the above mentioned studies.

Triglycerides:

Comparison of Triglycerides in the whole study group.

Table- 15.

Studies	Mean Triglycerides(mg/dl)
Lee FS et al[14]	52.80 ± 12.88
Jones NS et al[13]	43
Present study	164.24 ± 46.96

Lee FS et al[14] studied various blood chemistry measures and serum lipids in 217 patients with hearing levels ranging from normal to moderate/severe. They found that the mean triglyceride values were 52.8±12.88 mg/dl.

Similarly, Jones NS et al[13] in their prospective study on 85 patients found the mean triglyceride levels to be 43 mg/dl.

In present study of 58 patients, the mean triglyceride levels were found to be 164.24 ± 46.96 mg/dl, and showed a significant negative correlation with advancing age. (p=0.0099)

LDL:

Comparison of LDL in the whole study group

Table- 16

Studies	**Mean LDL(mg/dl)**
Lee FS et al[14]	185.33 ± 20.32
Present study	108.1 ± 26.7

Lee FS et al[14] found in their study the mean LDL levels to be 185.33 ± 20.32, whereas in the present study the mean LDL value was found to be 108.1 ± 26.7. The levels of serum LDL had a positive correlation with the hearing levels in both male and female populations of the study group.

HDL:

Comparison of HDL in the whole study group

Table- 17.

Studies	Mean HDL(mg/dl)
Lee FS et al[14]	16 ± 6.43
Present study	48.77 ± 13.08

Lee FS et al[14] in their study found the mean HDL values to be 16.00 ± 6.43mg/dl. In the present study, the mean HDL values were found to be 48.77 ± 13.08mg/dl, and these values had a significant negative correlation with advancing age. (p=0.047)

Comparison of Lipids parameters with severity of hearing loss in the Present study.

Table- 18.

	HDL	**LDL**	**TGL**	**TC**
Normal values	***40-60mg/dl***	***100-160mg/dl***	***80-200mg/dl***	***150-250mg/dl***
Mild	53.81	100	177.75	184.68
Moderate	53.33	101.75	189.5	196.92
Moderately Severe	48.44	106.67	157	195.44
Severe	44.06	115.93	148.33	187.73
Profound	45	107	123.5	177.5

ATP III Classification of LDL, Total and HDL Cholesterol (mg/dl)

Table- 19.

LDL Cholesterol	
<100	Optimal
100-129	Near optimal/ above optimal
130-159	Borderline high
160 – 189	High
≥ 190	Very high
Total Cholesterol	
<200	Desirable
200 - 239	Borderline high
≥ 240	High
HDL Cholesterol	
< 40	Low
≥ 60	High

Comparing the various lipid parameters with NCEP Adult Treatment Panel III guidelines[41, 42] it can be noted that total cholesterol in our study population is within normal limits among different degrees of hearing loss. The serum levels of LDL were found to be near optimal among all the hearing loss groups. The HDL and triglyceride levels on the other hand is found to be high in case of mild and moderate hearing loss, low in cases of moderately severe and severe hearing loss.

Friedrich G et al[6] in 1981 found no variation in serum triglyceride levels or even the total cholesterol concentrations among the forty-nine patients which they studied suffering from neuro-otological symptoms i.e. dizziness, tinnitus or SNHL. These patients had a significantly raised LDL concentration and ratio of LDL/HDL in serum. Hence the study suggested a positive association to exist between atherosclerosis and dysfunction of the inner ear.

Gates GA et al[43] reported that no correlation existed between hearing levels and total cholesterol or triglycerides in a population, but HDL in women had a negative correlation with hearing levels.

In this present study HDL and triglycerides negatively correlated with hearing levels in both males and females.

HDL-CH (High Density Lipoprotein Cholesterol) is a type of cholesterol carried by HDL and indirectly reflects HDL levels in serum. HDL eliminates extra cholesterol from blood and organs by counter transport thus slowing the process of atherosclerosis.[44] HDL can also reverse endothelial dysfunction, inhibit LDL oxidation, stimulate endothelial cell proliferation and prostacyclin generation.[45] Lowered HDL level diminishes these benefits and increases the risk of thrombosis.

A study by Ullrich, Aurbach and Drobik[46] commented about hyperlipidaemia and all atherogenic risk factors, and found no major pathological role they had to play in Sudden SNHL.

Also, Kazmierczak H and Doroszeweska G[47] came to a conclusion that disturbance of glucose metabolism with raised insulin levels could be responsible for

causing inner ear diseases. But the role of disturbance of lipid metabolism still remains imprecise.

Likewise, Claudia Rudack et al[48] found correlation between HDL-CH and LDL-CH and Sudden SNHL.

These results suggest that a high serum LDL and low HDL shows a strong association with atherosclerosis-related micro circulatory disturbances of blood supply of cochlea and also increased sensitivity of the cochlea to the heard noise. When summed up with daily exposure to noise, such changes can lead to varying amounts of hearing loss. Antilipid therapy and low cholesterol diet can provide significant improvement by way of lowered tinnitus intensity and improvement in average hearing thresholds.

CONCLUSION

The cholesterol and triglycerides are the two major lipid components that are found in serum. They are present along with other phospholipids and certain free fatty acids. Due to their high tendency to be associated with various diseases and certain metabolic abnormalities, such serum lipids have become very important variables that need to be measured routinely by doing laboratory tests.

Auditory disturbances in the general population and its association with serum lipids have been extensively studied both clinically and experimentally. On contrary, their relation still remains a controversy.

In this study alteration in different lipoprotein fractions in various degrees of sensorineural hearing loss was studied.

1. The male: female ratio in this series is approximately 1.15:1.
2. The serum levels of total cholesterol do not have much influence on severity of sensorineural hearing loss.
3. The serum levels of LDL directly correlate with the severity of sensorineural hearing loss in both sexes.
4. The serum levels of HDL and triglycerides negatively correlate with the severity of sensorineural hearing loss in both sexes.

From the observations in the study, we can conclude that there is a need for continual assessment of serum lipid levels in people with sensorineural hearing loss and it should be managed effectively to prevent the further progression of sensorineural hearing loss.

SUMMARY

This study was conducted in KLES Dr Prabhakar Kore Hospital, Belagavi during a study period of one year on patients aged between 18 and 60 years with sensorineural hearing loss attending ENT & HNS outpatient department.

All patients' through history taking and examination was done. These patients were subjected to Pure Tone Audiometry for assessment of hearing threshold and fasting serum lipid profile estimation. The following results were noted.

i. Male to Female ratio in the study group was 1.15:1
ii. There was no statistical significance for total cholesterol among various degree of SNHL in both males and females.
iii. The serum level of low density lipoprotein was low in the mild sensorineural hearing loss and high in severe sensorineural hearing loss in both sexes and thus directly correlated with severity of degree of sensorineural hearing loss.
iv. The serum levels of high density lipoprotein and triglycerides were found to be high in mild group and low in severe sensorineural hearing loss in both sexes and thus negatively correlated with degree of sensorineural hearing loss.

This study confirms the findings of similar studies that LDL has a positive correlation with degree of SNHL and HDL has a negative correlation, but no statistically significant figures were found in this study population.

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ANNEXURE – I- INFORMED CONSENT

"LIPID PROFILE IN PATIENTS WITH SENSORINEURAL HEARING LOSS"- A one year hospital based observational study in KLES Dr. Prabhakar Kore Hospital.

PRINCIPAL INVESTIGATOR: **Dr. _____**

Post Graduate student

Department of Otorhinolaryngology.

CO-INVESTIGATOR: **Dr. _____**

Vice Principal, J.N. Medical College

Professor, Department of Otorhinolaryngology

INTRODUCTION AND PURPOSE:

The present study is conducted among patients with sensorineural hearing loss attending the out-patient department of ENT & HNS in KLE's Dr.Prabhakar Kore Charitable Hospital and Medical Research Centre, Belgaum and will be investigated for your serum Lipid levels on out-patient basis. You are requested to participate in the study and your participation is completely voluntary.

PROCEDURE:

If you agree to participate in this study, the relevant data will be collected as per the proforma and the final diagnosis will be confirmed.

After getting inducted in the study, you will be evaluated for sensorineural hearing loss with pure tone audiometry and functions of cochlea. Patient will also be investigated for the serum Lipid levels and the association will be studied.

BENEFITS:

Patient will not be eligible for any kind of monetary benefits or free services by virtue of your participation in the study.

RISKS:

Methods applied to do the study are safe.

COST OF PARTICIPATION:

The cost of the Investigation will be borne by the Study Subject. The other indirect expenses will be borne by the Investigator.

PRIVACY AND CONFIDENTIALITY:

The results of the study may be published in journals for scientific purposes. However, your identity will not be revealed. All information collected will be coded so that no one other than the investigator will know your identity.

WITHDRAWAL FROM THE STUDY:

You can withdraw from the study at any time if you wish to do so.

AUTHORIZATION TO PUBLISH THE RESULTS:

The researcher may use the information gathered from this study for presentation in scientific meetings. However, your identity will not be revealed.

QUERIES AND CONTACT:

If you have any questions about rights as a research participant you can contact Dr Roopa M Bellad, Professor, department of Paediatrics, Jawaharlal Nehru Medical College Institutional Ethics Committee on human subjects' research.

CONSENT SUMMARY:

I have been explained all the contents of this consent form in my local language and having understood and clarified all my queries about the study to the best of my knowledge, I hereby give my voluntary consent for participation in the study. I do sign the informed consent form in front of an eyewitness whom I recognize.

Name and Signature/ left thumb impression of the participant:

Name and Signature of the interviewer:

Name and Signature/ left thumb impression of the eyewitness (Relative):

Signature of the guide:

Date:

ANNEXURE – II– ETHICAL CLEARANCE LETTER

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
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Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 74 **Date: 22/11/2017**

To,

PG student in ENT&HNS,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"LIPID PROFILE IN PATIENTS WITH SENSORINEURAL HEARING LOSS – A ONE YEAR HOSPITAL – BASED OBSERVATIONAL STUDY IN KLE'S DR. PRABHAKA KORE HOSPITAL",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE –III - PROFORMA

"LIPID PROFILE IN PATIENTS WITH SENSORINEURAL HEARING LOSS" – A one year hospital based observational study in KLES Dr. PRABHAKAR KORE CHARITABLE HOSPITAL.

Date:

O.P. No:

Name: Age:

Sex: Occupation:

Address: Phone No:

1) CLINICAL PROFILE:

Chief Complaint:

History of Present Illness

Past History:

Personal History:

Family History:

2) GENERAL PHYSICAL EXAMINATION -

Blood Pressure:

Pulse:

Respiratory Rate:

Pallor

Icterus

Clubbing

Cyanosis

Lymphadenopathy

Oedema

3) ENT EXAMINATION

- EAR EXAMINATION:

Right Left

Pinna

Pre auricular area

Post auricular area

External auditory canal

Tympanic membrane

i. Tuning fork tests:

Rinne's test 256 Hz

512 Hz

1024 Hz

Weber's test:

Absolute Bone Conduction test:

ii. Facial nerve examination:

- NOSE EXAMINATION

a. External appearance:

Root

Bridge

Dorsum

Alae

Tip

Columella

Cold spatula test:

b. Anterior Rhinoscopy:

c. Posterior Rhinoscopy:

- PARANASAL SINUS EXAMINATION :
- THROAT EXAMINATION :
- NECK EXAMINATION :

4) DIAGNOSIS:

5) ROUTINE TESTS:

CBC : **GRBS:**

LFT: **MR:**

6) PTA :

7) LIPID PROFILE :

Total Cholesterol –	[150-250 (mg/dl)]
Triglycerides –	[80-200 (mg/dl)]
High Density Cholesterol (HDL) –	[35-80 (mg/dl)]
Low Density Cholesterol (LDL) –	[62-160 (mg/dl)]

ANNEXURE – IV - KEY TO MASTER CHART

F	:	Female
M	:	Male
dB	:	decibel
SNHL	:	Sensor neural hearing loss
LDL	:	Low density lipoproteins
HDL	:	High density lipoproteins
Yrs	:	years

S. No.	Age (yrs)	Sex	HDL (mg/dl)	LDL (mg/dl)	Triglycrides (mg/dl)	Total Cholesterol (mg/dl)	Hearing Loss (Right)	Degree of Hearing Loss (Right) dB	Hearing Loss (Left)	Degree of Hearing Loss (Left) dB	Hearing loss in worse ear	Degree of hearing loss dB
1	25	M	80	90	200	310	moderate	50.3	moderate	46	moderate	50.3
2	48	M	82	64	162	178	Mild	30	moderate	43	moderate	43
3	32	M	61	147	310	270	moderate	50	moderate	48	moderate	50
4	20	M	53	91	207	185	moderate	56.6	moderate	56.6	moderate	56.6
5	34	F	53	83	86	153	severe	73	profound	90	profound	90
6	31	M	59	38	264	150	minimal	23.3	minimal	23.3	mild	26
7	41	F	49	79	202	167	mild	26	mild	26	mild	26
8	25	M	56	93	226	194	mild	36.6	moderate	43.3	moderate	43.3
9	58	M	33	121	173	189	mild	24	mild	23	mild	24
10	45	F	80	110	85	220	mild	31.6	normal	18.3	mild	31.6
11	40	F	65	120	85	220	severe	75	severe	75	severe	75
12	39	F	78	130	220	230	Mild	25	mild	25	mild	25
13	42	F	56	103	176	170	mild	40	mild	40	mild	40
14	51	F	49	98	153	187	minimal	20	moderately severe	65	moderately severe	65
15	55	M	37	95	149	172	mild	30	mild	39	mild	39
16	31	F	31	163	86	211	minimal	24	normal	18	minimal	24
17	33	M	40	165	208	186	severe	88	mild	30	severe	88
18	56	F	70	98	200	230	moderately severe	65	moderately severe	65	moderately severe	65
19	50	F	47	122	158	206	moderate	53.3	moderate	51.6	moderate	53.3
20	60	F	45	123	163	202	severe	76.6	moderately severe	58.3	severe	76.6
21	45	F	48	118	160	202	Mild	35	severe	85	severe	85
22	41	F	40	124	163	195	normal	13.3	moderately severe	58.3	moderately severe	58.3
23	18	M	48	130	167	200	moderately severe	68.3	severe	88.3	severe	88.3
24	40	M	50	121	147	195	mild	30	normal	15	mild	30
25	52	F	36	114	119	174	minimal	20	minimal	20	minimal	20
26	51	M	35	106	109	162	moderate	50.3	moderate	50.3	moderate	50.3
27	40	M	46	139	169	219	severe	78.3	mild	26.6	severe	78.3
28	33	M	34	103	146	166	severe	73.3	normal	15	severe	73.3
29	59	M	34	94	110	149	moderately severe	68.8	severe	75	severe	75
30	55	M	39	137	166	209	moderate	55	moderate	55	moderate	55
31	59	M	46	100	82	179	severe	73.3	severe	85	severe	85
32	51	M	60	92	86	200	moderately severe	60	moderate	53.3	moderately severe	60
33	29	M	58	119	143	196	minimal	23.3	mild	25	mild	25

S. No.	Age (yrs)	Sex	HDL (mg/dl)	LDL (mg/dl)	Triglycrides (mg/dl)	Total Cholesterol (mg/dl)	Hearing Loss (Right)	Degree of Hearing Loss (Right) dB	Hearing Loss (Left)	Degree of Hearing Loss (Left) dB	Hearing loss in worse ear	Degree of hearing loss dB
34	60	F	47	123	151	201	severe	73.3	severe	83.3	severe	83.3
35	35	F	57	116	161	189	moderately severe	56	severe	71.6	severe	71.6
36	55	M	44	113	143	195	moderate	48.3	moderately severe	63.3	moderately severe	63.3
37	26	M	58	122	145	200	mild	30	normal	13.3	mild	30
38	60	F	37	131	161	202	moderate	41.6	profound	91.6	profound	91.6
39	57	F	25	134	143	197	severe	78.3	severe	86.6	severe	86.6
40	60	M	40	128	146	190	moderate	45	moderate	41.6	moderate	45
41	50	M	64	120	158	190	Mild	37	mild	35	mild	37
42	28	M	52	31	205	135	severe	80	moderately severe	65	severe	80
43	43	F	49	78	202	167	minimal	18.3	mild	40	mild	40
44	52	F	56	93	226	194	mild	40	mild	35	mild	40
45	45	F	51	90	206	182	Mild	33.3	mild	33.3	mild	33.3
46	51	F	37	95	149	172	moderately severe	60	moderately severe	65	moderately severe	65
47	31	F	40	165	208	186	minimal	26	minimal	20	minimal	26
48	29	M	31	163	86	211	moderately severe	63.3	normal	11.6	moderately severe	63.3
49	57	M	36	114	119	174	normal	15	moderate	50	moderate	50
50	42	M	46	139	169	219	moderately severe	60	minimal	23.3	moderately severe	60
51	29	F	34	103	146	166	normal	11.3	mild	28.3	mild	28.3
52	54	F	34	94	110	149	minimal	21.3	minimal	23.3	minimal	23.3
53	51	M	35	106	109	162	severe	71.6	severe	81	severe	81
54	30	M	39	137	166	209	severe	80	severe	73	severe	80
55	30	F	59	38	264	150	normal	15	moderately severe	46.6	moderately severe	46.6
56	57	F	49	78	202	167	Mild	26.6	minimal	23.3	mild	26.6
57	56	M	52	91	207	135	moderate	46.6	normal	13.3	moderate	46.6
58	19	M	59	38	264	150	Moderate	53.3	normal	10	moderate	53.3