"ASSESSMENT OF HEARING IN PATIENTS WITH VITILIGO -A ONE YEAR OBSERVATIONAL STUDY IN KLES Dr. PRABHAKAR KORE HOSPITAL"

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Dr. B.P. BELALDAVAR M.S (ENT), D.L.O, Ph.D,
Professor & Head of Department
Department of Otorhinolaryngology
and Head & Neck Surgery
J.N.Medical College,
Nehru Nagar, Belagavi-590010

Professor & Head
Department of E.N.T.
J. N. Medical College
Belagavi

Date: 30/12/2022
Place: Belagavi

Dr.(Mrs)N.S.MAHANTASHETTI M.D,(Peads)
Principal
J.N.Medical College,
Nehru Nagar,
Belagavi -590010

PRINCIPAL
J.N. Medical College,
BELAGAVI-590 010

Date: 2/1/2023
Place: Belagavi

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Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MHRD (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

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Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BE0120008.
Postgraduate Student,
2020-21 Batch,
Department of ENT,
J. N. Medical College, Belagavi.

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K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 66 **Date: 25/01/2021**

To,

PG student in Otorhinolaryngology and Head & Neck Surgery,
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 **"ASSESSMENT OF HEARING IN PATIENTS WITH VITILIGO –A ONE YEAR OBSERVATIONAL STUDY IN KLES DR PRABHAKAR KORE HOSPITAL",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ABSTRACT

Melanocytes present in epidermis, mucous membranes, and other tissues are reduced in vitiligo, which is a chronic multifactorial illness with a complex pathophysiology. The first person to mention the existence of pigment cells in the cochlea in the inner ear was Alphonse Corti in 1831. Melanocytes are particularly prevalent in places with apparent significant secretory or metabolic function, such as Reissner's membrane, the vascular stria, and other highly vascularized areas. Only a few studies have examined the assessment of hearing in vitiligo patients, and a number of anomalies have been noted. In this regard, the goal of the current study is to use pure tone audiometry (PTA) to further demonstrate the auditory involvement in vitiligo patients.

Objective: To assess the prevalence of auditory involvement in patients with vitiligo

Methodology: It was a one-year observational study. The study included a total of 71 vitiligo patients between the age group of 18 - 60 years. Each patient underwent ENT examination, Pure Tone Audiometry (PTA), and dermatological evaluation.

Results: The mean age of the subjects in this study was 34 years, with a male predominance. When the correlation between hearing loss and vitiligo was evaluated and the left and right ears were compared, there was statistically no significant hearing loss in either ear. A mean value was calculated after taking into account the duration of vitiligo and the Vitiligo Area Scoring Index (VASI) with that of hearing loss individually, however it revealed no association between the two.

Conclusion:Based on the study results, we can conclude that there is a need for ongoing assessment of hearing status in patients with Vitiligo.

LIST OF ABBREVIATIONS

PTA	-	Pure Tone Audiometry
SNHL	-	Sensory Neural Hearing Loss
SV	-	Stria vascularis
OSL	-	Osseous spiral lamina
EP	-	Endocochlear potential
OAE	-	Otoacoustic emissions
ABR	-	Auditory brainstem response
MOC	-	Medial olivo cochlear
BPPV	-	Benign paroxysmal positional vertigo
VASI	-	Vitiligo area scoring index
K	-	Potassium
dB	-	Decibel, Unit of sound intensity
WHO	-	World health organisation
Hz	-	Hertz
TEOAE	-	Transient evoked otoacoustic emissions
BAEP	-	Brainstem auditory evoked potential
et al	-	et alii (Latin; ‘and others’)

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INTRODUCTION

The frequency of Vitiligo (Leukoderma) is 0.1 to 0.2 percent worldwide[1]. Cells producing pigments (Melanocytes) present in epidermis, mucous membranes, and other tissues are reduced in this multifactorial illness, which has a complicated pathophysiology. The pathophysiology behind the loss of epidermal melanocytes has been the subject of several ideas, although the exact explanation is still unknown. The body's active melanocytes, which include the pigment cells in the retina, inner ear, and hair, are all affected. Numerous propositions have been put out, including the role of genetic vulnerability to vitiligo as well as autoimmunity, cytotoxicity, biochemistry, oxidant-antioxidant, neurological, and viral mechanisms underlying the loss of epidermal melanocytes. Incomplete penetrance, numerous susceptibility loci, and genetic variability are characteristics of vitiligo[1]. The gene involved for melanin manufacture, oxidative stress response, and autoimmune control is linked to the inheritance of vitiligo.

A potential symptom of vitiligo is depigmented macules that progressively get larger and give rise to new lesions. Latest studies in this field have shown that disease development may include a systemic mechanism because vitiligo is associated with ophthalmological and hearing difficulties, including Sensorineural hearing loss (SNHL). However, there has been much discussion throughout the years regarding how common SNHL is among vitiligo sufferers.

Hearing is impacted by systemic diseases including Vogt-Koyanagi and Waardenberg Syndrome, which implies that it is possible that cell producing the pigment melanin is involved,and have an important function in the inner part of ear. The first person to mention the existence of cells responsible for production of

pigments in cochlea in inside part of ear was Alphonse Corti in 1831. Cells producing pigments are notably abundant within stria vascularis(SV), central bony column, osseous spiral lamina(OSL), vestibular membrane, along regions with strong blood flow that appear to have essential secretory or metabolic function[2]. According to Savin[3] (1965), cells that produce pigments are completely or to limited extent attached to capillary walls, where transfer of substances, matters takes place. As a result, pigments help to maintain cell membrane homeostasis and helps in easing movement of materials a distance spanning both sides. Cells producing pigments are found in layers of the stria vascularis in inside part of ear, and they are necessary for the cochlea to mature normally. For the cochlear hair cells to operate and for optimal hearing, bodily fluid that fills the membranous labyrinth of the inner ear, alongside exceptionally elevated Endocochlear Potential (EP), which is typically approximately -80 milli Volts and seems crucial. It is also thought that EP is developed and/or maintained by cells producing pigments in the SV of the inside part of ear. Therefore, when these cell numbers gets lesser, there occurs a consequent decrease in EP, which may result in hearing loss. The central auditory system also contains cells producing pigments, as evidenced by the study on nervous system of albino rabbits, and revealed that those were twenty-four percent lesser in size than those of control group ,which proves same, cells producing pigments do not exist exclusively in peripheral acoustic apparatus.

Only a few studies have examined the assessment of hearing in vitiligo patients, and a number of anomalies have been noted. The majority of studies on this issue have used otoacoustic emission (OAE) and auditory brain stem response (ABR) to determine patient's auditory involvement. More study of vitiligo patients is needed

to fully understand the relationship between cells producing pigments, function and hearing.

The goal of the current study, which employs Pure Tone Audiometry (PTA), is to further demonstrate the auditory involvement of vitiligo patients.

AIM & OBJECTIVE

- To assess the prevalence of auditory involvement in patients with vitiligo.

REVIEW OF LITERATURE

The idea that SNHL is substantially more prevalent among vitiligo patients in a vast number of clinically controlled studies with a prevalence ranging from 4% to 68.8%, was supported by a study done in 2017 by Marrigje Aagje de Jong et al. They also stated that, since SNHL appears to be more prevalent towards increasing frequency range, audiometry should be done at very high audio frequencies like 10 and 12 kHz.[2]

Neel Prabha et al conducted a study in 2020, found that the age range 41 to 60 years had the highest prevalence of SNHL cases (63.6%), which was statistically significant. 5 out of 10 patients (66.7%) and 5 out of 32 (86.5%) of those with vitiligo for more than 5 years both had sensorineural hearing loss. They came to the conclusion that elderly people with vitiligo may be more susceptible to audiological problems. Additionally, these patients should be made aware of the risk that exposure to ototoxic drugs and noise pose.[4]

In a research by Mohammed E. S., Said E. A., Sayed D. S., Awad. S. M, and Ahmed. M. H, 37% of vitiligo subjects were found to have SNHL. Of them,Sixty-seven percent subjects experienced bilateral hearing loss at high frequencies (2-8kHz). They suggested that there might be a connection between people with vitiligo's ocular abnormalities and their peripheral auditory abnormalities. [5]

According to a research by Parvane Mahdi, Masood Rouzbahani, Amin Amali, Samad RzaiiKhiabanlu, and Mohammed Kamali, 38.09% of vitiligo patients had high frequency SNHL. There were no connection found amidst age, time span of Vitiligo, as well as audiological abnormalities.[6]

According to a 2013 study by Fleissig, M. Gross, I. Ophir, T. Bdolah-Abram, and A. Ingber, 68.8% of vitiligo patients had SNHL, and they also had a much higher prevalence of the condition than the general population. The most typical type of audiogram discovered in vitiligo patients has a notch in it. Additionally, they observed that patients whose primary site of onset was mucosal involvement had considerably more normal hearing levels (p=0.004).[7]

In a study to ascertain the relationship between vitiligo and hearing loss, Sharifian M R came to conclusion that Leukoderma patients had a greater degree of sensorineural hearing loss than healthy individuals. It most likely happened as a result of the loss of epidermal pigmentary cells occurring concurrently with tissues and cells that accommodate pigments in inside part of ear, losing their defensive purpose. [8]

According to study conducted by Rosanna Mariangela Giaffredo Angrisani et al and others to determine, in case lesser cells producing pigments,had any impact on internal ear action, quantifying and investigating OAE, along with establishing the action of the medial olivocochlear (MOC) nervous circuit, by examining how OAE suppression affects those who have skin conditions like vitiligo ,found that 66.7% subjects with no acoustic abnormalities displayed limited (at 4 KHz) or absent OAE, implicating internal ear malfunction. In terms of the kind of vitiligo, the length of the disease, or age, did not show any likelihood that the relationship between changes in the occurrence of OAE (p = **.**648, p = **.**406, and p = **.**510).[9]

Study published in 2020, Abeir Osman Dabbous et al[10] examined the role of the cochlea in vitiligo. In this study, it was discovered that the majority (76%) of vitiligo patients had bilaterally normal hearing and that at most twenty-four percent among them had SNHL towards increasing frequencies when tested using PTA. In

comparison to the controls, patients with depigmentation presented with internal ear impairment, without any clinical signs or symptoms.

In a study conducted in 2017 by Eman Abd Elmohsin Dawoud et al to assess audiological and labyrinthine actions in subjects with and without depigmentary disorder, the results of PTA and OAE showed positive correlation connecting both groups of subjects. 10 cases of labyrinthitis , five cases of benign paroxysmal positional vertigo(BPPV) were present amongst fifty percentage of patients with Vitiligo.[11]

Vitiligo was identified as a systemic disease in a study by Torello Lotti, MD et al. According to their claims, cells producing pigments are also present in the internal part of ear, primarily in massively vascular parts of cochlea, which occur to be crucial for the production of the action potentials related to the bodily fluid that fills the membranous labyrinth of the inner ear needed to perceive sound normally.[12]

Researcher BN Akay et al and others found that acoustic impairment were present in thirty-seven percentage of subjects in a study done in 2010 to explore the symptoms, hereditary factors and lab reports along with relation of this depigmentary ailment with other IgG4-related systemic disease. Of these patients, forty-nine percent had unilateral minimal hearing loss (>30 dB) and the remaining fifty-one percent had bilateral hearing loss (>30 dB) over (2k–8k cycles/sec). [13]

R. Genedy, S. Assal, A. Gomaa, B. Almakkawy, and A. Elariny evaluated the ophthalmological and hearing impairments in leukoderma cases and found that there was a considerably huge preponderance of acoustic and ophthalmological impairment in cases in contrast to controls, but no noteworthy difference in sharp-sightedness. [14]

In a related study, D. S. Krupa Shankar et al found that age beyond thirty is substantially correlated with impaired audiological findings, retinal findings, increased Absolute Eosinophil Count and Postprandial Blood Sugar levels. They also found that the aetiopathogenesis of pigment disorder is complicated structural and integral in place of focal.[15]

According to a study by K. Aydogan et al, Vitiligo patients who showed changes in PTA are typically asymptomatic; therefore, we believe that neurophysiologists and biologists would be more interested in these changes than physicians. Postmortem histopathology analyses have confirmed investigations of anomalies in the audiological and brainstem auditory circuits in individuals. [16]

In a review, J. Mathews and B. N. Kumar found that autoimmunity is more frequently regarded as a cause of abrupt or progressive SNHL.[17]

Researcher Sundus Aslan et al have shown vestibular, sensory cells in base of cochlea are more delicate and can get damaged than those at the apex. Because SNHL was accompanied with a rise in hearing thresholds at increasing frequencies but showed a reduction in OAEs at 4 kHz, we also believed that the hearing loss was likely caused by an issue with the vestibular sensory cells located outside towards the base of the cochlea. [18]

Hoda Rahimi et al.[19] conducted a study in which incidence and characteristics of hearing loss were examined in individuals with depigmentation using PTA and OAE. In all of the evaluated intervals, neither PTA nor OAE exhibited a significant variation between the categories.

Vitiligo

Unknown as to its cause, vitiligo is a long-lasting skin condition marked by depigmentation. It is distinguished by symmetrical or asymmetrical white macules and patches that may gradually enlarge over time due to the degeneration of functional melanocytes. From less than 0.1% to more than 8% of people globally have the same condition.[1]The quality of life for the sufferer is severely compromised by vitiligo, which can appear at any moment throughout life. Typically, it shows no symptoms. Cells producing pigments are stem cells from the precursor cells. The lining epithelium of the eye and the internal ear are among few places where precursor cells (melanoblasts) migrate throughout development process, in addition to epithelium and hair follicles.[19] Therefore, it appears that the condition does not only affect the skin but also has the potential to worsen the onset of widespread syndromes, which are mostly linked to the immune system. Contrast to incidence of one-two percent of chronic illness in the overall public, studies have found about ten to fifteen percent of vitiligo subjects gain autoimmune disorders. Existence of immune complexes against cells producing pigment, further corroborate the immunological pathophysiology of the illness and characterises some portion of the systemic connection.

The cheek, palm, periorificial area, breasts, and upper arms are typical areas impacted by vitiligo, which appears as depigmented lesion or spots encircled by healthy skin.Depigmentation of the surrounding skin frequently occurs after leukotrichia. On rare occasions, melanocyte-containing internal organs and mucosae may be impacted. The diagnosis is made clinically.

According to the extent of the lesion, vitiligo has recently been divided into three categories: localised (containing particular types like trichrome, quadrichrome, and inflammatory vitiligo), generalised and universal. Segmental and nonsegmental vitiligo are additional categories. The segmental type completely or partially corresponds to a dermatological section (The area of skin supplied by cutaneous branches of a single cranial or spinal nerve). This is distinguished by sudden occurrences of white blotches also may even involve the depigmentation mechanism of the hair follicles. The likelihood of this variety being related to sympathetic nerve dysfunction is relatively high. The nonsegmental illness type appears to be more frequently linked to systemic involvement.

The first scientist to mention the existence of melanocytes in the inner ear was Alphonse Corti (1831). [2] In the human cochlea, melanocytes are found in large numbers, within stria vascularis(SV), central bony column, osseous spiral lamina(OSL), vestibular membrane.Melanocytes are particularly prevalent in areas that are adequately vascular, and they appear to play a key role in secretory or metabolic activities. Although the specific functions of melanin and it are yet to be understood, it is quite likely that they have an autonomic activity in the internal ear.

According to Savin (1965)[3] ,cells that produce pigments are completely or to limited extent attached to capillary walls, where transfer of *substances*, matters takes place .According to this scholar, melanin is what allows materials to move between one end onto the other, preserving the balance equilibrium of the biological epithelium. Additionally, given the fact that hearing is regularly affected in systemically involved diseases affecting coloured structures (such as the eyeballs, skin, or hairs), including the Vogt-Koyanagi and Waardenburg syndromes, melanin

may play a crucial role in the interal ear. Numerous pigment producing cells exist found in the internal ear, particularly in the cochlea's basal region, which processes high sound frequencies and is most frequently damaged by ototoxic drugs and prolonged exposure to loud noises.

The maintenance of the SV and normal function of internal ear, the generation of EP, as well as the keeping of the cation as well as liquid differential in between interstitial fluid and the extracellular fluid is just a few among the many roles of these cells in internal ear ,that are crucial for hair cell survival. [18] Numerous authors have also claimed that the cochlea can be shielded from stressors, particularly loud noise, by the inner ear's melanocytes.[19] It is clear that pigment producing cells are prevalent in other areas elsewhere in periphery of acoustic pathway, since anomalies there in brain have been identified across both animals and people suffering from depigmentation disorders.

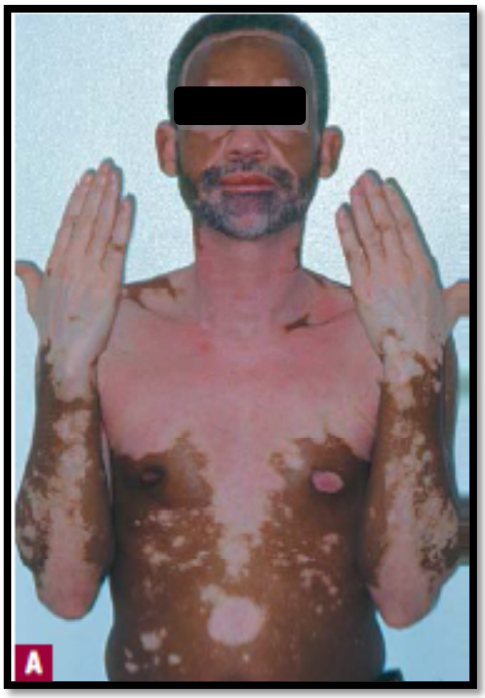

Fig 1. Generalised vitilgo

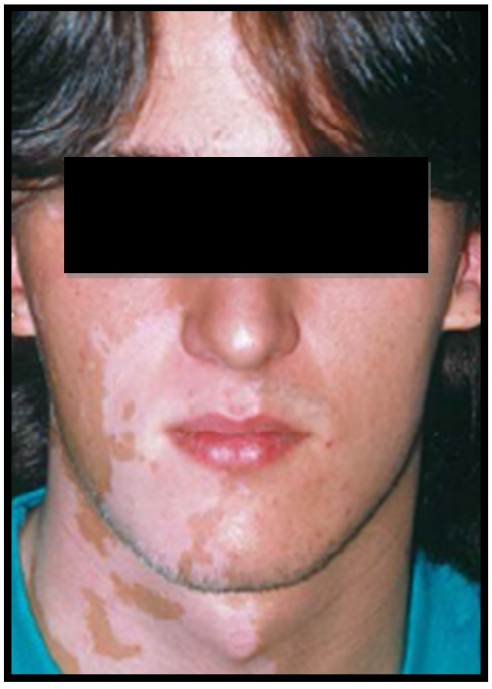

Fig 2. Segmental vitilgo

Vitiligo Area Scoring Index (VASI)

Hamzavi et al.[20] developed the VASI , which was the quantifiable measure that had been developed from the widely used PASI measure for psoriatic evaluation, in attempt to determine the area and severity of skin diseases. A technique that integrates contributions from every body component is used to determine literally the entire body VASI (feasible extent, zero – hundred)

VASI= Sum of ([Hand Units]$_{\text{Entire Body Site}}$ × [Underlying Residual Depigmentation])[20]

Overall background percentage with depigmentation present at every anatomical area is calculated utilizing this as a reference. One hand unit is equivalent with about one percent on average of the maximum surface area of the body and also is characterized as the area of the hand palm surface plus the volar surface of all the digits. The five unique prestigious portions of the body are the trunk, lower limbs (excluding the feet), upper limbs (with the exception of the hands), and feet. The upper extremities and axilla are included, and the lower extremities ,pelvis and groin regions have also been represented. Amount of residual colouring is expressed using one of the following percentages: 0, 10%, 25%, 50%, 75%, 90%, or 100%.At the hundredth level of depigmentation, there is no pigment; at the ninetieth level, there are specks of pigment; at the seventy-fifth level, the hypopigmented and coloured seem to be comparable; somewhere at twenty-fifth level, pigmented region is more than the depigmented area; and at the tenth level, there are no areas of hypopigmentation seen.

VASI SCORE	INTERPRETATION
-50	Very much worse
-50 to -25	Much worse
-25 to -10	Worse
-10 to 0	Minimally worse
0 to +10	Minimally improved
+10 to 25	Improved
25 to 50	Much improved
+50	Very much improved

Table 1: VASI Score

Treatment algorithm for Vitiligo[1]

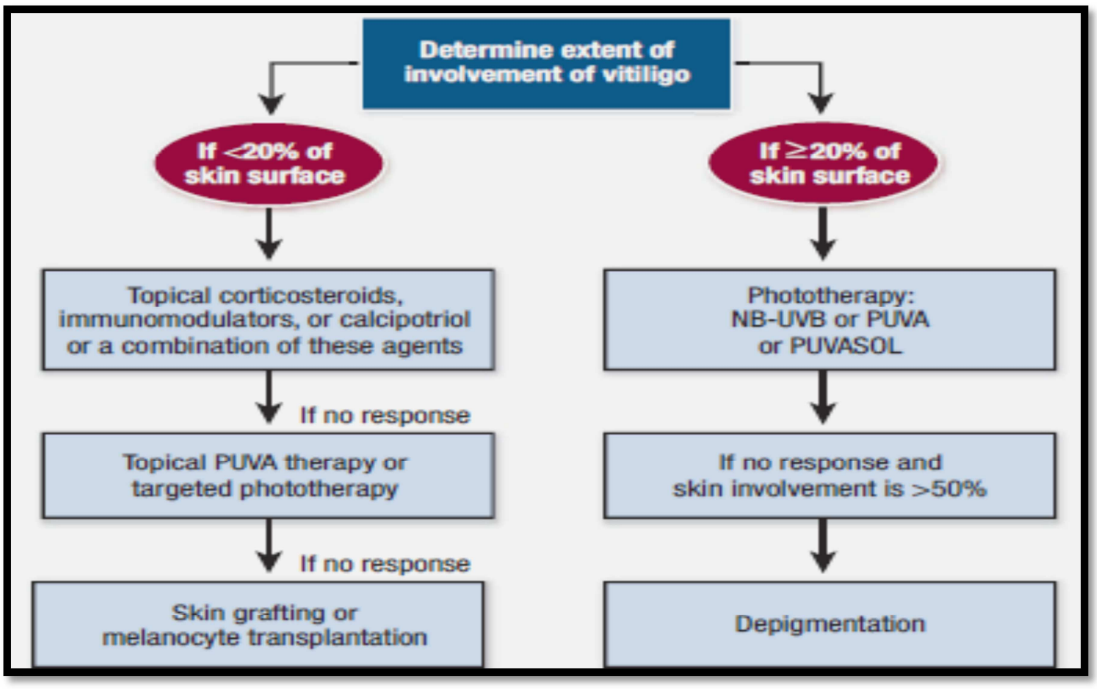

Fig 3:Vitiligo treatment algorithm

Treatments for Vitiligo

	TOPICAL	PHYSICAL	SYSTEMIC	SURGICAL
First line	Corticosteroids Calcineurin inhibitors	Ultraviolet B (narrow band) Systemic psoralen and ultraviolet A light		
Second line	Calcipotriol	Topical psoralen and ultra-violet A light Excimer laser	Corticosteroids (pulse therapy)	Grafting Melanocyte transplant

Fig 4:Vitiligo treatment

Inner Ear

The labyrinth and its related sensory structures, which are responsible for balance and auditory function, are located in the petrous portion of the temporal bone. The membranous labyrinth, which is located inside the bony labyrinth, can be divided into three areas that are unitarily connected: the pars superior, also known as the vestibular labyrinth except for the saccule, the pars inferior, also known as the cochlea and saccule, and the endolymphatic duct and sac.[22]

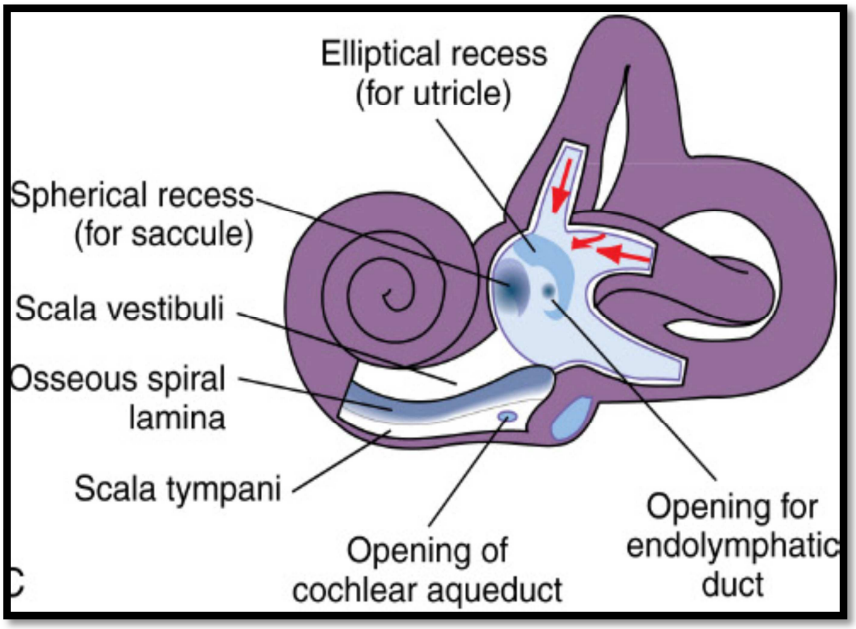

Fig 5:A cut section of bony labyrinth

Cochlea

The auditory component of the labyrinth, the cochlear duct, measures around 35 millimetres. The cochlear duct and related sensory organs resemble a 2.5–2.75-turn snail shell with a spiral shape. The endolymph-containing scala media or cochlear duct is triangular in shape transversely. The membrane extending from the bony shelf of the internal ear to the outer wall and supporting the organ of Corti and Reissner membrane, which constitute the triangle's superior and horizontal limbs, respectively, are produced by the SV with helical ligaments, which also forms the triangle's perpendicular aspect. The fluid in the scala vestibuli and scala tympani is perilymph, while the fluid that fills the cochlear duct is endolymph. The helicotrema near the tip of the cochlea serves as a communication channel for the perilymph of the two scalae. Significantly, the basilar membrane, which is one of the cochlear duct's structures, has a morphologic gradient, meaning that its width is narrowest at the base and broadest at the apex. In the organ of Corti, the spiral ligament and epithelial components even exhibit a morphologic gradient from base to apex. By introducing a specific tone or frequency to the sensory organ of the inner ear, this morphologic gradient, to an extent, controls the position of maximum stimulation of the basilar membrane and inner hair cells. In this way, the frequency scale is organised to spread out in an orderly form throughout the rest of membrane extending from the bony shelf of the internal ear to the outer wall and supporting the organ of Corti, with higher harmonics somewhere at base and low vibrations at the top apex.

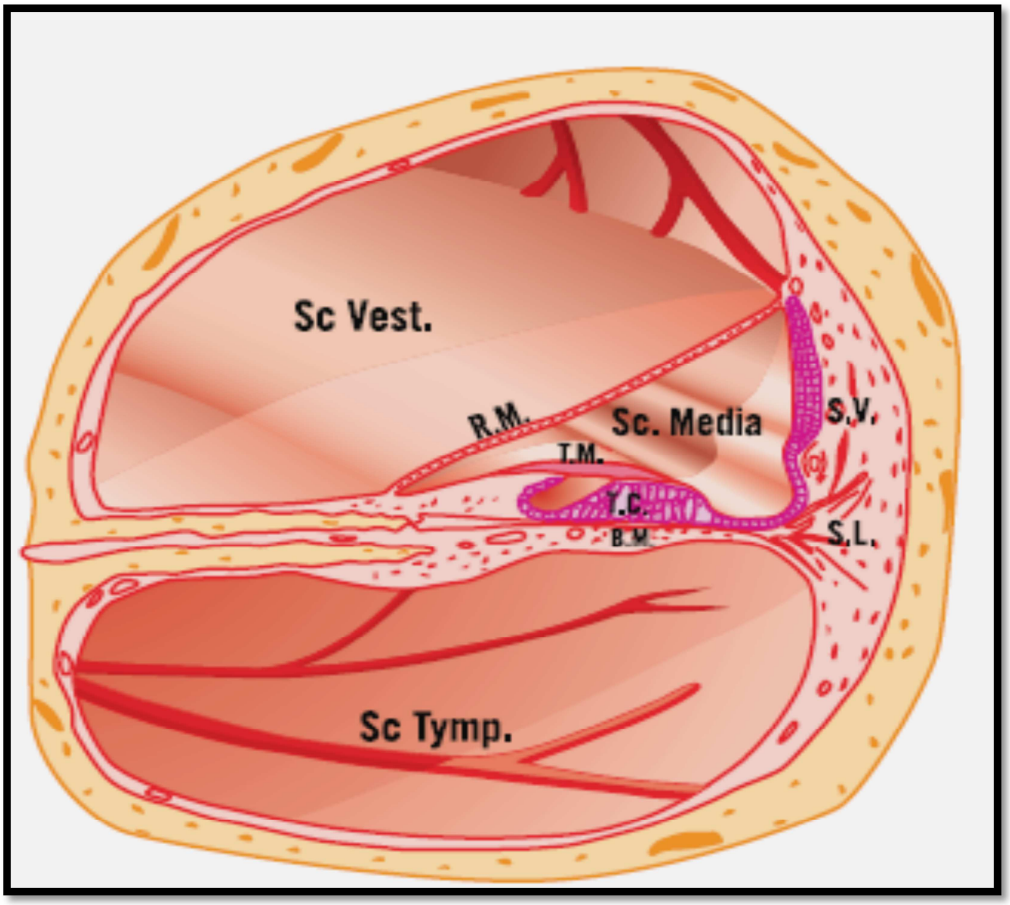

Fig 6: Section (diagrammatic) through the cochlea.[22]

The ciliated extremities of the hair cells protrude into or close to a covering structure, the tectorial membrane, in the advanced sensory organ known as the organ of Corti. It also comprises supporting cells that rest on the basilar membrane and inner and outer hair cells. The stereocilia, which typically number between 100 and 150 per cell, project through the cuticular plate, where the uppermost parts of hair cells are anchored. Actin-based protrusions of the exterior hair cells establish contact with the tectorial membrane, whilst the ones present over inner hair cells periodically drift loosely in the endolymphatic region underneath the membrane. One row of internal hair cells and three to five rows of outside hair cells are present. 30,000 spiral ganglion cells innervate human organ of Corti. Across the entire extent of internal ear

hearing apparatus, the spiral neural structures appear as clusters of neural structure cells. The majority of spiral ganglion neurons, 90–95 percent, are type I neurons, which are large, myelinated, and directly project a single dendrite to an inner hair cell. The cochlear nucleus complex, which includes the dorsal cochlear nucleus along with the anteroventral and posteroventral divisions of the ventral cochlear nucleus, receives axons from spiral neural structure cells.

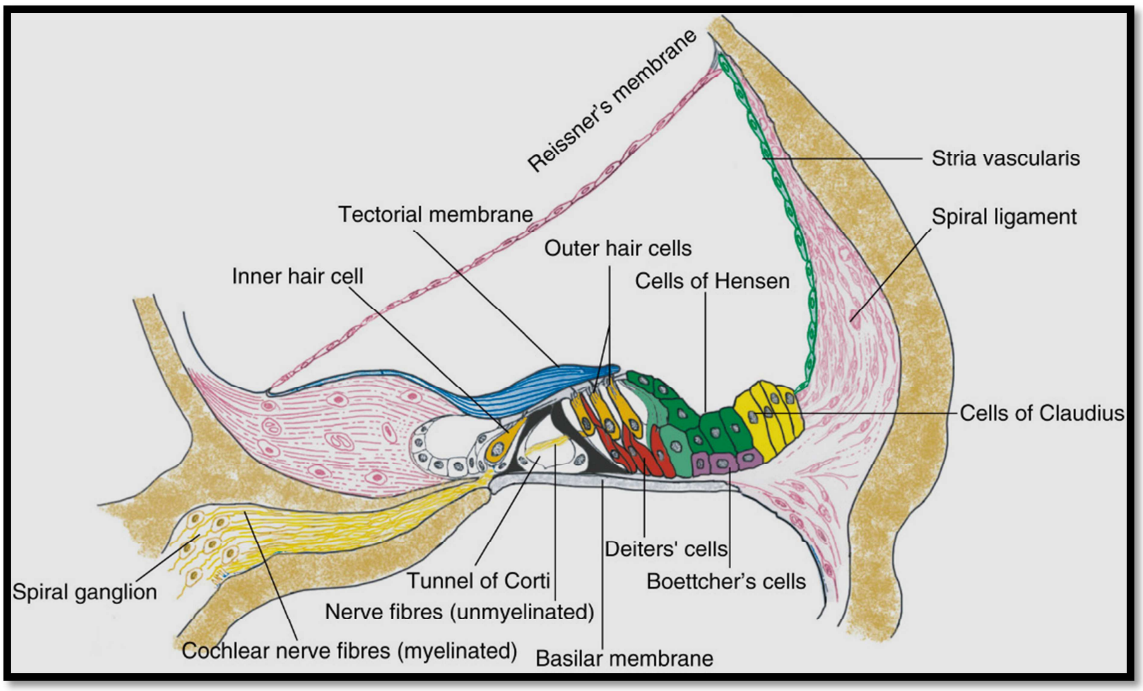

Fig 7: Structure of organ of Corti

In addition, the stria vascularis (SV) is a strip of tissue that runs the full length of the scala media and is between 150 and 300 m wide (depending on the species and locality). It is responsible for creating and maintaining both the EP and each high endolymphatic K+ concentration.

Three different cell types make up the SV, which surrounds a capillary network:

- The endolymphatic compartment's marginal cells
- Intermediary cells is an incredibly discontinuous layer completely surrounding the body of epithelium at intervals.
- Basal cells that divide the spiral ligament beneath the SV from it.

The SV is known to have the greatest rate of aerobic metabolism (i.e., where gas is used to produce energy from carbohydrates) throughout the entire body, most likely because of the significant energy demand caused by the amount of active particle transport that occurs within this tissue. Despite being responsible for the circulation of chemical element potassium (K), the role of basal and intermediate cells in strial function is poorly described . Because the potassium (K) in endolymph derives mostly from perilymph rather than blood, the helical ligaments as well as the chief cells of the cochlea are considered most likely the locations of circulation. A failure in EP production could also result from a pause in K circulation. The cochlear amplifier is driven by an energy source, or "battery," given by the EP. Any loss of EP results, as would be predicted, in a very significant impairment like hearing loss.

The intermediate cells are a subset of melanocytes, or cells that contain melanin, which grow from neural crest cells as the body develops. They are completely encompassed by the stria's corpus and interdigitate with an alternative cell type. When paired with proteins which metabolize and eliminate aerobic wastes, they include a range of enzymes that modify the production of energy from diverse materials, which include triglycerides. Melanin will also function as a free radical scavenger. These biological traits suggest that one role of Intermediary cells within

SV could be safeguarding stria amidst pressure alongside with supplying renewable resources of energy to continue functioning throughout instances of ischemic injury. The apical side invaginations of peripheral layers as well as it's extremely high amounts of K+ transporter protein Kir4.1 in the plasma membranes facing the living thing areas are characteristics of the intermediate cells. Since a strong positive potential is created in this area, EP is thought to be produced by the rapid, intense transit of K+ through Kir4.1 across the intermediate cytomembrane and into the intercellular space, followed by the immediate uptake of K+ by marginal cells. In order to prevent the potential from dissipating into the perilymph in the interconnecting gaps within spiral ligament, the basement layer of cells acts as an insulating shield.

Mechanism of hearing

The pinna gathers an ambient sound signal, which travels through the external meatus and strikes the tympanic membrane. Through a series of ossicles connected to the membrane, the membrane's vibrations are conveyed. The basilar membrane is moved by the auditory ossicles because the pressure of the labyrinthine fluids fluctuates. The Corti organ's hair cells are stimulated by this. These hair cells serve as transducers, converting mechanical energy into electrical impulses and causing them to move down the auditory nerve. As a result, the hearing mechanism can be broadly classified into:

1. Sound transmission by mechanical means (conductive apparatus).
1. Converting kinetic potential into electric power pulses (sensory system of cochlea).
2. The physiological process of cerebral electrical currents (neural pathways).

The basilar membrane and tectorial membrane are affected by movements of the auditory ossicles, which are transferred to the cochlear fluids and then to the hair cells. Cochlear microphonics, which cause the electrical discharge, are produced by hair cell deformation. Depending on its frequency, an acoustic wave will have the greatest amplitude at a certain location on membrane extending from the bony shelf of the internal ear to the outer wall and supporting the organ of Corti which further help set in motion of that area (travelling wave theory of Von Bekesy). The cochlea's basal turn is where higher frequencies are represented, and its apex is where gradually decreasing frequencies are squared off.

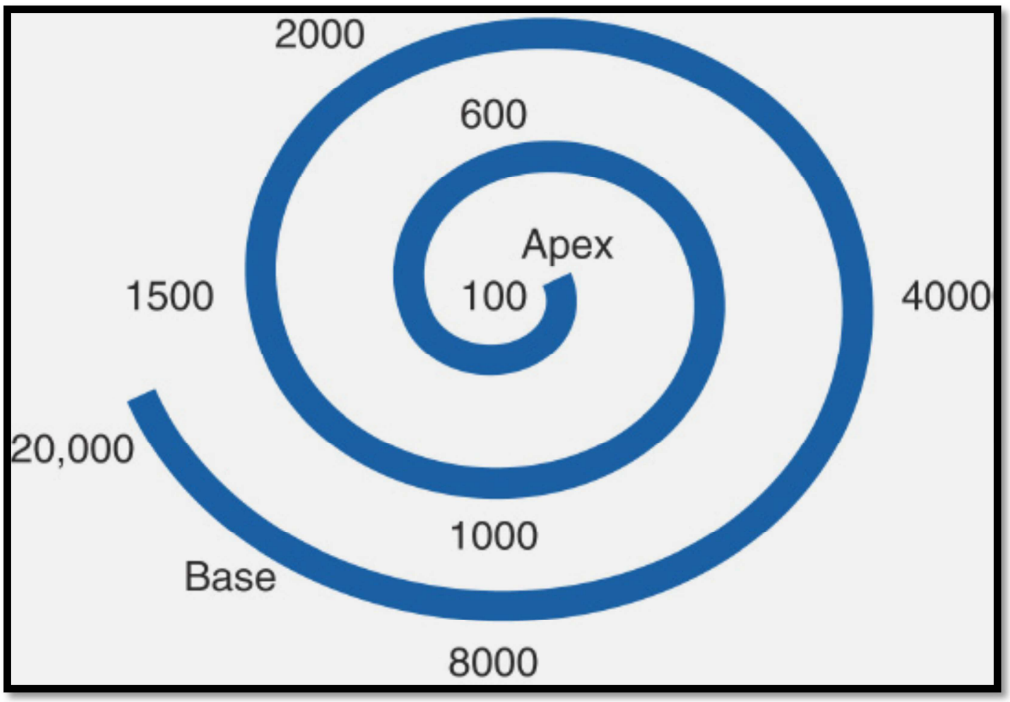

Fig 8: Frequency localization in the cochlea

Theories of hearing

- Place theory/ resonance theory (Helmholtz, 1857)

Place theory is a possible hearing theory that contends that where each frequency component vibrates the basilar membrane,which determines where we perceive sound and how loud it is.

- Frequency theory / telephone theory (Rutherford, 1886)

Due to each individual sine wave's displacement of the perilymph and endolymph fluid in the cochlea, the basilar membrane oscillates up and down. The cochlea's hair cells become excited as a result of the displacement of membrane. Once a wave enters the cochlea, its frequency and intensity are particularly sensitive to a particular nerve, causing that nerve to fire electric charges. That nerve cannot send another message until the first one has been sent and, consequently, until the nerve has recovered. Each nerve fibre in the auditory nerve sends this information to the auditory region, where it is put together and used to understand and process the audio signal.

According to frequency theory, sound waves are actually vibrations on the auditory nerve that are perceived by humans as a particular sound. The frequencies of these vibrations are the same as the frequencies of sound waves that travel from the ear back to the brain. However, the fact that an individual could perceive auditory vibrations up to 20KHz however nerve axons could never activate some of these levels, hence frequency hypothesis seemed to have a significant downside.

- Volley theory

According to the "volley theory," several neurons may fire at the same time, combining to create noises that have the same frequency as the initial sound stimulus. After extensive analysis, found that while period synchronisation is precise in around 5000 cycles per second, volley theory is insufficient to explain all of the frequencies that we typically hear. Sequential firing using groups of two to five fibres, with a limit of one kHz for each fibre.

More than 5 Kilocycles per Second: Place Theory

Frequency theory states 400 cycles per second.

Volley theory: 400 - 5000 cycles per second

- Bekesy's travelling wave theory

From the cochlea's base turn up to its apex, the basilar membrane vibrates as a result of an auditory stimulus. The vibration gradually grows in amplitude as it moves and reaches a maximum before wearing off. The point of maximum amplitude is used to compute auditory frequency. Low frequency auditory stimuli have their maximum amplitude near the cochlear apex, while high frequency auditory stimuli create waves with their maximum amplitude close to the basal turn of the cochlea. In 1961, Georg von Bekesy won the Nobel Prize for his travelling wave theory.

Auditory pathway

Sensory receptor cells receive stimulation from the bipolar cells of the central nervous systems. The auditory nerve, that transmits messages to the ventral and dorsal cochlear nuclei, is formed when the core axons of these cells come together. The

medial geniculate body, inferior colliculus, lateral lemniscus, superior olivary nucleus, and ultimately the acoustic center of the temporal lobe are the next locations for every crossed and uncrossed fibre after that.

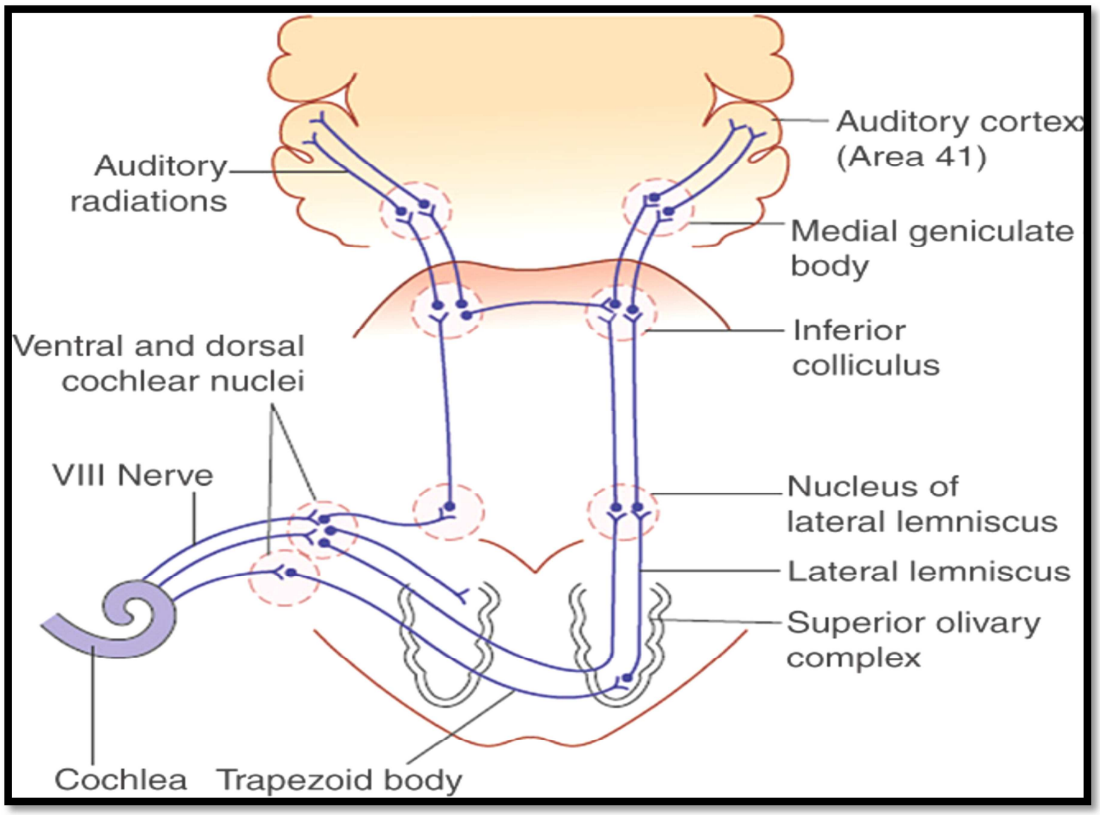

Fig 9: Auditory Pathway

Pure Tone Audiometry (PTA)

An audiometer is a piece of equipment that produces pure tones whose intensities may be changed in stages of 5 dB(Decibel). For tones at two hundred and fifty, five hundred, one thousand, two thousand, and four thousand Hz, skeletal transmission limits are often measured, whereas air conduction thresholds are established for tunes at one hundred and twenty-five,two hundred and fifty, five hundred, one thousand, two thousand, and four thousand Hz. How much intensity

must be increased above normal will determine how impaired your hearing will be during that spectrum. It is charted using an audiogram, a type of graph.

Grade of impairment*	Corresponding audiometric ISO value**	Performance	Recommendations
0 - No impairment	25 dB or better (better ear)	No or very slight hearing problems. Able to hear whispers.	
1 - Slight impairment	26-40 dB (better ear)	Able to hear and repeat words spoken in normal voice at 1 metre.	Counselling. Hearing aids may be needed.
2 - Moderate impairment	41-60 dB (better ear)	Able to hear and repeat words spoken in raised voice at 1 metre.	Hearing aids usually recommended.
3 - Severe impairment	61-80 dB (better ear)	Able to hear some words when shouted into better ear.	Hearing aids needed. If no hearing aids available, lip-reading and signing should be taught.
4 - Profound impairment including deafness	81 dB or greater (better ear)	Unable to hear and understand even a shouted voice.	Hearing aids may help understanding words. Additional rehabilitation needed. Lip-reading and sometimes signing essential.

* Grades 2, 3 and 4 are classified as disabling hearing impairment (for children, it starts at 31 dB)
** The audiometric ISO values are averages of values at 500, 1000, 2000, 4000 Hz.

Fig 10:World Health Organisation(WHO) Grades of Hearing Impairment

METHODOLOGY

Study design: Observational study

Study period: One year

Study population: Patients diagnosed with vitiligo willing to undergo ENT, dermatological examination and Pure Tone Audiometry.

Sample Size: 71 cases.

Ethical Clearance: Obtained from the Institutional Ethical Committee

Inclusion criteria: All subjects between the ages of 18 and 60

1. Diagnosed cases of vitiligo who are willing to undergo ENT, dermatological examination and Pure Tone Audiometry.

Exclusion criteria:

1. Patients suffering from hypopigmentary disorders other than vitiligo.
2. Patients suffering from external, middle and inner ear disorders
3. Traumatic head injury
4. Co-existent metabolic disorders such as diabetes mellitus, hyperlipidemias, thyroid dysfunction and renal disorders
5. Co-existent Hypertension
6. Exposure to noise
7. Ototoxic medications different from that used in the treatment of vitiligo.

SAMPLE SIZE: The minimum sample size

formula –

$$n = \frac{Ny}{[(N-1)\,E^2 + y]}$$

$y = Z(c/100)^2\, p(100-p)$ or $Z(c/100)^2\, p(1-p)$

p is proportion or prevalence

E=margin of error

Given, p = 68%, Taking E=15% of p =10.2

N=450

For a=5% $Za_{/2}$ =1.96

The sample size obtained is 71.

Procedure:

- Following the patient's informed consent, demographic information about all patients will be recorded on a predesigned proforma. A thorough clinical history will be obtained regarding the duration of vitiligo, treatment received, and whether it has affected other family members. Additionally, a history of hearing loss will be obtained.
- All patients will then undergo a clinical examination that includes a general physical examination, examination of the ear, nose, and throat, examination of the skin, and dermatological examination VASI.
- Tuning fork tests and otoscopic inspection will be done.
- A MAICO MA53TM audiometer will be used for the hearing evaluation.

- In a soundproof environment, the Pure Tone audiometry hearing threshold will be assessed at vibratory spectrum ranging from 125 to 8000 Hz for air conduction and 250 to 4000 Hz for bone conduction.

Interpretation of Audiogram

This study uses the WHO categorization system to categorise hearing loss. It falls under the following categories:

Table 2: WHO categorization of auditory impairment

Severity of Auditory impairment	Auditory Threshold (PTA)
Mild	26 to 40 decibel
Moderate	41 to 55 decibel
Moderate to Severe	56 to 70 decibel
Severe	71 to 90 decibel
Profound	> 91 decibel

Statistical analysis:

"Data was analysed 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"

For all the tests the value of p less than 5% (0.05) considered significant

RESULTS

The independent t test is used to compare all continuous variables, which are all provided as the average Standard deviation. The Chi square test is employed to find contrast among collected variables that are presented as percentages.

Seventy one subjects within the age group of 18 to 60 who had verified vitiligo presented to the dermatology and ENT OPD at KLES Dr. Prabhakar Kore Hospital and Medical Research Center and Charitable Hospital, Belgaum.

1. **Sex distribution**

In 71 patients who were involved in this study ,a male predominance was noted in the study

Gender	Number	Percentage (%)
Female	34	47.89
Male	37	52.11
Total	71	100.00

Table 3: Gender Distribution of the sample

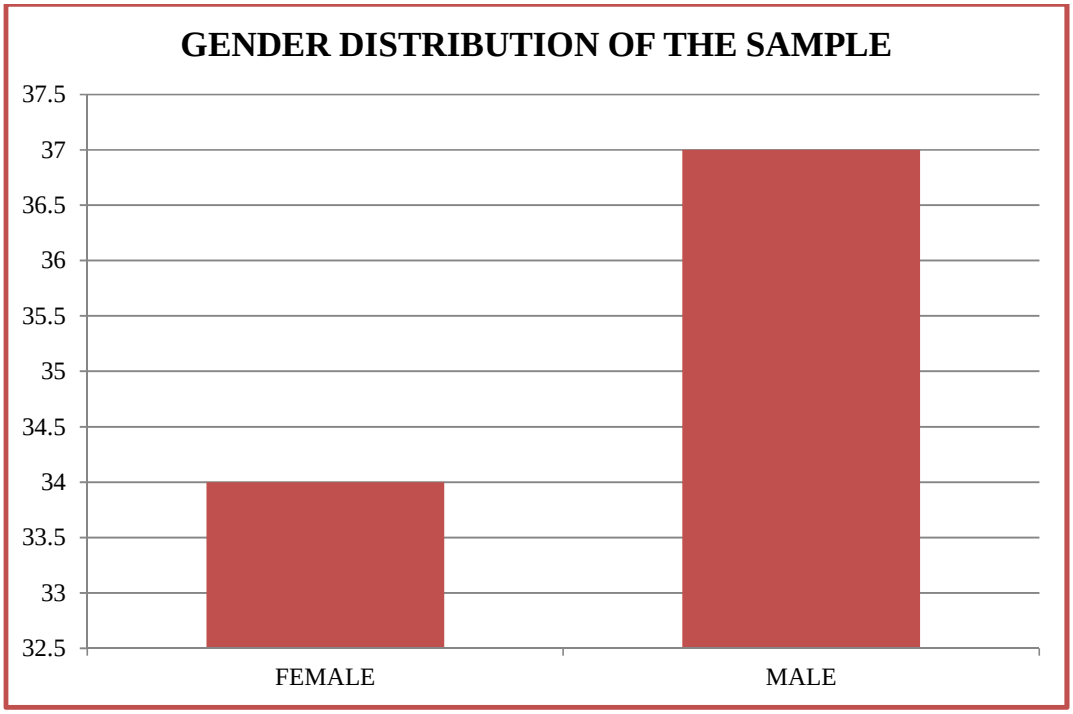

Figure 11: Gender Distribution of the sample

2. Age wise distribution of patients

In this study ,vitiligo patients within the age group 18-60 years were included. There has been a surge mostly in age demographics of 20-29 and 40-49 years.

AGE	NUMBER	%
< 20	2	2.82
20 - 29	30	42.25
30 - 39	14	19.72
40 - 49	17	23.94
50 - 59	8	11.27
TOTAL	71	100.00

Table 4: Age distribution of the sample

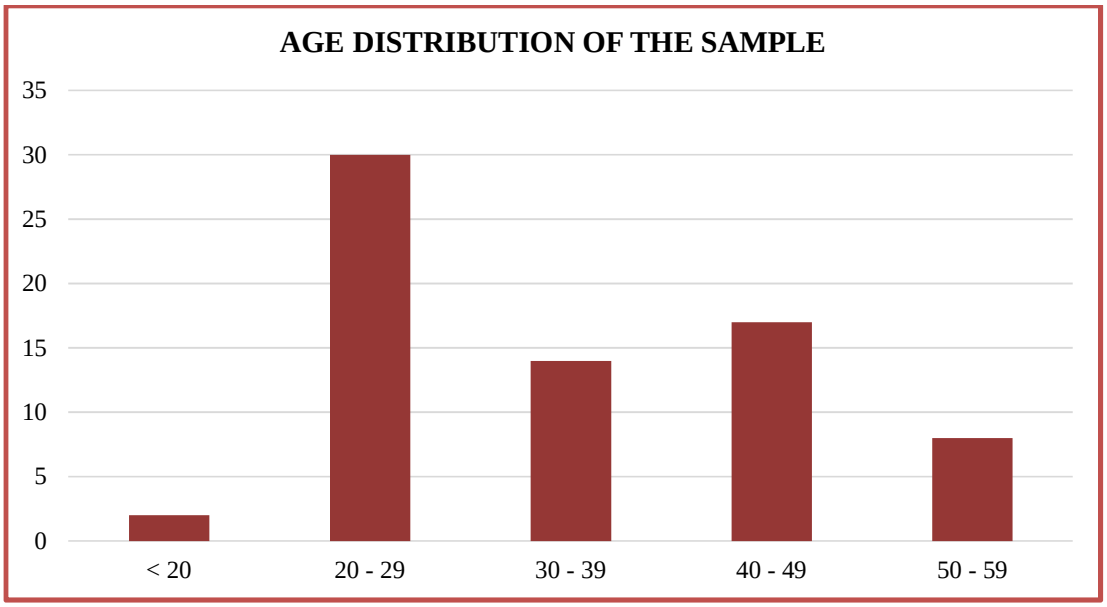

Figure 12: Age Distribution of the sample

Thirty-nine of the Seventy-one individuals who were a part of the study were above the age of Thirty, and the majority of them were men.

	AGE < 30		AGE ≥ 30	
GENDER	**NUMBER**	**%**	**NUMBER**	**%**
FEMALE	18	56.25	16	41.03
MALE	14	43.75	23	58.97
TOTAL	32	100.00	39	100.00

Table 5: Age distribution of the sample with respect to gender

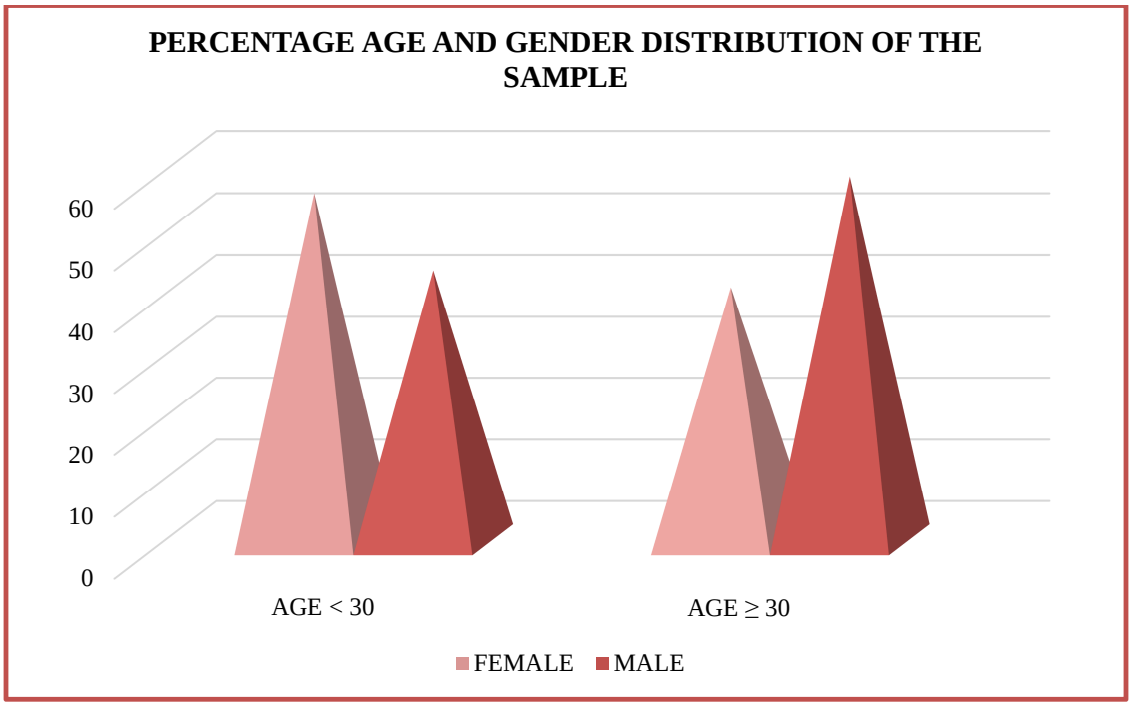

Figure 13: Age distribution of the sample with respect to gender

3. Type of Hearing loss seen in this study

In this study, out of 71 individuals with vitiligo, 36 have been observed to exhibit healthy auditory function, 35 have been identified to have bilateral sensorineural hearing impairment, and neither one were observed to have unilateral sensorineural hearing impairment or conductive auditory damage.

Out of these 35 patients, majority (26 patients) was found to have Bilateral mild SNHL,5 were found to have Bilateral moderate SNHL, and Bilateral severe SNHL was seen in 3 patients.

Hearing loss	High frequency	Normal	Total
B/l mild SNHL	26	0	26
B/l moderate SNHL	5	0	5
B/l severe SNHL	3	0	3
Normal	0	36	36
R-mild SNHL, L-moderate SNHL	1	0	1
Total	35	36	71

Table 6: Type of hearing loss

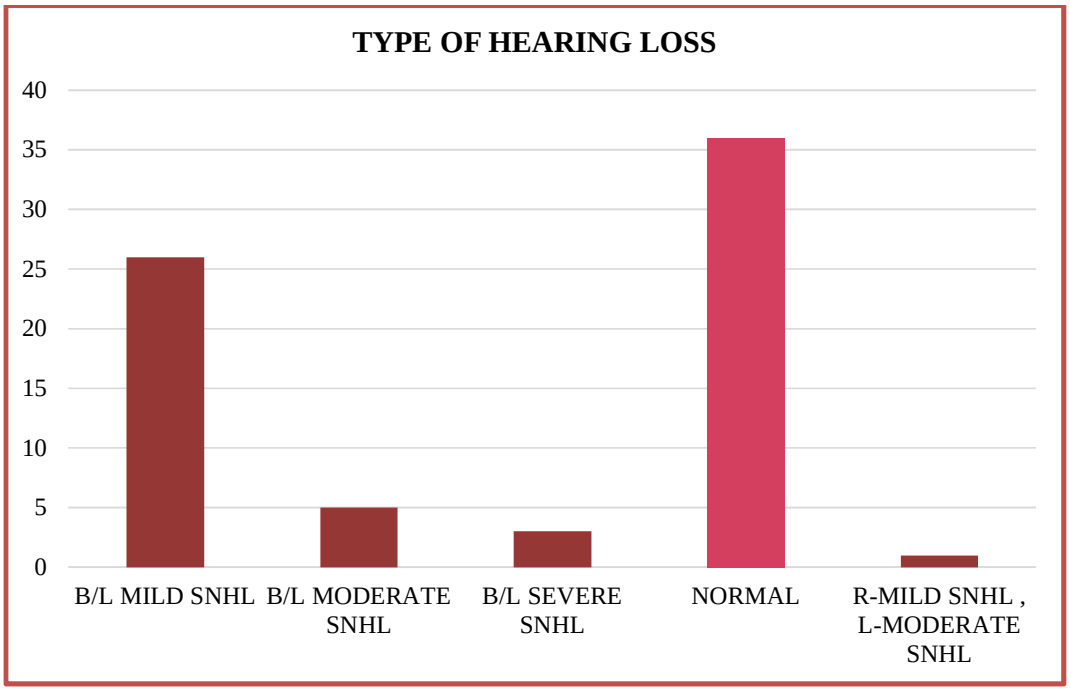

Figure 14: Type of hearing loss

Out of the 35 vitiligo patients in whom bilateral SNHL was recorded,22 of them were found to be above 30 years of age.

	AGE < 30		AGE ≥ 30	
Hearing loss	Number	%	Number	%
Normal	19	59.38	17	43.59
SNHL	13	40.63	22	56.41
Total	32	100.00	39	100.00

Table 7: Relation between age and hearing function

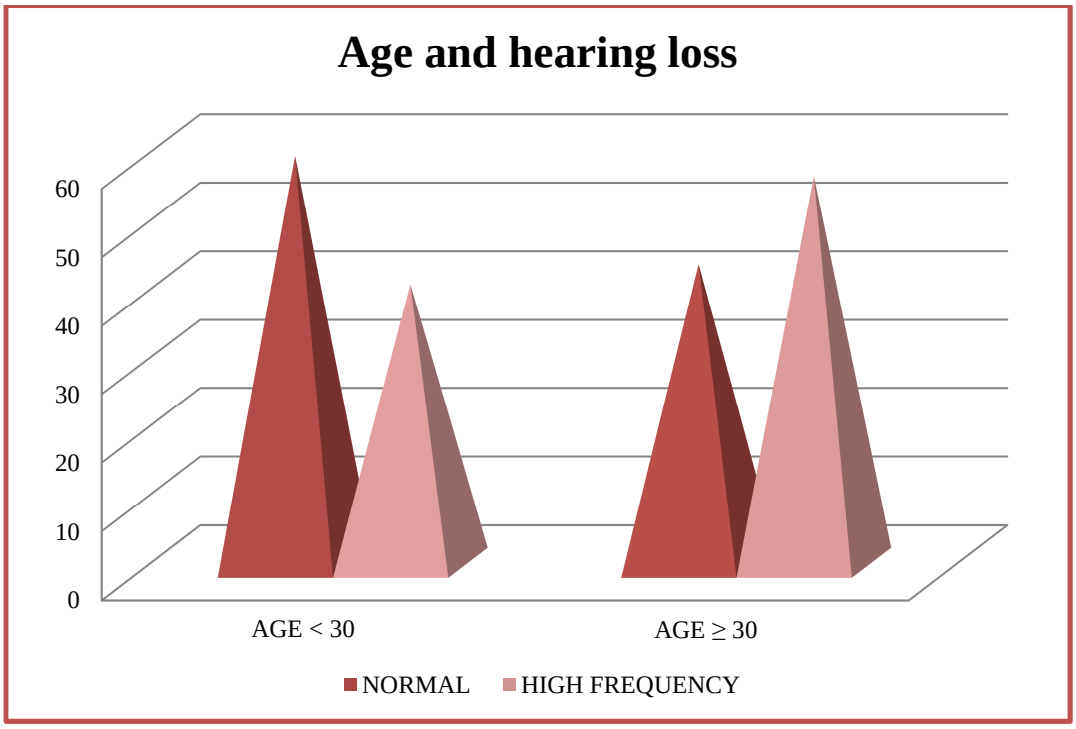

Figure 15 :Relation between age and hearing function

4. Correlation Between Hearing loss and Vitiligo

The Relevance of hearing loss and Vitiligo was calculated and comparison was carried between right and left ear which showed statistically no significant hearing loss bilaterally.

HEARING LOSS									
LEFT EAR				RIGHT EAR					
Average	Standard Deviation	Minimum	Maximum	Average	Standard Deviation	Minimum	Maximum	p Value	Inference
26.93	15.81	15	90	28.33	15.68	15	80	0.5974	NS

Table 8: Correlation between Hearing loss and vitiligo

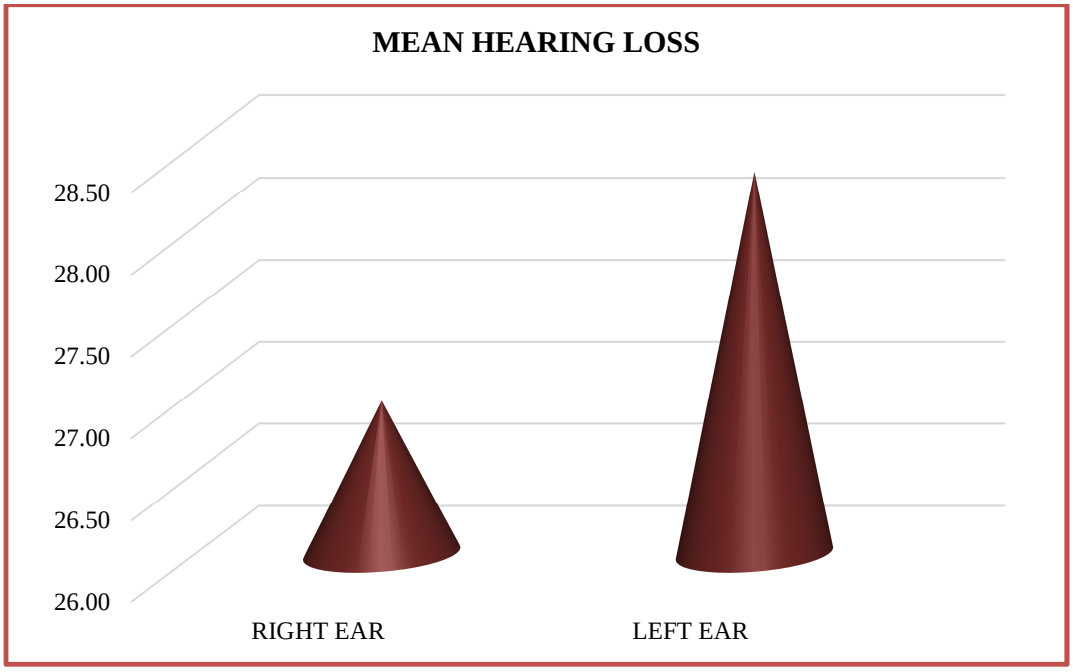

Figure 16: Correlation between Hearing loss and vitiligo

5. Timespan of disease

According to the current study's analysis on the time span of Vitiligo,out of 71 patients 44 patients had history of Vitiligo since less than 20 years.

	DURATION < 20		DURATION ≥ 20	
GENDER	**NUMBER**	**%**	**NUMBER**	**%**
FEMALE	23	52.27	11	40.74
MALE	21	47.73	16	59.26
TOTAL	44	100.00	27	100.00

Table 9: Duration of Vitiligo

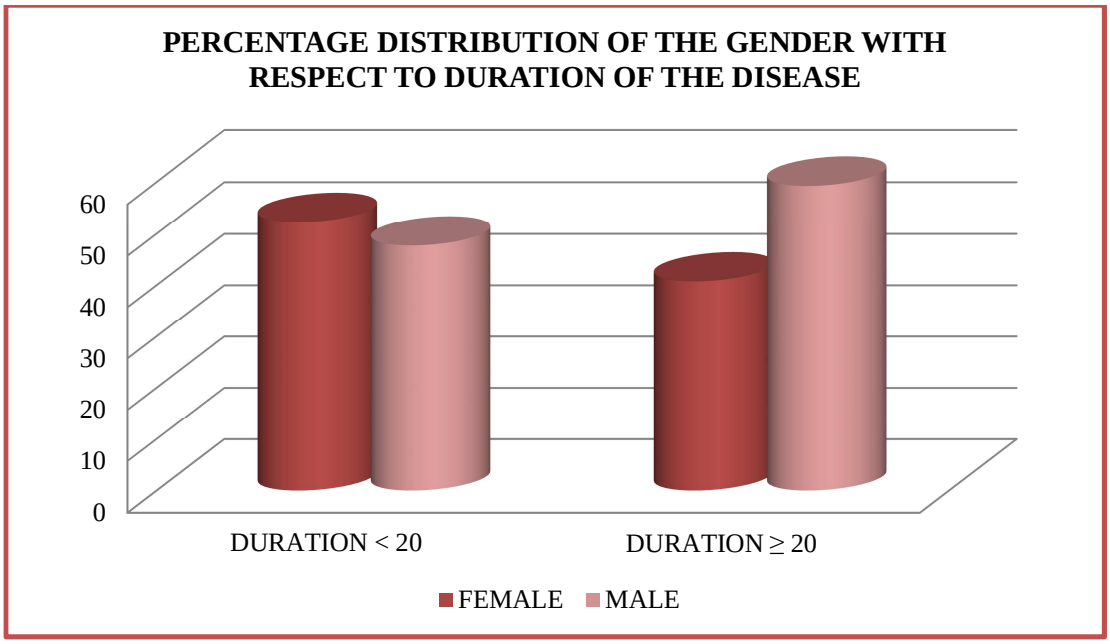

Figure 17: Duration of Vitiligo with respect to gender distribution

6. Duration of Vitiligo and relationship with Hearing

The duration of disease was noted to look for any relevance with hearing loss and a mean value was created there was no positive relationship between duration in Vitiligo and hearing impairment.

	DURATION < 20		DURATION ≥ 20	
HEARING LOSS	NUMBER	%	NUMBER	%
NORMAL	25	56.82	11	40.74
SNHL	19	43.18	16	59.26
TOTAL	44	100.00	27	100.00

Table 10: Relationship of time span of illness and auditory impairment

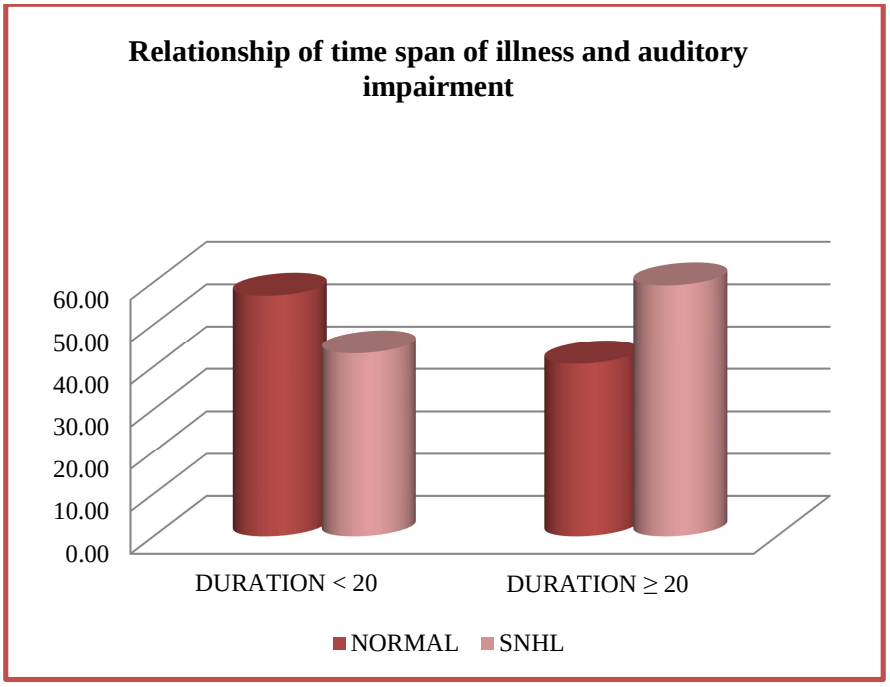

Figure 18: Relationship of time span of illness and auditory impairment

	Time span < 20		Time span ≥ 20	
HEARING LOSS	NUMBER	%	NUMBER	%
NORMAL	25	56.82	11	40.74
B/L MILD SNHL	14	31.82	12	44.44
B/L MODERATE SNHL	3	6.82	2	7.41
B/L SEVERE SNHL	2	4.55	1	3.70
R-MILD SNHL, L-MODERATE SNHL	0	0.00	1	3.70
TOTAL	44	100.00	27	100.00

Table 11: Relation between time span of disease with type of auditory impairment

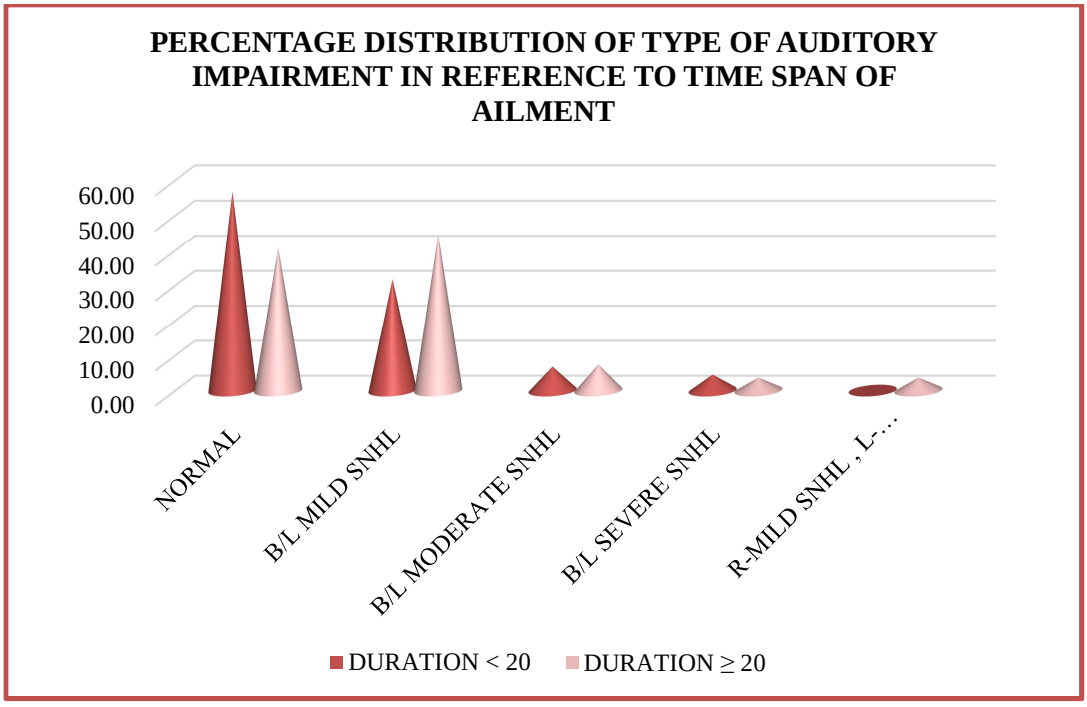

Figure 19: Relation between time span of ailment with type of auditory impairment

7. VASI Score and Relationship with Hearing

In 71 patients, VASI index computed for each patient to look for any correlation with hearing loss and a mean value was created, which showed no positive relationship between the same.

	NORMAL HEARING (N=36)				HEARING LOSS (N=37)					
	Average	**Standard Deviation**	**Minimum**	**Maximum**	**Average**	**Standard Deviation**	**Minimum**	**Maximum**	**P Value**	**Inference**
Vasi Score	8.38	5.97	0.75	21.4	9.24	6.29	0.9	21.4	0.5552	Ns

Table 12: Correlation between VASI score Hearing loss

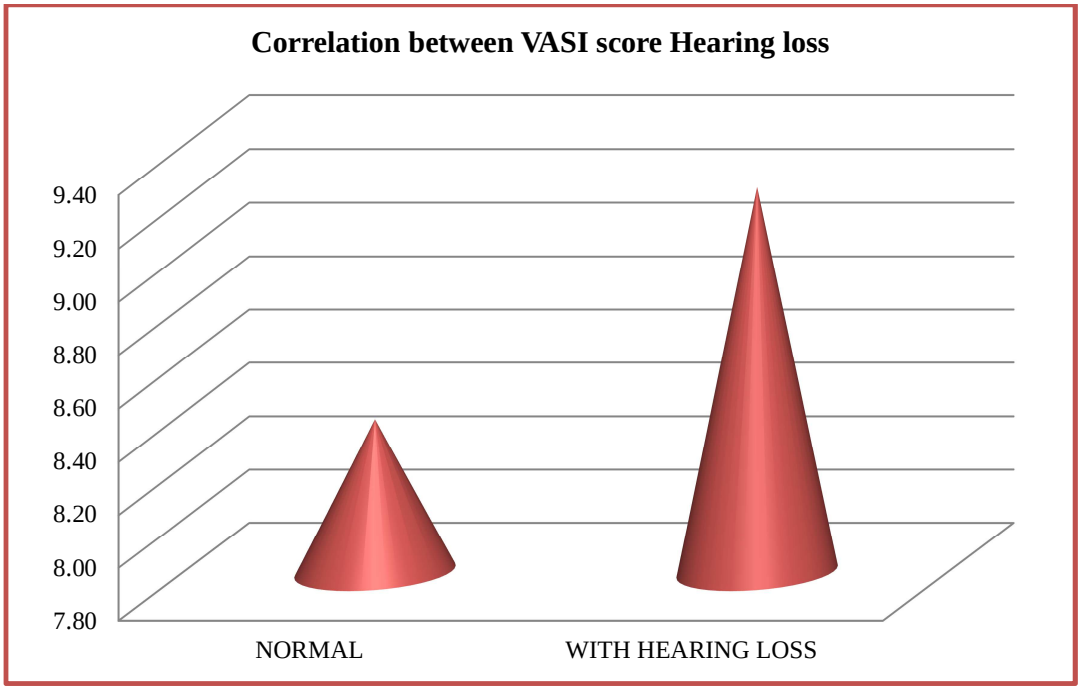

Figure 20: Correlation between VASI score Hearing loss

DISCUSSION

Several studies have been conducted to evaluate SNHL in vitiligo patients. According to certain research, hearing loss and the signs of depigmentation in vitiligo are both caused by a systemic mechanism. While later investigations discovered that hearing loss among individuals with vitiligo manifested in the higher frequencies, the initial studies examining hearing performance among vitiligo patients based on audiology tests did not indicate a significantly greater prevalence of SNHL. [2]

Seventy one individuals with vitiligo were included in the current study, of whom 34 patients (47.89%) and 37 patients (52.11%) were male and female, respectively. There was a noticeable male predominance. Similarly, a research by Neel Prabha et al, which comprised 52 patients, revealed a male predominance (male: female 28:24) [4].However, in a research by Enass S. Mohamed et al[5], the distribution of sexes was as follows: In the study group, there were 27 (67.5%) female patients and 13 (32.5%) male patients, while there were 14 (70%) female patients and six (30%) male patients in the control group, demonstrating a female predominance as observed in studies by Sharifian MR [8] and Rosanna Mariangela Giaffredo Angrisani [9]. In a research by Parvane Mahdi et al[6], there was no statistically significant result noted in patients with vitiligo,which was similar to the results obtained in our study

In our study, vitiligo patients between the ages of 18 and 60 were included. The majority of patients belonged to the age groups of 20 to 29 years (42.24%) and 40 to 49 years (23.94), with a mean age of 34. In contrast, in a study by Neel Prabha et al., the mean age of patients was 26.7 years, ranging from 7 to 60 years[4]. In a different study by Enass S. Mohameda et al[5], the mean ages of vitiligo patients and healthy controls were 29.33±8.03 and 31.1±6.3 years, respectively. Similar to our

investigation, E.Fleissig et al[7] did a study with a mean age of the sample of 32.72 ± 16.39. In a study by Sharifian MR[8] et al, the case group consisted of 40 girls and 10 males, with an average age of 22 years (12-40years). In a study carried out by Eman Abd Elmohsin Dawoud[10], the mean age for the control group was 29.77 years with a range of 16-49 years, while for the study group was 31.27 years with a range of 14-47 years. [11]

In a study by Bowen Huang et al.[27] on the elderly population aged 60 to 69 years, men were more likely than women to experience cognitive impairment, which contributed to gender disparities in the relationship between hearing loss and cognition.

Out of the 71 vitiligo patients, 35 (49.29%) were observed to have bilateral sensorineural hearing loss (SNHL), all of which were limited to high frequencies, whereas 36 (50.7%) had normal hearing. It was discovered that none of the patients had unilateral SNHL or conductive hearing loss. 26 (74.28%) of the patients had bilateral mild SNHL. Vitiligo patients did not exhibit any discernible hearing loss, as indicated by p values of 0.5974 and 0.1649 in the right and left ears, respectively. SNHL had a similar impact on both genders. However, according to a research by Al-Mutairi et al.[26], 22% of their vitiligo cases and 18% of the controls had hypoacusis. This difference was not statistically significant, which was the same as in our investigation.

In comparison to 6% of the controls, 69% of vitiligo patients had SNHL, according to Fleissig et al[7]. Statistically, this difference was significant (p = 0.001). In about 25% of the cases, the SNHL was moderate to severe. Similar findings were made in the study of Abhishek Maheshwari et al[31].,where subclinical sensorineural

hypoacusis was discovered in 32% of vitiligo patients. 4% of the control group had hypoacusis (2 patients). All of the hypoacusis instances were sensorineural in nature. However,according to Gopal et al[23] 20% of the patients with vitiligo had hypoacusis, compared to 2% of the controls. The majority of individuals with hypoacusis (87%) had conductive hearing loss, whereas the remaining patients had sensorineural impairments.Mean pure tone thresholds for 0.25–16 kHz were higher in the study group than in the control group, according to Shalaby et al.[24], although this difference was not statistically significant. About 15% of the vitiligo patients with audiologic alterations had no symptoms related to hearing sensitivity. Therefore, it may be assumed that the hearing organ is only little impacted and that most patients do not notice any hearing loss, while some vitiligo patients who had near-normal pure tone thresholds in standard audiometry (0.25-8 kHz) demonstrated abnormalities at protracted high frequency. Several investigations have revealed a decreased percentage of SNHL in vitiligo patients compared to our study.According to Aydogan et al.[16], eight people (14%) with extensive vitiligo experienced sensorineural hypoacusis. Pure tone thresholds at 1,000, 2,000, 4,000, 6,000, and 8,000 Hz were significantly lower in the group with active sickness than in the group with stable disease, according to study by Chang Kee Hong et al.[27] (p 0.05).

Enass S. Mohameda et al, one of the few studies that used additional audiological testing methods, reported that no statistically significant differences existed between the study and control groups for any of the Auditory Brainstem Response (ABR) parameters tested, which supported the finding that even additional audiological tests were found in the current study[5]. Transient Evoked Otoacoustic Emissions (TEOAEs) were significantly less reproducible in the group with vitiligo than in the control group, according to Shalaby et al. [24]. The vitiligo group's

repeatability was 33.3% as opposed to the control group's 98%. Significant variances were also visible in the TEOAE at different frequencies. In a research by Chang Kee Hong et al.[27], the active illness group showed a significantly lower peak I latency and significantly higher interpeak I-III and interpeak I-V latencies than the stable disease group (p 0.05). Electrocochleography revealed that, when compared to controls, vitiligo patients had significantly higher summation potential (SP) amplitudes, action potential (AP) amplitudes in the left ear, and SP/AP ratios in both ears (p 0.05). Brainstem auditory-evoked potentials (BAEP) were used in a study by Rabab Ahmed Koura et al.[30], and cases had significantly longer latencies for the right wave III, the right and left wave V, and the interpeaks for the right wave III and the right and left wave V than controls. Between the two groups, there was no statistically significant variation in the delay of the BAEP wave I or the interpeak III-V.

Neel Prabha and colleagues conducted a study in which ten patients (19.2%) had sensorineural hearing loss (SNHL). Three patients (5.7%) had unilateral SNHL and seven (13.5%) had bilateral SNHL. In 10 of the 20 ears, there was high frequency loss, and in 6 of the 20 ears, there was both low and high frequency loss. Five of the 12 ears with speech frequency involvement showed mild hearing loss, and one showed moderate to severe hearing loss[4] . Another study by Enass S. Mohameda et al. revealed that the mean hearing thresholds at frequencies of 4, 8, and 10 kHz for the study group were statistically elevated when compared to the equivalent values for the control group. The majority of them, 11/15 (73.3%), had mild hearing impairment, and 10/15 (66.7%) had bilateral high-frequency hearing impairment[5]. In a study by Sharifian MR et al.[8], sensorineural hearing loss was bilateral in 63.1% of cases, isolated in 10.6% of cases to the left ear, and isolated in 26.3% of cases to the right ear.

In the current study, it was discovered that there is a positive association between getting older and hearing loss, however it was not statistically significant, with a p value of 0.1856. However, it is inferred in a research by E. Fleissig et al.[7] that SNHL in vitiligo patients develops at an older age and may worsen with ageing. The study also showed a tendency for older vitiligo patients and late onset vitiligo to have SNHL with greater severity. Age and PTA showed a statistically significant positive connection (p 0.05) at all investigated frequencies in both the vitiligo group and the controls, according to Abeir Osman Dabbous et al.[10]. In a study by Neel Prabha et al.[4], the majority of instances of SNHL were found in people between the ages of 41 and 60 (63.6%), which was statistically significant (p-value 0.00). According to Shankar et al. [15], the age group of people above 30 was substantially related with aberrant auditory findings. Sharma et al.[16], however, found no correlation between age and SNHL in vitiligo.

In present study, duration of vitiligo was taken into consideration, and was seen that 44(61.97%) patients had history of disease for less than 20 years, and that there was no positive correlation between duration of disease and hearing loss with the p value of 0.4182 and 0.5202 in right and left ears respectively. In study conducted by Neel Prabha et al Sensorineural hearing loss was present in 5 out of 10 patients (66.7%) having vitiligo for less than 5 years, and 5 out of 32 (86.5%) for more than 5 years[4]. There were no statistically significant differences in the present study related to the duration of vitiligo, which supported result of our study, and a study conducted by Rosanna Mariangela Giaffredo Angrisani[7]. In a similar study by Abeir Osman Dabbous et al.[10] vitiligo duration did not have any effect on cochlear function, not being correlated with either hearing loss or with TEOAE. It seems that once the inner ear melanocytes get damaged, cochlea dysfunction occurs irrespective

of time. Likewise, studies conducted by Aydogan et al.[16] and Gopal et al. [23] did not find any relationship between hearing loss and duration of vitiligo. According to Mahdi et al.[6] this could be explained by the possibility that otic melanocytes are affected at the start of vitiligo and then stabilize afterwards

However in a study conducted by Enass S. Mohameda et al it was found that vitiligo patients with a longer disease duration (>5 years), had higher hearing thresholds at conventional and high frequency audiometry and overall responses of TEOAEs but did not reach the level of statistical significance[5]. Likewise, Hong et al.[28] found a statistically significant association between hearing loss and longer duration of the disease. Al-Mutairi et al.[26] concluded that, contrary to observations in early onset vitiligo, late onset vitiligo did not seem to be associated with audiological abnormalities.

In present study, VASI score was calculated for each patient and mean VASI score obtained was 6.38 for patients with history duration of of disease for less than 20 years , and a mean of 12.49 for patients with a disease duration of more than 20 years, which was statistically significant with a P value <0.0001. However, there was no positive correlation noted between VASI score and hearing loss with a p value of 0.5552. Likewise, in a study conducted by Abeir Osman Dabbous et al.[10] VASI was not correlated with hearing loss nor with TEOAE. In agreement with our study, Shalaby et al.[24] and Fleissig et al.[7]found no relation between duration, severity of the disease (number of affected sites), and any audiological parameter. However, Anbar et al.[3] demonstrated that bilateral cochlear dysfunction was common in segmental and non-segmental vitiligo subtypes of the disease. Aslan et al.[18] found a correlation between hearing loss in vitiligo patients and body percentage involved as an

increasing rate of statistically significant hearing loss was observed, as the body percentage of vitiligo exceeded 10%.

Aydogan et al[16](2005) concluded that their findings, together with other published data, suggested that melanin might have an important role in establishing and/or maintaining the structure and function of the auditory system and in modulating the transduction of auditory stimuli in the inner ear.

In a study conducted by Abeir Osman Dabbous et al.[10] audiometric findings of the vitiligo group, and the statistically significant differences from the control group, suggest a subclinical involvement of the outer and inner hair cells of the cochlea in the basal turn, probably related to the vitiligo condition, but not influenced by vitiligo duration or severity.

CONCLUSION

Vitiligo is an acquired disorder of skin caused by loss or damage of epidermal melanocyte and characterized by well-defined depigmented macules.

The results of this study implies that as a result of the presence of melanocytes in the auditory apparatus, it is indicated that the disease probably targets the melanocytes of the whole body. Accordingly, patients with vitiligo should be evaluated with pure tone audiometry even if they do not exhibit any hearing difficulties. Older subjects with vitiligo might be at a higher risk for audiological abnormalities. These patients should also be informed regarding the associated risk with noise and ototoxic drug exposure.

In present study, Audiological evaluation was done and found to have hearing impairment in 49.2 % of the population but it was statistically insignificant

Hence, from the observations in the study, we can conclude that there is a need for complete auditory evaluation in patients with vitiligo, and further studies should include a larger sample size and application of other auditory assessment tests as well.

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 Vitiligo attending ENT & HNS outpatient department".

All patients underwent thorough history taking and clinical examination. These patients were subjected to Pure Tone Audiometry for assessment of hearing threshold and Dermatological examination was done to determine VASI Score.

The following results were noted-

- Male predominance was observed in the study, with a surge noted mostly in age demographics of 20-29 and 40-49 years
- Relevance of hearing loss and Vitiligo in comparison between right and left ear which showed statistically no significant hearing loss bilaterally.
- There was no positive relationship between duration in Vitiligo and hearing impairment.
- VASI index computed for each showed no positive relationship between significant hearing loss bilaterally.

But, according to literature it is suggested that patients diagnosed with vitiligo should undergo hearing assessment on regular intervals, as sensory neural hearing loss was prevalent in this population.

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ANNEXURE I – CONSENT FORM

INFORMED CONSENT

"ASSESSMENT OF HEARING IN PATIENTS WITH VITILIGO - A ONE YEAR OBSERVATIONAL STUDY IN KLES Dr. PRABHAKAR KORE HOSPITAL''

PRINCIPAL INVESTIGATOR: **Dr.**
Post Graduate student
Department of Otorhinolaryngology
and Head and Neck Surgery.
J.N. Medical College,
KAHER, Belagavi.

GUIDE: **DR.**
Head of Department, Professor,
Department of Otorhinolaryngology
and Head and Neck Surgery.
J.N. Medical college,
KAHER, Belagavi.

CO-GUIDE: **Dr.**
M.D (Dermatology &Venereology)
Head of Department, Professor,
Department of Dermatology,
J.N. Medical College,
KAHER, Belagavi.

INTRODUCTION AND PURPOSE: The present study is conducted among patients with vitiligo attending the out-patient department of Dermatology and referred to ENT & HNS in KLE's Dr. Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi and will be investigated for pure tone audiometry. 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 hearing with Pure tone audiometry and functions of cochlea 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 queries regarding the study, you can contact **Dr. Harsha Hegde**, Chairperson, JNMC, IEC & Scientist D, ICMR, National Institute of Traditional Medicine.

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:

Legally authorised Relative:

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

Name and signature of the interviewer:

Signature of the guide:

Date:

ANNEXURE II – PROFORMA

"ASSESSMENT OF HEARING IN PATIENTS WITH VITILIGO - A ONE YEAR OBSERVATIONAL STUDY IN KLES Dr PRABHAKAR KORE HOSPITAL"

Date:

Name:

Age:OP/IP no:

Sex: Date of assessment:

Address: Date of discharge:

Occupation: Diagnosis:

CLINICAL PROFILE:

Chief Complaint:

History of Present Illness

Past History:

Personal History:

Family History:

Treatment history:

DRUGS TAKEN DURATION

Oral Corticosteroids	
Cyclosporin	
Methotrexate	
Calcipotriol	
Azathioprine	
Phototherapy(UVB / Systemic psoralen and UVA)	
Other medications	

I) General Physical Examination -

Build:

Nourishment:

Blood Pressure:

Pulse:

Respiratory Rate:

Pallor :

Icterus :

Clubbing :

Cyanosis :

Lymphadenopathy :

Edema :

II) ENT Examination

1. **EAR EXAMINATION:**

	Right	Left
Pinna		
Pre auricular area		
Post auricular area		
External auditory canal		
Tympanic membrane		

TUNING FORK TESTS:

Rinne's test	256 Hz	
	512 Hz	
	1024 Hz	
Weber's test:		
Absolute Bone Conduction test:		

FACIAL NERVE EXAMINATION:

2. **NOSE EXAMINATION**

External appearance

- Root
- Bridge
- Dorsum
- Alae
- Tip
- Columella

Cold spatula test

Anterior Rhinoscopy

Posterior Rhinoscopy

Paranasal Sinus Examination

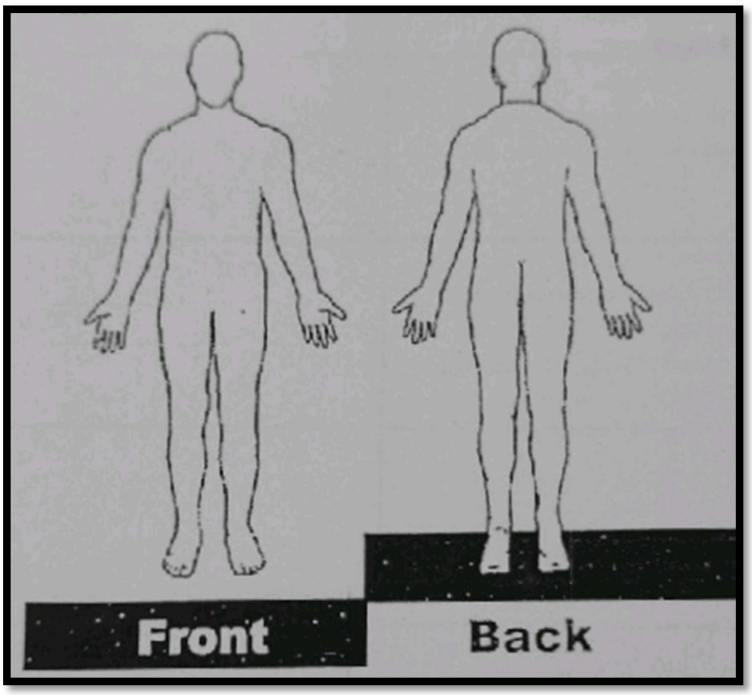
3.THROAT EXAMINATION:

4.NECK EXAMINATION:

5. MUCOCUTANEOUS EXAMINATION:

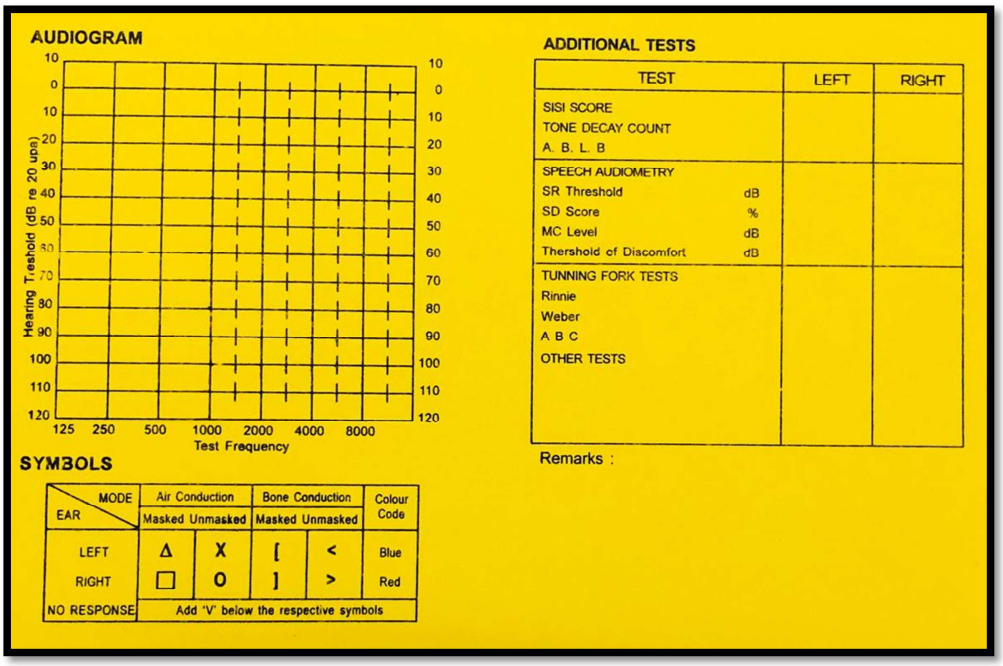
- Primary lesions – Macule / Patch / Extensive
- Colour of lesions- Hypopigmented / Depigmented / Hyperpigmented / Erythematous / others
- Distribution of lesions- Localized / Generalized / Segmental
- Sites of Distribution- Head / trunk / UL / LL
- Shape of lesions- Round / Irregular
- Number of lesions-
- Borders of the lesion- Well defined / ill defined
- Hair over the lesion- Present / absent
- If Hair is present over the lesion, then – Pigmented / Non-pigmented
- Any other skin conditions- Yes / NO
- Mucosal Involvement- Yes / No
- Percentage of BSA-
- Activity of disease

6.VITILIGO AREA SCORING INDEX(VASI):



DIAGNOSIS:

PURE TONE AUDIOMETRY: RIGHT LEFT



ANNEXURE III – PHOTOGRAPHS

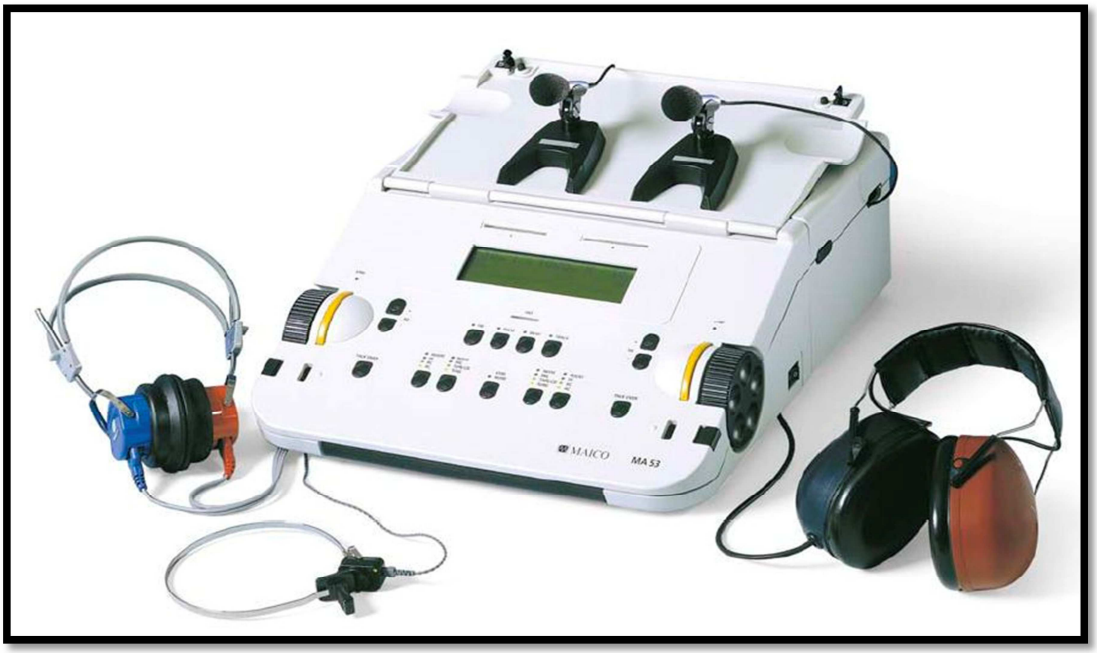


IMAGE 1 MAICO MA53 audiometer

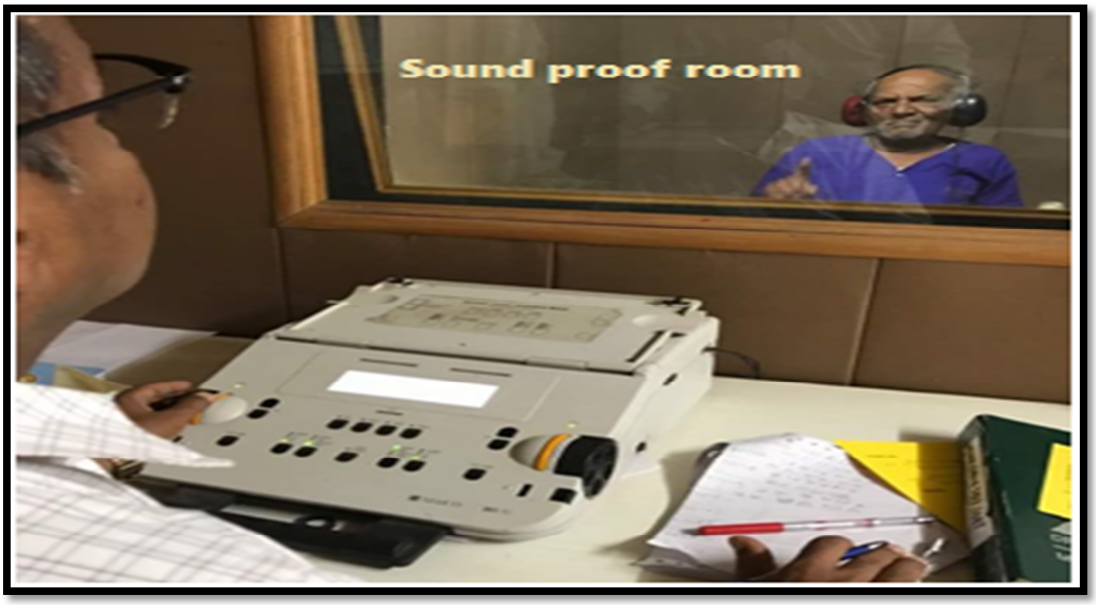


IMAGE 2 PTA in Progress

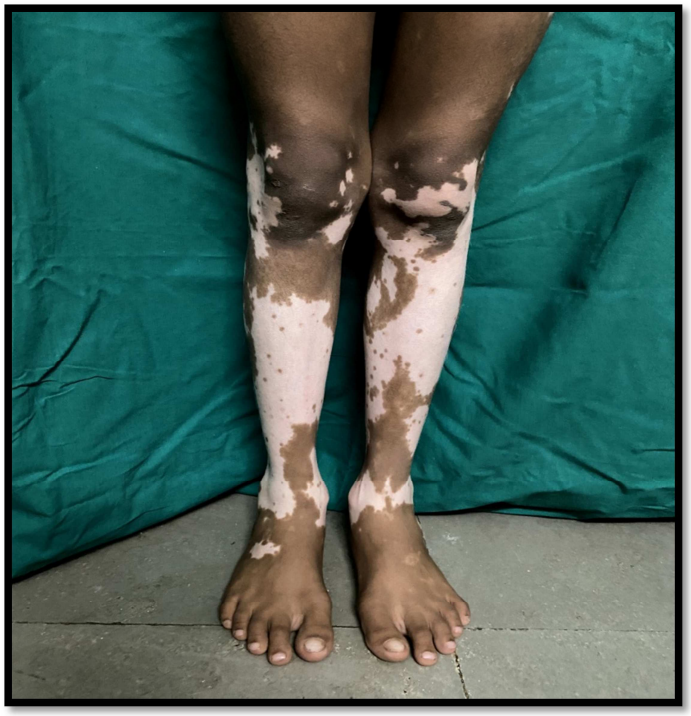


IMAGE 3 Vitiligo vulgaris (Bilateral lower limb)

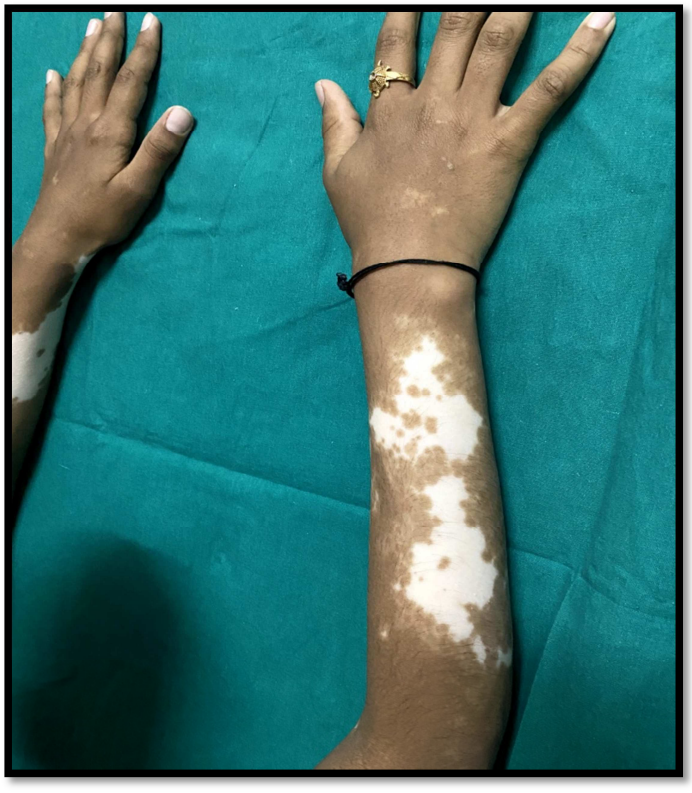


IMAGE 4 Vitiligo vulgaris (Bilateral upper limb)

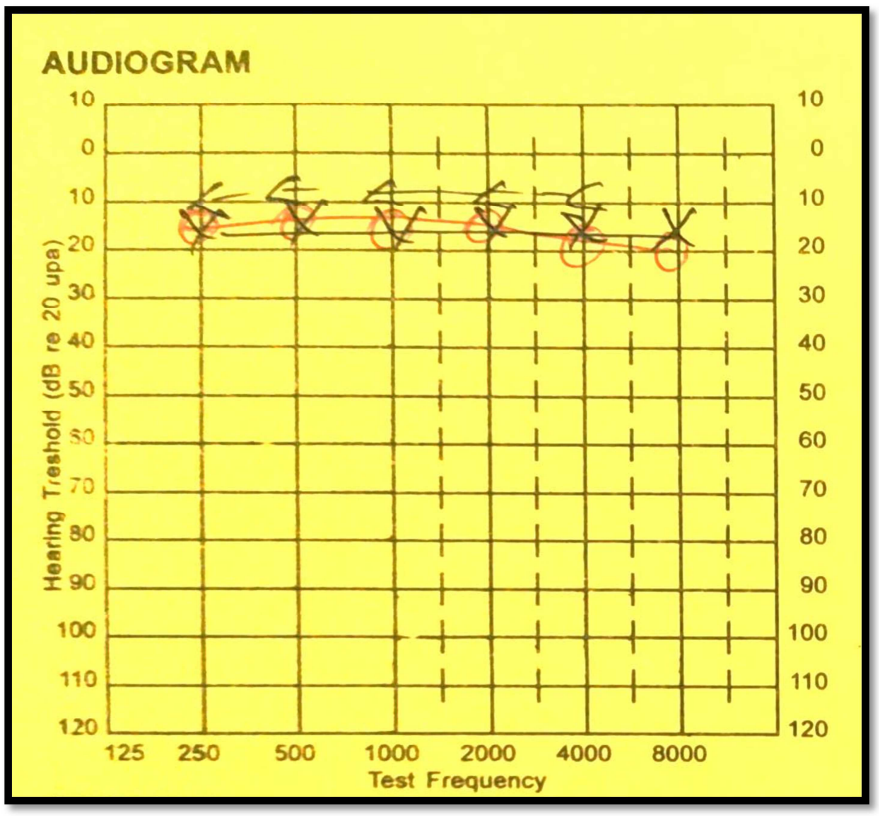


IMAGE 5 PTA showing Bilateral normal hearing

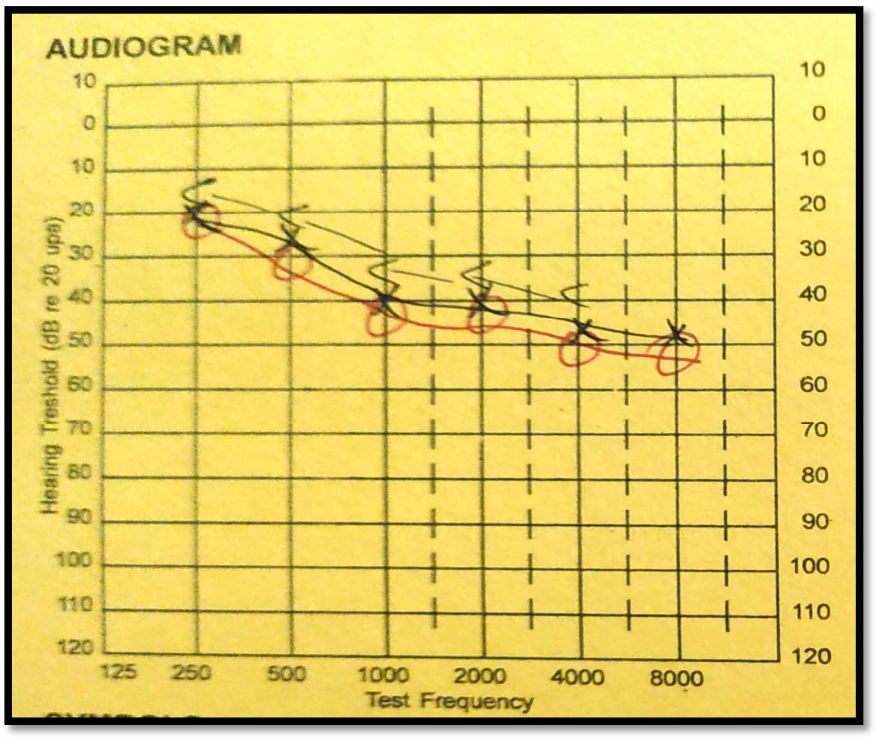


IMAGE 6 PTA showing Bilateral sensorineural hearing loss

ANNEURE IV – KEY TO MASTER CHART

F : Female

M : Male

dB : Decibel

SNHL : Sensory Neural Hearing Loss

B/L : Bilateral

ANNEXURE V- MASTER CHART

NAME	OP NO:	AGE	SEX	RIGHT EAR	LEFT EAR	HEARING LOSS	FREQUENCY OF HEARING LOSS	DURATION OF DISEASE	VASI SCORE
SMITA	3914082	20	F	15	15	NORMAL	NORMAL	7	7.4
ANITA	6223294	42	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	20	3
ASHWINI	3938755	29	F	35	40	B/L MILD SNHL	HIGH FREQUENCY	15	14.6
SHIVANAND	4970842	23	M	30	40	B/L MILD SNHL	HIGH FREQUENCY	10	13.4
SAGAR B	6402212	23	M	15	15	NORMAL	NORMAL	7	5.4
VIRENDRA	6254009	28	M	30	30	B/L MILD SNHL	HIGH FREQUENCY	6	5.4
LAXMI	6157075	26	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	11	2
PRABHA	5292106	45	F	15	15	NORMAL	NORMAL	20	21.4
SUMITRA	3616305	24	F	15	15	NORMAL	NORMAL	12	7.4
MAHANTAPPA	6026321	41	M	32.3	40	B/L MILD SNHL	HIGH FREQUENCY	15	8
ALIKA	4784073	22	F	15	15	NORMAL	NORMAL	10	5.4
BHARATI	6345716	41	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	30	21.4
KIRATI	6294205	34	F	15	15	NORMAL	NORMAL	20	20.4

YESSAGANIB	4783087	52	M	35	40	B/L MILD SNHL	HIGH FREQUENCY	30	2
RESHMA S	6032542	42	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	20	21.4
ASHNA	6158505	28	F	15	15	NORMAL	NORMAL	10	2
PANDURANGA	5429877	38	M	25	25	NORMAL	NORMAL	5	7.4
RUTAMMA	6286738	32	F	35	30	B/L MILD SNHL	HIGH FREQUENCY	10	9.2
SAMEENA	5982575	28	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	3	2
NEELKAMAL	6061552	21	M	15	15	NORMAL	NORMAL	22	1
RUTYA	6323036	27	F	15	15	NORMAL	NORMAL	10	9.2
SUNITA	3914082	23	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	7	3
MAHANTESH	6045321	34	M	35	30	B/L MILD SNHL	HIGH FREQUENCY	20	16.2
VAISHNAVI	4098883	29	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	3	2
ASHWITHA	5999628	30	F	30	30	B/L MILD SNHL	HIGH FREQUENCY	10	9.4
MALLAVA	6141674	56	M	35	30	B/L MILD SNHL	HIGH FREQUENCY	30	8.3
GAJANAN	6277590	35	M	15	15	NORMAL	NORMAL	15	2
NAGAYYA	6272161	34	M	15	15	NORMAL	NORMAL	20	19.4
INDRADEVI	3936519	56	F	35	30	B/L MILD SNHL	HIGH FREQUENCY	30	5.4

SULEKHA	6258511	22	F	30	40	B/L MILD SNHL	HIGH FREQUENCY	10	12
PRASHANT	6315805	46	M	50	45	B/L MODERATE SNHL	HIGH FREQUENCY	23	15
LOKAPPA	6195410	26	M	15	15	NORMAL	NORMAL	10	2
SHREYA	2995362	21	F	15	15	NORMAL	NORMAL	15	3
SHASHIKALA	5871633	48	F	35	40	B/L MILD SNHL	HIGH FREQUENCY	30	21.4
YALLAPA	5958479	50	M	60	70	B/L SEVERE SNHL	HIGH FREQUENCY	10	13
RAMESH	5752916	19	M	15	15	NORMAL	NORMAL	5	1
SHABAZ	5529573	30	M	90	80	B/L SEVERE SNHL	HIGH FREQUENCY	20	19.4
PRAKASH	6375305	46	M	15	15	NORMAL	NORMAL	30	12
DEEPEN	3954825	28	M	55	50	B/L MODERATE SNHL	HIGH FREQUENCY	12	7.4
KALLAPA	6351119	42	M	30	50	R-MILD SNHL , L-MODERATE SNHL	HIGH FREQUENCY	42	5.4
CHAITRA	4159581	21	F	15	15	NORMAL	NORMAL	10	4.5
SHRUTI	6363558	25	F	60	50	B/L MODERATE	HIGH FREQUENCY, SNHL	7	2

PRAVEEN	6216806	48	M	15	15	NORMAL	NORMAL	23	21.2
ULLAS	5388377	58	M	55	50	B/L MODERATE SNHL	HIGH FREQUENCY	30	9.3
GEETA	5952008	45	F	80	80	B/L SEVERE SNHL	HIGH FREQUENCY	10	11.4
SANJANA	4314414	50	F	15	20	NORMAL	NORMAL	10	19.4
PADMASHREE	5949628	44	F	15	15	NORMAL	NORMAL	30	15.3
RADHIKA	3416972	22	F	15	15	NORMAL	NORMAL	10	9.15
YELLAPA	1034497	37	M	30	31.6	B/L MILD SNHL	HIGH FREQUENCY	20	11.7
SHOAIB	6034237	27	M	35	30	B/L MILD SNHL	HIGH FREQUENCY	12	4.5
CHIKAPPA	6197624	23	M	15	15	NORMAL	NORMAL	10	6.3
THUKAPPA	6197262	43	M	15	15	NORMAL	NORMAL	16	7.2
ABHIJIT	5897278	31	M	30	30	B/L MILD SNHL	HIGH FREQUENCY	12	1.8
DEEPA	5378354	21	F	15	15	NORMAL	NORMAL	5	0.9
NIKHIL	5123556	19	M	15	15	NORMAL	NORMAL	10	8
KAVERI	3626629	52	F	30	30	B/L MILD SNHL	HIGH FREQUENCY	30	2
PRIYANKA	5354844	28	F	15	20	NORMAL	NORMAL	4	7

SHUBHAM	5090473	21	M	15	15	NORMAL	NORMAL	10	6
SEETHADEVI	4674431	52	F	30	30	B/L MILD SNHL	HIGH FREQUENCY	35	10
NARAYAN	6013574	30	M	15	15	NORMAL	NORMAL	10	8
SANHAL	5426784	36	M	30	30	B/L MILD SNHL	HIGH FREQUENCY	20	16.4
PRAVEEN	6240909	26	M	30	30	B/L MILD SNHL	HIGH FREQUENCY	10	6
MANJUNATH	5169062	43	M	15	15	NORMAL	NORMAL	20	10.5
RANJANA	6134391	45	F	15	15	NORMAL	NORMAL	23	5
RESHMA N	6072542	47	F	15	20	NORMAL	NORMAL	10	8
DAYANAND	5977687	38	M	15	15	NORMAL	NORMAL	12	9
REYANABANU	4808524	22	F	15	15	NORMAL	NORMAL	1	0.75
SADDAM	5983471	41	M	20	20	NORMAL	NORMAL	20	12.6
RAMAN	5983637	39	M	15	15	NORMAL	NORMAL	20	10
ACHYUT	4990713	22	M	55	50	B/L MODERATE SNHL	HIGH FREQUENCY	10	0.9
MOHD.CHOPDAR	6199282	20	M	15	20	NORMAL	NORMAL	5	6.3