"INCIDENCE OF SENSORINEURAL HEARING LOSS IN PATIENTS WITH ALLERGIC RHINITIS -A ONE YEAR CROSS SECTIONAL STUDY IN KLES DR.PRABHAKAR KORE HOSPITAL BELAGAVI"

BY

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Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"INCIDENCE OF SENSORINEURAL HEARING LOSS IN PATIENTS WITH ALLERGIC RHINITIS- A ONE YEAR CROSS SECTIONAL STUDY IN KLES DR. PRABHAKAR KORE HOSPITAL BELAGAVI",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
AR	Allergic Rhinitis
aCL	Anticardiolipin
APC	Antigen Presenting Cells
CHL	Conductive Hearing Loss
CNI	Olfactory Nerve
CNV	Trigeminal Nerve
CROS	Contralateral Routing Of Signal
DPOAE	Distortion Product Otoacoustic Emissions
ETD	Eustachian Tube Dysfunction
IgE	Immunoglobulin E
IHC	Inner Hair Cells
INV	Internal Nasal Valve
LLC	Lower Lateral Cartilage
LLSAN	Levator Labii Superioris Alaeque Nasi
MRI	Magnetic Resonance Imaging
NIHL	Noise-Induced Hearing Loss
OAE	Otoacoustic Emissions
OHC	Outer Hair Cells
OMC	Osteomeatal Complex
OME	Otitis Media With Effusion
RAST	Radio Allergo Sorbent Tests
SFOAE	Stimulus-Frequency OAE

SNHL	Sensorineural Hearing Loss
TCR	T Cell Receptor
TEOAE	Transient Evoked OAE
DPOAE	Distortion Product OAE

ABSTRACT

Title:

Incidence of Sensorineural Hearing Loss in Patients With Allergic Rhinitis -A One Year Cross Sectional Study in Kles Dr. Prabhakar Kore Hospital Belagavi.

Objectives:

To study the incidence of sensorineural hearing loss in patients with allergic rhinitis.

Methods:

A one year study was undertaken and 75 patients with allergic rhinitis, who satisfy the inclusion criteria, who gave consent were taken for our study purpose. A thorough history has been obtained and complete examination of the ear, nose and throat has been performed. Pure tone audiometry, Distortion product otoacoustic emission (DPOAE) has been performed and assessed for sensorineural hearing loss.

Results:

The present study revealed highest number of participants belonged to Age group 21 to 30 years (33.3%) followed by 10 to 20 years (30.7%) with mean age of 28 years, The male: female ratio was 1:0.6 ,Mean duration of symptoms was 2.82 years , Running nose (94.7%) was the most common symptom noted among study participants followed by nasal block (90.7%).It is noted that incidence of hearing loss noted in 54.7% cases on right side year and 40% on left side ear ,Sensorineural hearing loss was the most common type of hearing loss noted with incidence of 42.7% in right ear and 36% on left side ear noted among study participants. The study observed that there is statistical significance associated between duration of AR symptoms and sensorineural hearing loss, this indicates that severity of SNHL

increased with increase in duration of AR symptoms.Distorting product otoacoustic emissions (DPOAE) are absent or abnormal in 56% cases of right ear and 45.3% cases of left ear with reduced or absent distortion product amplitude and a lower signal-to-noise ratio in higher frequency ranges , which shows outer hair cells dysfunction.

Conclusion:

The current research indicates that the occurrence of sensorineural hearing loss (SNHL) is more prevalent among individuals with allergic rhinitis; DPOAE tests demonstrate reduced or absent distortion product amplitudes and a lower signal-to-noise ratio in higher frequencies. Allergens might affect the inner ear function, particularly outer hair cells can cause sensorineural hearing loss which does not reverse once occurred. Present study indicates that individuals with Allergic rhinitis are more prone to hearing loss and severity of hearing loss is increased with duration of symptoms of allergic rhinitis. Therefore, early diagnosis and treatment of allergic rhinitis are essential. Additionally, in patients experiencing symptoms for more than 4–6 months we recommend hearing assessments to enable timely intervention and reduce the risk of permanent hearing loss.

Keywords: Allergic rhinitis , Sensorineural hearing loss, Outer hair cells ,Pure Tone Audiometry, Distortion product otoacoustic emission

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INTRODUCTION

"Allergic rhinitis (AR) is a prevalent type-I hypersensitivity response of the nasal mucosa, chiefly driven by immunoglobulin E (IgE). It presents through a variety of clinical symptoms, such as nasal blockage, clear nasal discharge, sneezing, and itching in the nasal and nasopharyngeal areas. This condition can be divided into perennial and seasonal types, with the former often resulting in ongoing nasal blockage and a reduced sense of smell, while the latter typically exhibits extra symptoms like conjunctivitis. Upper airway allergies rank among the most frequent issues encountered by patients in the outpatient department of otorhinolaryngology, where individuals display a range of symptoms associated with the head and neck. The prevalence of allergic rhinitis is considerable, affecting a large segment of the population and resulting in significant morbidity."[1]

"The connection between allergic rhinitis and ear-related complications has garnered increased focus in recent years. Several studies indicate that allergic rhinitis plays a role in the onset of otitis media with effusion (OME), particularly through mechanisms that involve Eustachian tube dysfunction (ETD). Allergic inflammation can block the Eustachian tube, leading to negative pressure in the middle ear and ensuing conductive hearing loss. "[1]

"Epidemiological evidence consistently associates allergies with higher rates of OME, with the reported prevalence of allergies among affected individuals ranging from 25% to 89%. While the conductive loss component of the allergic rhinitis has been well studied before, hence, the primary objective of this study is to evaluate the incidence of sensorineural hearing loss in patients with allergic rhinitis. Understanding these relationships may enhance clinical management strategies for

patients with allergic rhinitis, potentially leading to improved outcomes. It is believed that this relationship is mediated by inflammatory cytokines released during allergic reactions, which may also contribute to inner ear dysfunction resulting in sensorineural hearing loss. Inner ear damage may occur due to cytokines (such as tumor necrosis factor, TNF) released during inflammation, which can also result in sudden deafness. "[1]

"Research has indicated that food allergies can raise total IgE and specific IgE (sIgE) levels, potentially leading to sudden sensorineural hearing loss in patients with immune-mediated deficiencies. This is likely due to the activation of anticardiolipin (aCL) antibodies or their interaction with anti-beta2 GP1 antibodies, which are deficient in patients with immune-mediated deficiencies, leading to injury of the outer ear cells (OHC). Although the auditory effects of allergic rhinitis are becoming more acknowledged, the underlying mechanisms are still not well understood. The endolymphatic sac, a crucial structure within the inner ear, is thought to be involved in local immune responses, which may be altered in individuals with upper airway allergies. This involvement raises concerns about the potential effects of allergic rhinitis on various parts of the auditory pathway, including the outer, middle, and inner ear structures."[1]

"A study examining the relationship between AR and hearing has reported that AR is the most prevalent respiratory disorder exacerbated by air pollution. They found an average hearing loss of 10 ± 9.1 dB in individuals with AR, compared to 2.5 ± 2.2 dB in the control group ($P < 0.0005$), concluding that AR may lead to conductive hearing loss in adults."[1]

"Supporting results were found by Dees and Lefkowitz[2], as well as Rózańska-Kudelska et al[3] who discovered that a majority of individuals displayed conductive hearing loss, particularly recurrent secretory otitis media."[2,3]

Research by Toubi E et al[4] revealed that, "food allergies can increase total IgE and sIgE levels and result in sudden sensorineural hearing loss in patients with immune-mediated deficiency. The likely mechanism involves externally activated anticardiolipin (aCL) antibodies or their interactions with anti-beta2 GP1 antibodies, which are insufficient in patients with immune-mediated deficiencies, leading to injury of the outer ear cells (OHC)."[4]

Furthermore, Lee JS et al[5] indicated that, "conditions such as hyperlipidemia, hypertension, and diabetes also constitute risk factors for hearing loss. These patients exhibit thick blood, abnormally aggregated platelets, and small fat emboli, which can cause the blood vessels supplying the inner ear to become very thin due to hypoxia. Consequently, OHCs may suffer damage from inadequate blood supply, resulting in hearing impairment. Hearing loss can be assessed through pure tone audiometry, which determines the extent of hearing loss."[5] Conversely, otoacoustic emissions (OAEs) are sounds originating from the cochlea's sensory hair cells. OAEs can be recorded in quiet conditions but are more commonly measured in response to sound stimulation. Continuous sinusoidal stimuli provoke distortion product otoacoustic emissions (DPOAEs), which can be utilized to evaluate the condition of cochlear outer hair cells.[6] DPOAEs, a type of OAE examination, can detect slight hearing loss at higher frequencies. A failure to pass the DPOAE test indicates dysfunction, reduced counts, or loss of OHCs. Therefore, DPOAE is typically employed to determine if hearing loss caused by tympanitis, sinusitis, or other forms of inflammation results from OHC impairment.[7]

Given the limited existing literature on the audiological implications of allergic rhinitis, this study aims to fill a critical gap and contribute valuable insights into the intersection of otorhinolaryngology and allergy.

AIMS AND OBJECTIVES

- To study the incidence of sensorineural hearing loss in patients with allergic rhinitis.

REVIEW OF LITERATURE

1. SENSORINEURAL HEARING LOSS[8]

"Hearing loss is a prevalent issue for which individuals are often referred to secondary care to see an otolaryngologist. There are two main categories of hearing loss: conductive and sensorineural hearing loss. Sensorineural hearing loss (SNHL) is the most widespread form and represents the majority of hearing loss cases. SNHL pertains to any hearing loss caused by a condition affecting the cochlea, auditory nerve, or central nervous system. Patients experiencing new-onset hearing loss should be thoroughly evaluated and undergo complete audiometric assessment by a multidisciplinary team, which includes an otolaryngologist, audiologist, radiologist, and speech/language therapist."[8]

Etiology and epidemiology[8]:-

"The most common causes of sensorineural hearing loss are Congenital - syndromic and nonsyndromic, presbycusis, noise-induced hearing loss, head injury, Meniere's disease, otoxicity -aminoglycosides, loop diuretics, some chemotherapeutic agents, Systemic conditions - meningitis, diabetes,Vestibular schwannoma,Others - autoimmune, barotrauma, perilymphatic fistula.The incidence of sensorineural hearing loss varies in different countries. In India, the prevalence of sensorineural hearing loss (SNHL) varies, with studies reporting rates ranging from 4.5% to 18.3% in the general population . Due to different studies using varying thresholds when classifying hearing loss, there is little consensus in the literature regarding the epidemiology of age-related hearing known as presbycusis. In presbycusis, hearing loss prevalence doubles every decade of life from the second through to the seventh decade, and is nearly universal past the eighth decade of life. Another important cause

of hearing loss in the adult population is noise-induced hearing loss (NIHL). It has been estimated that 16% of adults worldwide disabling hearing loss is occupational noise related. This remains a common occupational disease despite legislation in place in most developed countries to prevent NIHL."[8]

"Congenital hearing loss is typically sensorineural and can arise from multiple causes. In individuals who received comprehensive prenatal care, congenital infectious sources like cytomegalovirus are uncommon, with genetic factors being the most frequent causes. Numerous genetic syndromes include hearing loss as a characteristic, and the occurrence of SNHL in childhood necessitates a comprehensive evaluation."[8]

Pathophysiology[8]

"Sensorineural hearing loss (SNHL) happens when hair cells in the inner ear, the vestibulocochlear nerve, or the brain areas that process sound are damaged. This type of hearing loss is different from conductive hearing loss, which occurs when sound waves can't reach the inner ear. The ear has three main parts: the external ear, the middle ear, and the inner ear. The external ear includes the pinna and the external auditory canal. The middle ear has the tympanic membrane, ossicles, Eustachian tube opening, oval window, and round window. The inner ear contains the cochlea and part of the auditory nerve. Each part of the ear plays a role in hearing, but SNHL specifically involves problems in the inner ear. Sound travels to the cochlea through the stapes and the oval window. Once sound enters the cochlea, outer hair cells amplify the sound, and inner hair cells convert it to electrical signals. When sound reaches the cochlea, it creates a traveling wave along the basilar membrane. This wave stimulates the outer hair cells, which act as amplifiers. The basilar membrane is

organized to respond to different sound frequencies; it reacts to high-frequency sounds at the base and low-frequency sounds at the apex. The inner hair cells change the traveling wave into electrical signals, which then connect to the spiral ganglion and form the auditory nerve."[8]

Many pathophysiological processes that damage inner ear results in SNHL.

1. "Structural abnormality of cochlear components: e.g., trauma or congenital conditions."[8]
2. "Aberrant metabolic activity: Cochlear function is determined by the transport of ions."[8]
3. "Vascular: Interference with the vascular supply to the cochlea can occur in conditions such as noise trauma, ototoxicity, and systemic vascular events, which will affect the function of the stria vascularis."[8]

"Overcrowding of the basilar membrane preventing OHCs motility and IHCs transduction capabilities: Prevalent in conditions such as diabetes and autoimmune pathology."[8]

"Noise trauma: With noise trauma, the vibrational shift between the tectorial and basilar membranes increases, and this shift can damage the stereocilia of the OHCs. In turn, the stiffness of the organ of Corti decreases. Aminoglycoside antibiotics, such as gentamicin, are potassium channel blockers and stop the hair cells from depolarizing. They can also change the perilymph ion concentration leading to damage of the hair cell bundle causing permanent hearing loss."[8]

"According to Schuknecht's classification in presbycusis, three major cochlear structures can independently degenerate and influence the degree of hearing loss; afferent neurons, the organ of Corti, and stria vascularis. "[8]

A. "Sensory-steep high-frequency hearing loss with preserved speech perception - degeneration of the organ of Corti."[8]
B. "Neural – down sloping high-frequency hearing loss with a disproportionate loss of speech perception - degeneration of spiral ganglion cells."[8]
C. "Strial/metabolic - a flat SNHL with preserved speech perception - degeneration of stria vascularis."[8]
D. "Cochlear conductive - progressive downsloping high-frequency SNHL - increased stiffness of the basilar membrane."[8]

Histopathology[8]

"The organ of Corti serves as the sensory epithelium within the cochlea and is found in the scala media. It comprises sensory hair cells and non-sensory supporting cells. Among the sensory cells are outer hair cells (OHCs) and inner hair cells (IHCs), which are uniquely organized: there is one row of IHCs and three rows of OHCs, with supporting cells in between. Each hair cell has a bundle of stereocilia arranged in rows, with their height increasing in one direction on the cell's upper surface. When the tectorial and basilar membranes move, they cause the stereocilia to bend, activating or deactivating receptors on the hair cell's surface. This action allows potassium ions to enter the hair cell, leading to depolarization. Consequently, calcium channels open, and the auditory nerve sends sound wave information to the brain."[8]

History and Physical[8]

"When assessing a patient with sensorineural hearing loss (SNHL), it is essential to take a thorough history. Key information to gather includes the age of onset, the laterality of symptoms, the rate of decline, the presence of fluctuating symptoms, and any associated symptoms such as tinnitus, aural fullness, disequilibrium, and vertigo. Establishing the premorbid hearing level is crucial for directing rehabilitation and determining whether the hearing loss is new or a deterioration of an existing condition. It is also important to inquire about any previous ear surgeries, history of noise exposure, past head trauma, barotrauma, or exposure to ototoxic medications like aminoglycosides."[8]

"Patients with presbycusis typically report a gradual decline in hearing. They may turn up the television louder than usual and frequently ask others to speak more loudly. Often, family members are the first to notice these changes. A history of personal or occupational noise exposure is common in cases of noise-induced hearing loss. Gathering social history is vital as it helps to guide management and provides insight into how the patient's symptoms impact their lives and those of their families. Many individuals with hearing loss find the condition isolating; activities they once enjoyed, such as going to the cinema, dining out, and socializing with family and friends, become stressful, leading them to withdraw from such experiences. "[8]

"When seeing patients in the clinic with new hearing loss, it is important to perform a focused otological examination along with a complete head and neck exam, including an assessment of all cranial nerves, even though these examinations usually appear normal. "[8]

Evaluation[8]

"A complete audiometric evaluation is the gold standard for evaluating a hearing loss and should be performed to evaluate someone with sensorineural hearing loss. In clinical practice, tuning fork tests, a quick and easy bedside investigation, are usually performed first alongside a pure tone audiogram (PTA) and tympanometry. "[8]

Rinne and Weber Tests

"This is an examination conducted at the bedside using a 512Hz tuning fork, which is beneficial for clinicians attempting to differentiate between conductive hearing loss and sensorineural hearing loss (SNHL). However, a minimum difference of 20 dB between the ears or between the thresholds of conduction and sensorineural hearing is necessary for these tests to be effective." [8]

"The Weber test consists of striking the tuning fork against the knee and placing it at the center of the patient's forehead. The patient is then asked to determine which ear perceives the sound most prominently. In cases of unilateral SNHL, the patient will hear the sound more prominently in their unaffected or 'better' ear, while in the presence of conductive hearing loss, the sound will be perceived more in the affected or 'poorer' ear. In bilateral SNHL, the sound will not be perceived favorably in either ear." [8]

"The Rinne test begins with striking the tuning fork and then testing it in two locations: first on the patient's mastoid process until the sound is no longer audible and then about 1cm away from the external auditory canal. The first position assesses bone conduction and the second evaluates air conduction. A normal result, or a Rinne positive outcome, occurs when the patient indicates that they can still hear the sound

when the tuning fork is in front of their ear, indicating that air conduction is superior to bone conduction and there is no conductive loss. Conversely, if the test shows a Rinne negative result, the patient will state they could not hear the sound when the tuning fork is positioned in front of their ear, indicating that bone conduction is superior to air conduction and suggesting conductive hearing loss. In cases of SNHL, when testing the affected ear, the Rinne test should yield a positive result as there is no conduction loss." [8]

Pure Tone Audiogram

"Patients are frequently referred to an outpatient clinic for an audiogram to assess their hearing. This test evaluates both air and bone conduction pathways, and simply put, the thresholds for both are charted on a graph that displays a curve at ascending sound frequencies up to 8000Hz. In cases of sensorineural hearing loss (SNHL), the curves for both air and bone conduction deteriorate, with no detectable air-bone gap. The configuration of the curve varies based on the specific underlying condition. For instance, in presbycusis, you will observe a high-frequency loss that slopes downward. On the other hand, in conductive hearing loss, the air conduction curve declines and shifts downward, while the bone conduction curve remains unchanged. The distinction between these two curves results in the presence of an air-bone gap."[8]

Otoacoustic Emissions (OAEs)

"These are sounds generated by the inner ear (cochlea) in response to auditory stimuli. These sounds can be measured and are used clinically to assess hearing, especially in newborns and individuals who may not be able to reliably participate in traditional hearing tests. "[8]

"A small probe with a microphone and speaker is placed in the ear canal,The device plays soft clicking or tonal sounds into the ear.If the cochlea (especially the outer hair cells) is functioning properly, it will produce an "echo" or sound response.The probe's microphone picks up this response and records it. "[8]

Types of OAEs

1. **Spontaneous OAEs (SOAEs)**:
    a. Occur without external stimulation.
    b. Some people with normal hearing.
    c. No clinical significance for hearing loss assessment.
2. **Evoked OAEs**:

"Transient Evoked OAEs (TEOAEs): Triggered by short sounds such as clicks or tone bursts. They are often utilized in hearing screenings for newborns." [8]

"Distortion Product OAEs (DPOAEs): Induced by the interaction of two continuous tones (f1 and f2) within the cochlea, resulting in a distortion product. DPOAEs are employed to assess particular frequency areas of the cochlea." [8]

"Stimulus-Frequency OAEs (SFOAEs): Produced by a single pure tone; they are less commonly used due to the complexities involved in measurement." [8]

Uses of OAE Testing:

"Newborn hearing screening (common in hospitals), Monitoring cochlear function in people at risk of hearing loss (e.g., from noise exposure or ototoxic

medications),Diagnosing hearing loss in children or uncooperative patients, Differentiating between sensory (cochlear) and neural (nerve-related) hearing loss." [8]

Other Tests[8]

"Tympanometry: This test is utilized to evaluate the functionality of the middle ear and the movement of the tympanic membrane. It is commonly employed in clinical settings to determine the presence of otitis media with effusion and dysfunction of the eustachian tube. Additionally, the acoustic stapedial reflex can be measured, with the lowest sound intensity that induces the reflex being identified as the acoustic reflex threshold." [8]

"Electrophysiological tests: Auditory brainstem response testing assesses the activity of the nervous system and can be influenced by tumors at the cerebellopontine angle that put pressure on the cochlear nerve as well as by demyelination of neural pathways. This test is also useful for estimating hearing thresholds in infants." [8]

"Speech audiometry: This assessment is crucial for understanding how hearing loss affects communication abilities." [8]

"Head computed tomography scans, particularly with a thin temporal bone window, as well as brain magnetic resonance imaging (MRI), are conducted to investigate cochlear ossification, the existence of a cerebellopontine angle tumor, or indications of active mastoiditis." [8]

"Although laboratory tests are generally not necessary, there are exceptions. For instance, when evaluating a potential autoimmune origin of sensorineural hearing

loss (SNHL), tests such as erythrocyte sedimentation rate, antinuclear antibody, rheumatoid factor, and anti-microsomal antibodies may be performed." [8]

Origin and Definition of Allergy[9-11]:

"The term "allergy" finds its roots in the Greek words "allos," meaning "other," and "ergon," signifying "reaction." Coined by Clemens Peter Freiherr von Pirquet, allergy refers to the outcome of hypersensitivity reactions driven by immunological mechanisms."[9]

"The ability of an individual to produce IgE antibodies in response to various environmental antigens, predominantly glycoproteins, and develop hypersensitivity reactions is termed "atopy." An antigen that triggers a sudden hypersensitivity response is referred to as an "allergen"."[9]

"Pollen allergies often persist throughout the year in tropical regions, while in temperate climates, seasonal variations may result from allergies to small insects, bugs, and specific fungi. Perennial allergy involves continuous or intermittent exposure, leading to chronic or periodic symptoms, with sneezing and rhinitis congestion often dominating the clinical presentation. Understanding the intricacies of these immune responses is pivotal in managing allergic conditions effectively. "[9]

Immune System

"The immune system, which consists of various organs and a range of cell types, is essential in the development of allergic rhinitis. Tissue cells and white blood cells are derived from pluripotent stem cells found in the bone marrow. The lymphoid lineage leads to the formation of T lymphocytes, whereas the myeloid lineage

produces mononuclear and polymorphonuclear cells, as well as platelets and mast cells."[12]

"The initiation of an immune response involves the detection of pathogens or foreign substances, which triggers a response aimed at neutralizing the threat. This complex process is facilitated by a variety of cells and soluble molecules, including lymphokines and cytokines." [12]

"In the case of allergic rhinitis, the immune response is activated by the IgE-mediated identification of specific allergens, leading to the typical symptoms associated with the condition. Gaining a thorough understanding of the intricate interactions of immune components is vital for grasping the mechanisms that underlie allergic rhinitis." [12]

Hypersensitivity[13]

"Hypersensitivity reactions, commonly known as allergic reactions, represent an abnormal and exaggerated response of the immune system to an otherwise harmless antigen. This misdirected immune response can manifest in a spectrum of outcomes, ranging from mild skin irritation to severe and life-threatening anaphylaxis. „[13]

"Etiology: Hypersensitivity reactions are categorized into four main types based on the underlying immunological mechanisms: "[13]

"Type I (IgE-mediated): This prevalent type involves the binding of IgE antibodies to allergens on mast cells and basophils. These binding triggers degranulation and the release of inflammatory mediators such as histamine, leading to immediate symptoms such as rash, wheezing, and, in severe cases, anaphylaxis. "[13]

"Type II (cytotoxic): In this type, IgG or IgM antibodies directly attach to target cells, marking them for destruction by phagocytes. "[13]

"Type III (immune complex): Soluble antigen-antibody complexes precipitate in tissues, activating complement and neutrophils. This cascade leads to inflammation and tissue damage. "[13]

"Type IV (cell-mediated): T lymphocytes, sensitized by a specific antigen, release inflammatory cytokines upon re-exposure. This results in delayed-type hypersensitivity reactions. "[13]

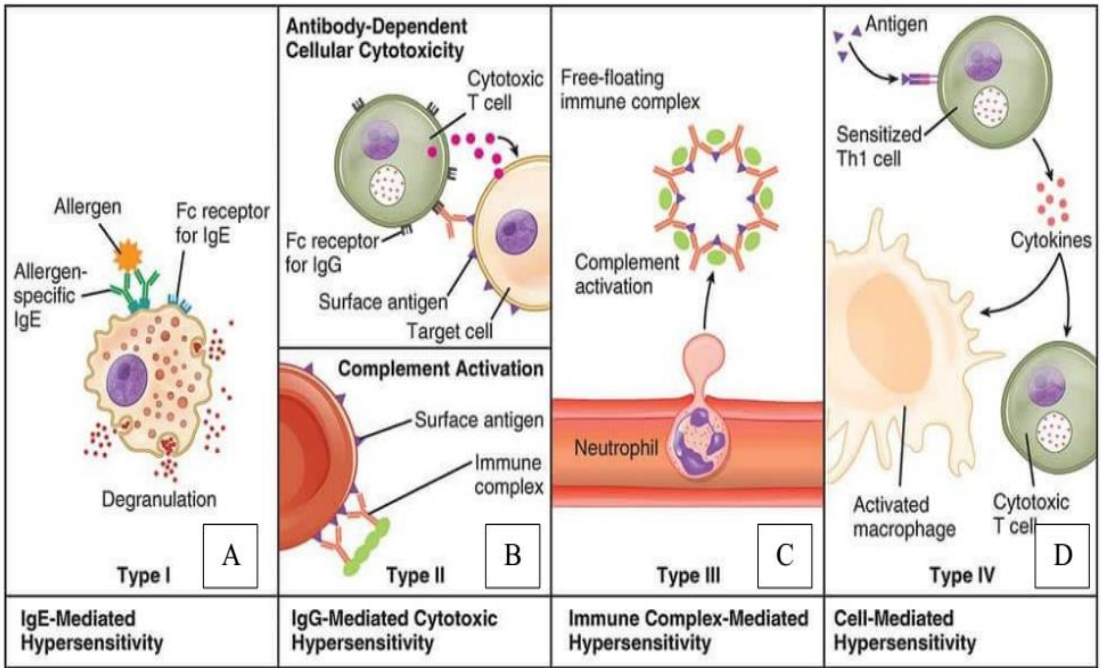

Image 1: Types of Hypersensitivity reactions; A – Type I, B – Type II, C – Type III, D –Type IV[14]

Hypersensitivity Reaction Types[9-11]:

1. Immediate Hypersensitivity Reactions:

- "Mechanism: Caused by the interaction of allergens with IgE antibodies bound to the mast cell surface. "[11]
- "Process: Allergen binding induces mast cell degranulation, leading to the release of vasoactive mediators. "[11]
- "Clinical Examples: Disorders associated with immediate hypersensitivity include asthma, urticaria (hives), anaphylaxis, atopic eczema, allergic rhinitis, and rhinitis. "[11]

2. Delayed Type Hypersensitivity Reactions:

- "Mechanism: T-cell mediated response where antibodies do not play a direct role. "[11]
- "Process: Involves the activation of T cells upon re-exposure to specific antigens, leading to the release of inflammatory cytokines."[11]
- "Clinical Examples: Conditions associated with delayed hypersensitivity include certain contact dermatitis reactions. "[11]

"In the context of allergies, IgE-mediated reactions are paramount, contributing to a spectrum of disorders. These include asthma, characterized by airway inflammation and bronchoconstriction; urticaria, presenting as itchy welts on the skin; anaphylaxis, a severe and potentially life-threatening systemic reaction; atopic eczema, a chronic inflammatory skin condition; and allergic rhinitis, marked by nasal symptoms triggered by allergen exposure. "[11]

"Understanding the distinct mechanisms of immediate and delayed hypersensitivity reactions is crucial for accurate diagnosis and targeted therapeutic interventions. "[11]

Rhinitis: Definition and Classification:

"The definition and classification of rhinitis lack universal consensus, with various terminologies and systems in use. The term "rhinitis" typically suggests an inflammatory condition affecting the mucous layer of the nasal cavity. However, as symptoms may manifest without evident inflammation, the term "rhinopathy" has been proposed as a potentially more accurate descriptor, though it is infrequently utilized. "[11]

1 **ALLERGIC RHINITIS:**

"Allergic rhinitis represents an IgE-mediated hypersensitivity disease affecting the mucous layer of the nasal cavity. Key characteristics include sneezing, itching, watery nasal discharge, and a sensation of nasal obstruction. Diagnosis of inhalant allergies involves a comprehensive evaluation combining patient history, physical examination, and results from skin tests or radioallergosorbent tests (RAST). This integrated approach aids in confirming the presence of allergic rhinitis and identifying specific allergens triggering the immune response. "[11]

Prevalence of Allergic Rhinitis in India:

"In the vast Indian population, a substantial 20-30% grapple with allergic rhinitis, and within this group, 15% further develops associated asthma. Atopy, an inherited predisposition to allergic reactions, is prevalent in 40% of the global population. Notably, the incidence of allergic rhinitis in Western countries ranges

from 1.4% to 39.7%. Allergic rhinitis, often abbreviated as AR, manifests as a symptomatic nasal disorder triggered by an IgE-mediated immune response to various allergens."[15]

Factors Influencing the Development of Allergic Rhinitis[16-18]:

The onset and development of allergic rhinitis hinge on specific factors:

i) Atopic Sensitivity:

- Individuals in an atopic state exhibit heightened sensitivity to particular allergens.
- Atopy refers to a genetic predisposition to produce exaggerated IgE antibody responses to common environmental allergens.

ii) Exposure to Allergens:

- Sensitized individuals must encounter and be exposed to the specific allergen.
- Exposure can occur through inhalation, contact, or other routes, leading to the activation of the immune response.

Primary Cause:

"The primary cause of allergic rhinitis is sensitivity to inhalant allergens. These allergens, which are typically airborne particles such as pollen, dust mites, mold spores, and animal dander, trigger an immune response in individuals with pre-existing atopic sensitivity. This immune reaction, mediated by IgE antibodies, results in the characteristic symptoms of allergic rhinitis, including nasal congestion, sneezing, itching, and watery discharge. Understanding and managing both atopic sensitivity and allergen exposure are crucial aspects of preventing and treating allergic rhinitis."[16]

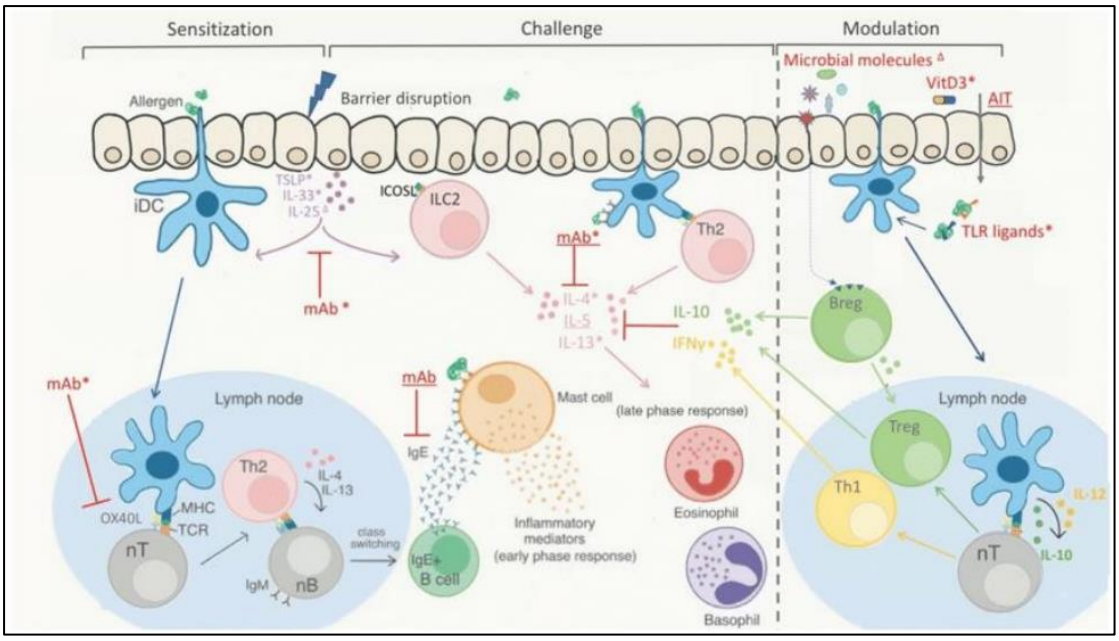

Image 2: "Mechanism of Sensitization in Allergic Rhinitis"[19]

Common Allergens:

Common allergens implicated in allergic rhinitis include:

- Grass pollens
- Tree pollens
- Fungal spores (varies by geographic location and season)

Causative Factors and Risk Factors:

"The causative factors for allergic rhinitis are multifactorial, encompassing both genetic and environmental influences: "[16]

a) "Hereditary Factors: "[16]
b) "A familial history of atopy, characterized by a predisposition to allergic reactions, is a significant risk factor. "[16]

c) "Environmental Exposures: Exposure to environmental factors plays a crucial role in the development of allergic rhinitis. "[16]

d) "Gender: Allergic rhinitis is more common in men. "[16]

e) "Socioeconomic Status: Individuals with lower socioeconomic status may be more susceptible. "[16]

f) "Breastfeeding: Breastfeeding is associated with a potential protective effect against allergic disorders. "[16]

g) "Childhood Use of Antibiotics: Childhood use of antibiotics increases the likelihood of developing allergic disorders. "[16]

h) "Obesity: A body mass index (BMI) higher than 30 increases the likelihood of allergies due to hormonal and T cell function variations. "[16]

i) "Nutrition: Dietary factors, including higher consumption of processed foods, omega-6 fatty acids, and lower consumption of omega-3 fatty acids and fresh foods, influence the development of allergies. "[16]

j) "Stimulant Use: Ethanol intake, as seen with alcohol consumption, can modulate the immune system and cytokine production. "[16]

k) "Lack of Microbial Exposure: Hygienic conditions and the magnitude of allergies are inversely related, with developmental countries having a lower allergy rate than developed countries. "[16]

l) "Autoimmune Disorders: Th2 immune complex responses in allergic conditions are influenced by a well-developed Th1 immune response. "[16]

m) "Smoking: Smoking is associated with an increase in IgE levels. "[16]

Chemical Mediators of the Immune System in Allergic Rhinitis:

1. Histamine:

1. "Histamine, found in mast cells and basophils, is a primary mediator of allergic reactions. "[16]
2. "Actions include the contraction of smooth muscles, leading to airway constriction, wheezing, increased secretion production, heightened vascular reactivity, and enhanced permeability of fluid into tissues. "[16]
3. "Nasal mucosa has three histamine receptor subtypes, with H1 and H2 contributing to allergic symptoms like nasal itching and mucosal edema. "[16]

2. Prostaglandins:

1. "Mast cell degranulation results in the production of prostaglandins. "[16]
2. "Prostaglandin-D2 specifically causes rhinitis, redness, chemosis, mucus discharge, and eosinophilic infiltration. "[16]
3. "Other prostaglandins like PGE2 and PGI2 also contribute to allergic symptoms. "[16]

3. Leukotrienes:

- "Leukotrienes exist in two types: cysteinyl and non-cysteinyl. "[16]
- "They play a role in maintaining chronic inflammatory responses, and allergic disorders are associated with elevated concentrations of leukotrienes. "[16]

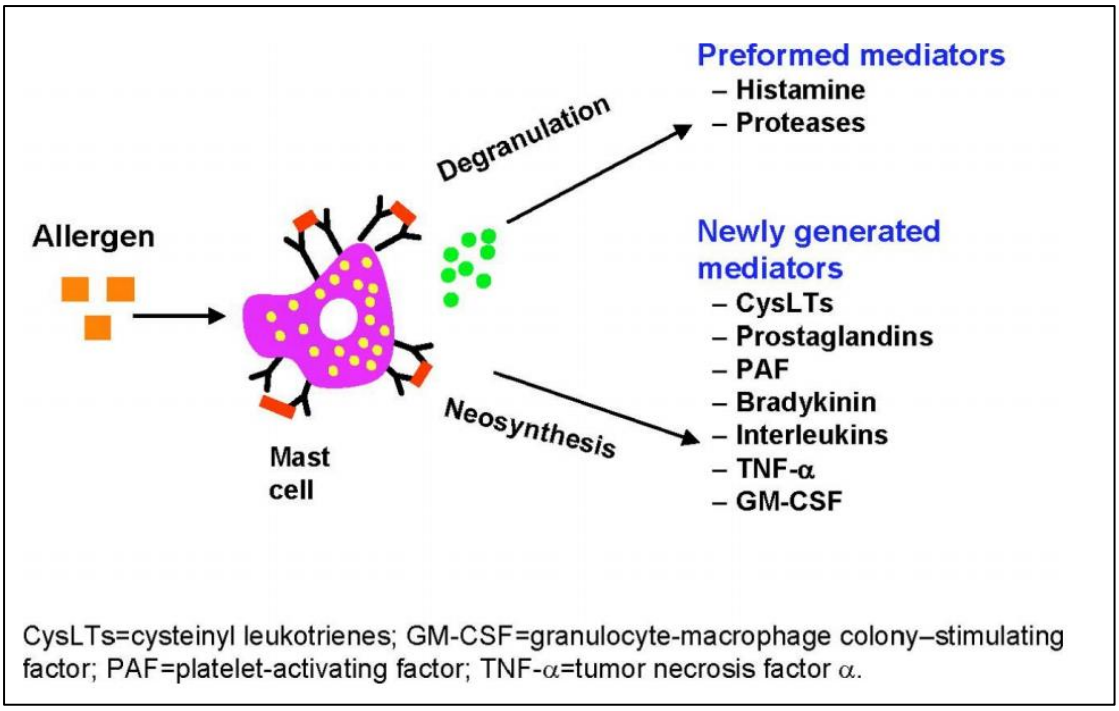

Image 3: "Chemical Mediators of Allergic Rhinitis"[20]

Pathogenesis of Allergic Rhinitis:

"Allergic rhinitis is characterized by an immediate hypersensitive, IgE-mediated response of the nasal mucosa, initiating an inflammatory process. "[20]

1. "Allergen Intrusion: When an allergen enters the body, it is recognized by Antigen Presenting Cells (APCs). "[20]
2. "Antigen Presentation: The allergen is presented as an antigen-Major Histocompatibility Complex-II (MHC-II) complex to T lymphocytes. "[20]
3. "T Cell Activation: T cell activation occurs through three signals: Association of the MHC-peptide complex with the T cell receptor (TCR). "[20]
4. "Binding of CD28 to B7 (CD80/CD86) expressed on the APCs (co-stimulation). "[20]
5. "Differentiation of activated T cells into Th2 cells in the presence of the Interleukin-4 cytokine. "[20]

"This activation of Th2 cells initiates an immune cascade leading to the release of various chemical mediators, such as histamine, prostaglandins, and leukotrienes. These mediators contribute to the characteristic symptoms of allergic rhinitis, including nasal itching, mucosal edema, and increased secretion production. ,,20

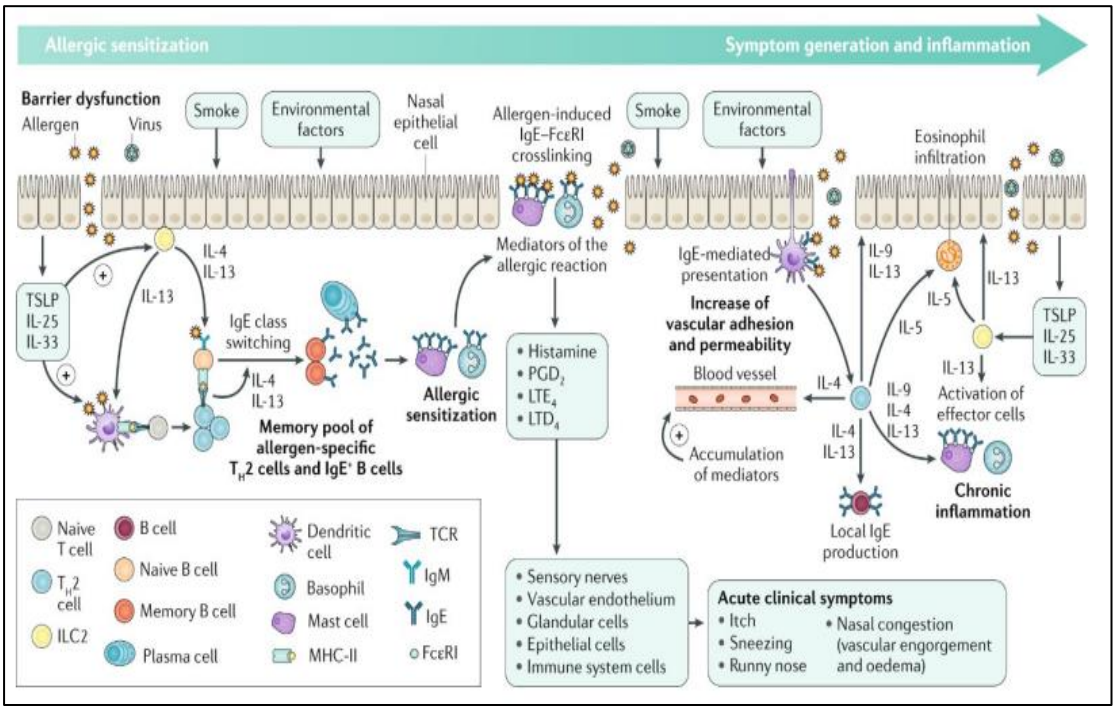

Image 4: "Immunopathogenesis of Allergic Rhinitis"[21]

"Upon initial exposure to an allergen, the human immune system initiates sensitization, generating specific IgE antibodies against the allergen. Upon subsequent exposure to the same allergen, these antibodies, now bound to mast cells, interact with soluble antigens that penetrate the nasal mucosa. This interaction triggers mast cell degranulation, leading to the release of chemical mediators. The immediate response manifests as classical allergic rhinitis symptoms, including nasal discharge, itching, sneezing, and congestion (Figure ^). Prolonged and continuous exposure to the allergen further exacerbates the allergic response, leading to the invasion of migratory

cells into the nasal mucosa. This heightened sensitivity, known as nasal hyperreactivity, extends the reactivity of the nasal mucosa to various non-specific irritants. Common irritants include perfumes, tobacco smoke, traffic fumes, domestic sprays, and bleach. Understanding this sequence of events sheds light on the development and persistence of allergic rhinitis symptoms, offering valuable insights for effective management. "[21]

Phases of Allergic Rhinitis: The Immunological Response

"The intricate dance between allergens and the immune system unfolds in distinct phases during allergic rhinitis. The initial encounter with an allergen triggers a cascade of events, initiating the early phase of allergy. This phase is marked by the cross-linking of multiple adjacent IgE antibodies, activating mast cells. The ensuing degranulation of these activated mast cells unleashes inflammatory mediators within a brisk 30-minute timeframe, sparking the early allergic reaction. As the immunological drama continues, a second act emerges, known as the late phase reaction. This phase unfurls 4-6 hours after allergen exposure and can persist for 1-2 days. "[21]

"During this extended period, the nasal mucosa becomes a battleground, witnessing the infiltration of various immune cells—neutrophils, eosinophils, basophils, macrophages, and Th2 cells. This infiltration is orchestrated in response to cytokines released by the initially activated mast cells. "[21]

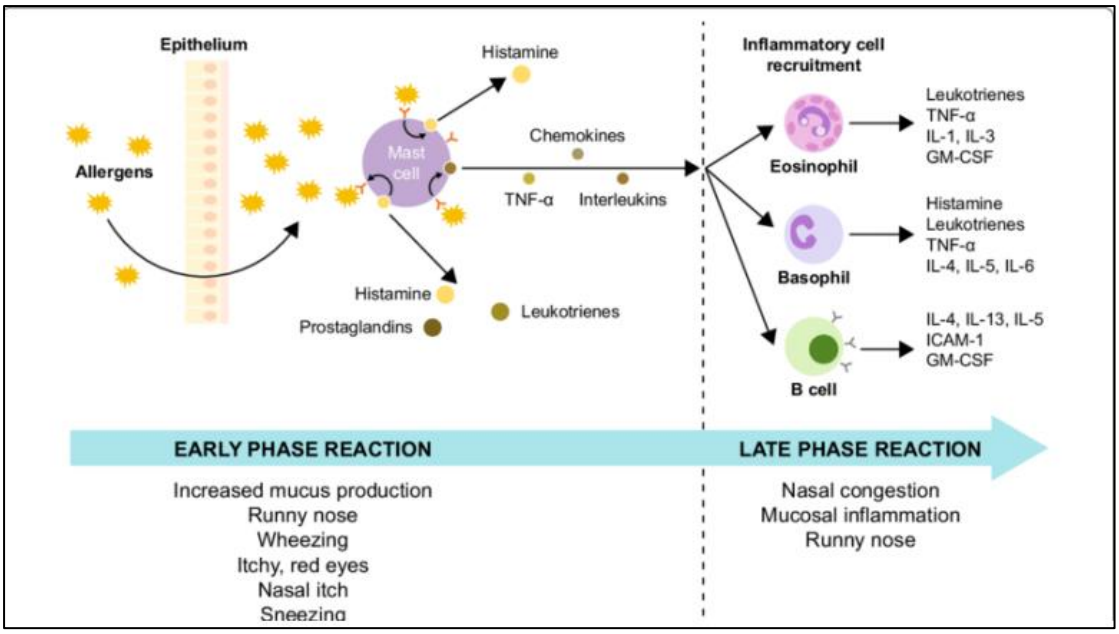

Image 5: "Phases of Allergic Rhinitis"[22]

The Interplay of Environmental Factors in Allergic Rhinitis

"In the intricate orchestration of allergic rhinitis, environmental pollutants assume a pivotal role, exhibiting a dual capacity for both provocation and modulation. Elevated concentrations of irritants possess the potential to induce acute nasal manifestations in susceptible individuals. Paradoxically, diminished exposure can also incite clinical symptoms, as observed in conditions such as 'idiopathic' rhinitis and the heightened reactivity of nasal mucosa, particularly in individuals with allergic rhinitis-associated hyperreactivity. "[22]

"Exhaust fumes, containing nitrogen dioxide, ozone, and diesel particulates, constitute a complex amalgamation of environmental challenges. Notably, diesel particulates exhibit adjuvant properties, amplifying antibody production in vivo as demonstrated in animal studies. Recent research suggests that pre-exposure to a combination of nitrogen dioxide and ozone may accentuate subsequent pollen-induced immediate symptoms of rhinitis. This intricate interplay between pollutants

and the immune system introduces a layer of complexity in comprehending the triggers and modifiers influencing the manifestation of allergic rhinitis within a medical context. ”[22]

Clinical Features

“Accurate clinical diagnosis and assessment of dominant symptoms are crucial for the effective treatment of rhinitis. Allergic rhinitis diagnosis is generally straightforward and relies on a detailed clinical history. While various allergens can trigger allergic rhinitis, the clinical presentation remains consistent as the same mediators are released regardless of the allergen type. A thorough evaluation, including a detailed history, local examination of the nose, and skin prick tests, is essential for all patients with rhinitis symptoms. In some cases, additional tests such as flexible and rigid nasal endoscopy, mucociliary clearance studies, and immunological tests may be necessary. It's important to recognize that the nose serves as a 'window' to the respiratory tract and can provide insights into systemic diseases elsewhere in the body. ”[21]

Symptoms

“Allergic rhinitis is characterized by distinct symptoms, including sneezing, watery rhinorrhea, itching of the nose, and nasal obstruction. The manifestation of pruritus, nasal obstruction, sneezing, and rhinorrhea is attributed to sensory nerve stimulation, mucosal edema, and increased mucus secretions. These symptoms typically emerge within 5-15 minutes of exposure to the allergen. Sneezing often occurs in spasms of 10-20 episodes, potentially leading to patient exhaustion. Profuse watery rhinorrhea is a common feature. Itching of the nose and nasal obstruction,

usually of moderate severity, are prevalent and bothersome complaints associated with allergic rhinitis."[21]

Signs

"During a physical examination for allergic rhinitis, it is crucial to assess beyond the nasal region. The examination should encompass facial features, eyes, ears, oropharynx, neck, lungs, and skin. Thorough scrutiny is necessary to identify physical findings that might indicate a systemic disease linked to rhinitis. This comprehensive approach ensures that potential signs of allergic rhinitis are not limited to the nasal area alone but are considered in the broader context of the patient's overall health."[21]

Nasal Examination in Allergic Rhinitis

"In the examination of the nose for allergic rhinitis, certain distinctive features may be observed. The presence of a "nasal crease," a horizontal line across the lower half of the bridge of the nose, is indicative of repeated upward rubbing of the nose tip, known as the "allergic salute." Swelling or boggy appearance of the nasal turbinates may be noted, and they often appear pale. Allergic rhinitis is typically associated with thin and watery nasal secretions. It's important to distinguish these characteristics from the thick and purulent secretions commonly seen in sinusitis, although allergic rhinitis can occasionally present with thick and colored mucus. Additionally, the nasal cavity should be examined for the presence of polyps and any deviation in the nasal septum." [23]

Ear, Eye, and Oropharynx Examination in Allergic Rhinitis[24]

"During the examination of individuals with allergic rhinitis, certain features may be observed in the ears, eyes, and oropharynx. In the ears, tympanic membrane retraction, the presence of air-fluid levels, or bubbles in the tympanic cavity, and restricted mobility of the tympanic membrane may be associated with allergic rhinitis, especially in cases involving Eustachian tube dysfunction or secondary otitis media."[24]

"Ocular examinations might reveal swelling of the palpable conjunctiva with excessive tear production. Dennie–Morgan lines, which are prominent creases below the inferior eyelid, are commonly associated with allergic rhinitis. "[24]

"The term "cobblestoning" is used to describe streaks of lymphoid tissue on the posterior pharynx, a characteristic often observed in allergic rhinitis. Neck examination should include a check for evidence of lymphadenopathy or thyroid disease. When assessing the lungs, characteristic findings of asthma should be looked for. Finally, a thorough examination of the skin is crucial to evaluate for possible atopic dermatitis, another condition often seen in conjunction with allergic rhinitis. "[24]

Complications of Allergic Rhinitis: A Multifaceted Impact

"Allergic rhinitis, beyond its symptomatic expression, can engender a spectrum of complications, underscoring the systemic ramifications of this immune-mediated disorder. Otitis media, marked by inflammation of the middle ear, stands as a notable complication, with atopy and allergic rhinitis amplifying susceptibility to this condition. "[21]

"Chronic sinusitis, characterized by persistent inflammation of the sinuses, emerges as a sequela of perennial allergic rhinitis, accentuating symptoms such as nasal discharge and obstruction. The ethmoidal region, in particular, may witness the development of bilateral polypi, a distinctive feature associated with chronic perennial allergic rhinitis. "[21]

"The intricate interplay between allergic rhinitis and bronchial asthma unfolds as a clinical coexistence. This tandem occurrence arises from the shared attribute of inducing hyperresponsiveness in both nasal mucosa and the airway. Addressing rhinitis becomes pivotal in ameliorating symptoms of bronchial asthma, emphasizing the interconnectedness of these respiratory conditions and the potential for comprehensive therapeutic interventions. "[21]

Investigations[21]:

Unraveling the intricacies of allergies demands a nuanced approach, and our arsenal of diagnostic tools navigates this intricate terrain:

1. Skin Prick Test:

"The allergy skin test, also known as immediate hypersensitivity testing, is an in vivo method employed to determine immediate (IgE-mediated) hypersensitivity to specific allergens. This diagnostic approach involves the percutaneous introduction of an extract of a suspected allergen, inducing an immediate wheal and flare reaction in the skin's early phase. Skin prick testing, a common method within this approach, is conducted by placing a drop of allergen extract on the skin and then scratching or pricking a needle through the epidermis under the drop. The size of the resulting

wheal and flare reaction provides an indication of the degree of sensitivity to the allergen. Alternatively, the allergen can be introduced intradermally. "[21]

"Skin prick tests are favored over scratch or intradermal tests due to their higher reproducibility, lower risk, and reduced likelihood of false-positive responses. These tests are cost-effective, accurate, rapid, and allow for the assessment of sensitivity to a variety of allergens in a single session. "[21]

"In vitro allergy tests, such as the radioallergosorbent test (RAST), measure the amount of specific IgE to radioisotope-labeled allergens in a blood sample. While these tests offer an estimate of allergic sensitivity based on the quantity of specific IgE produced, their sensitivity and specificity may not match the accuracy of skin testing. "

2. Serum Ig E Test:

- Diagnostic linchpin highlighting elevated Ig E levels.
- A systemic indicator of allergic predisposition.

3. Nasal Cytology:

- Microscopic scrutiny of nasal smears.
- Grunwald or Giemsa staining reveals inflammatory cells and bacteria.
- Provides insights into nasal pathology.

4. Nasal Swabs for Bacteriology and Viral Testing:

- Complements the diagnostic landscape.
- Comprehensive view of nasal responses.
- Guides tailored management strategies.

5. Nasal Provocation Test in Allergic Rhinitis

"The nasal provocation test is a method used to challenge the nasal mucosa by introducing a small amount of allergen at the end of a toothpick, which the patient then sniffs into each nostril. The objective is to observe whether allergic symptoms are reproduced as a response to the allergen exposure. "[24]

"While the nasal provocation test can provide valuable insights, it has limitations. This method is considered crude and is technically demanding, as it necessitates testing one agent at a time. Additionally, the test is time-consuming. Furthermore, there is a potential risk of precipitating severe reactions such as anaphylaxis or bronchospasm, making careful monitoring and supervision essential during its administration. Despite these challenges, the nasal provocation test can contribute to a more comprehensive understanding of allergic rhinitis in specific clinical contexts. "[24]

Causes of SNHL in patients with Allergic rhinitis

"Allergic rhinitis typically affects the nasal passages, causing symptoms like sneezing, congestion, and runny nose. It can also impact the **Eustachian tube**, which connects the middle ear to the back of the throat. While allergic rhinitis is more commonly associated with **conductive hearing loss** due to middle ear issues, it may indirectly contribute to **sensorineural hearing loss (SNHL)** in specific scenarios. "[24]

1. Eustachian Tube Dysfunction and Middle Ear Problems

- "Allergic rhinitis can lead to **inflammation** and swelling of the Eustachian tube, causing it to become blocked. "[24]
- "This blockage can result in **negative pressure** in the middle ear, leading to fluid buildup (**otitis media with effusion**). "[24]
- "Persistent middle ear issues can cause changes in the inner ear's environment, potentially damaging the **cochlea** and leading to SNHL over time. "[24]

2. Inner Ear Inflammation

1 "Allergic reactions can trigger **systemic inflammation**, which may extend to the inner ear (cochlea). "[24]

2 "Inflammatory mediators like **histamines** and **cytokines** released during an allergic response can affect the blood supply to the cochlea, leading to **ischemia** (reduced blood flow) and potential damage to the **sensory cells** (hair cells). "[24]

3. Autoimmune Response in the Inner Ear

- "Chronic allergies may induce an **autoimmune response**, where the body mistakenly attacks its own inner ear cells. "[24]
- "This autoimmune process can lead to **cochlear damage**, resulting in sensorineural hearing loss. "[24]

4. Vascular Changes:

a. "Allergic inflammation may impair cochlear blood flow, reducing oxygen supply and causing sensory cell damage. "[24]

5. Ototoxic Medications

- "People with severe allergic rhinitis may use **antihistamines** and **decongestants** frequently. "[24]

- "Long-term or high-dose use of certain medications, particularly **nasal sprays containing corticosteroids**, can potentially have ototoxic effects (though rare). "[24]

6. Chronic Sinus Infections and Spread of Inflammation

- "Allergic rhinitis can lead to chronic sinusitis, which may cause inflammation to spread to the inner ear structures. "[24]

- "This inflammation can impact the **cochlear nerve** or the delicate structures of the inner ear, resulting in SNHL. "[24]

A study done by ***Zeng B et al***[25] (2024) did "systemic review study with aim to analyze clinical data on the coexistence and potential causal interaction between allergic diseases and inner ear conditions. The epidemiologic evidence found overwhelmingly supports an association between allergic disease and particular inner ear disorders represented by a high prevalence of allergic reactions in some patients with Ménière's disease (MD), idiopathic sudden sensorineural hearing loss (ISSHL), and acute low-tone hearing loss (ALHL). In addition, patients with MD, ISSHL, and ALHL had higher levels of total serum IgE than healthy subjects. Finally, in some cases, changes in cochlear potential may have been induced by antigen exposure, while desensitization alleviated allergy and inner ear-related symptoms. The exact mechanism of interaction between the auditory/vestibular and immune systems is not

fully understood, and further clinical and basic research is needed to understand the relationship between the two systems fully. ”[25]

A study by ***Kumar S et al***[26] (2023), “over 12 months, focused on the impact of allergic rhinitis on hearing in patients aged 10-55. A total of 70 patients were included, with 44 diagnosed with perennial allergic rhinitis and 26 with seasonal allergic rhinitis , revealed that allergic rhinitis was most prevalent in younger adults (ages 10-30), predominantly among males, comprising 67.43% of cases. Perennial allergic rhinitis was more common (62.86%) than seasonal (37.14%). Notably, patients with perennial allergic rhinitis experienced more hearing impairment (28.41%) compared to those with seasonal rhinitis (15.4%), primarily of the conductive type. The authors concluded that both types of allergic rhinitis negatively affect Eustachian tube function, potentially leading to increased middle ear effusion and otitis media. Effective diagnosis and management of allergic rhinitis are crucial to preventing these middle ear complications. ”[26]

Acharya S et al[27] (2022) “did study with aim to assessment of sensorineural hearing loss incidence in allergic rhinitis. Around 60 patients with clinically diagnosed allergic rhinitis were compared with age and gender matched controls. All participants were subjected for pure tone audiometry; distortion product otoacoustic emission and tympanometry. Facts and statistics were analysed by chi-square and t- test with significance at $p<0.05$. Study population had sensorineural hearing loss which was in high frequencies, distortion product otoacoustic emissions revealed low distortion product and mostly “A” type tympanogram when compared with the controls. They found patients having allergic rhinitis are prone to sensorineural hearing loss. ”[27]

A study done by ***Sahni D et al***[28] (2022) to assess "the audiological profile in AR and effect of AR on inner ear functions. 100 cases of AR patients (55 males, 45 females, mean age group 21–30 years) and 100 controls (65 males, 35 females, mean age group 41–50 years) were enrolled in study. Thirty two patients among case group had sensorineural hearing loss, pronounced at 4000 and 8000 Hz frequencies. 18 patients showed conductive hearing loss in the form of type B or type C tympanogram. 32 patients of AR patients showed unusual oto-acoustic emission test. We found higher prevalence of high frequency sensorineural hearing loss in pure tone audiometry and abnormal OAEs in patients having upper airway allergy. The likely seat of damage appears to be the inner ear as evidenced by recordings of OAE in allergic patients. "[28]

Mishra S et al[29] (2020) conducted the study "with objective to assess the effect of allergic rhinitis on Eustachian tube function (ETF) and middle ear ventilation among 100 cases of AR. They observed statically significance increase in the proportion of type-A tympangram cases at first visit was 42ears [21%] when compared second visit was176 ears [88%]. At first visit value of middle ear pressure ranged from -150 to 4 decapascal (daPa), means was -123 and at second visit middle ear pressure was from -75 to 4 and means was -11, with a statically significant improvement in middle ear pressure. Those patients had allergic rhinitis and poor ETF, seen significant improvement ETF at 6 week after anti allergic treatment.($P<0.05$). They find out that allergic rhinitis has a definite relationship with ETF. Anti- allergic treatment has a favorable effect on the middle ear pressure and ETF. "[29]

A study done by ***Dwarakanath VM et al***[30] (2019) with "aim to develop an audiologic profile in individuals with allergic rhinitis. Fifteen individuals with allergic rhinitis and 15 individuals without any history of allergic rhinitis were subjected for pure-tone

audiometry, distortion product otoacoustic emission, auditory brainstem response, tympanometry, auditory reflex thresholds, and Eustachian tube function tests. All these tests were repeated after 1 month on the same individuals. Results indicated poorer pure-tone thresholds with reduced DP amplitude compared to normal. This can be attributed to the changes in outer hair cell function due to allergens (Singh et al., 2011). None of the individuals exhibited any middle ear or Eustachian tube dysfunction. As the pure-tone thresholds or distortion product otoacoustic emission amplitude did not improve overmedication, the effects on inner ear function might not be seasonal. It can be concluded from the present study that individuals with AR are more prone to sensorineural hearing loss. "[30]

MATERIALS AND METHODS

i. **Study Setting**: Study was done at KLES Dr Prabhakar Kore Charitable hospital, Belagavi, Karnataka, India.

ii. **Study design:** - It was a cross-sectional study conducted at ENT & HNS department of KLES Dr Prabhakar Kore Charitable hospital, Belagavi, Karnataka, India.

iii. **Study subject:** - All the cases of clinically diagnosed allergic rhinitis with symptoms more than 4 to 6 weeks at ENT & HNS department of KLES Dr Prabhakar Kore Charitable hospital, Belagavi during the study period.

iv. **Inclusion criteria:-**

a Age 10 to 50 years.

b Symptoms duration more than 4 to 6 weeks.

c Who gave consent to participate in the study.

v. **Exclusion criteria:** -

A. Patients with a history of factors causing hearing loss like use of ototoxic agents, metabolic and systemic diseases.

B. Noise exposure.

C. History of neurological factors causing hearing loss.

D. Ear diseases causing hearing loss like tympanic membrane perforation, cholesteatoma, tumors of middle ear and mastoid, acoustic neuroma.

5. **Sampling Technique :-**Convenient Sampling

6. **Study period :-** one years

7. **Sample Size:**

Sample size at 95% confidence intervals, 20% tolerable error, 10% attrition is

$$n = \frac{Z^2_{1-\alpha/2} \cdot pq}{(20\% \, of \, p)^2} \times 1.10$$

N = 74.9

Required sample :- 75 patients with allergic rhinitis. Where,

p = 60%,

q = 100 - p = 40

Z_1-α/2 = 1.96

Here: - p is the percentage of population revealed with sensorineural hearing loss in PTA of patients with allergic rhinitis.

8. <u>Data Collection: -</u>

1. All the patients with allergic rhinitis, who satisfy the inclusion criteria were taken for our study purpose.

a. Written Informed Consent was taken. After approval from the institution's ethical committee, written, informed, valid consent was obtained from all patients qualifying the inclusion criteria after explaining the study protocol.

b. The research was submitted to the Institutional Ethics Committee (IEC) for ethical approval, and once clearance was granted, the study commenced.

c. All chosen participants were contacted and met in person to discuss the study details following the acquisition of informed consent

d. Strict measures were taken to ensure confidentiality throughout the survey process and in the handling of the information shared by each respondent.

e. Patients with allergic rhinitis, age and who satisfy the inclusion criteria has been enrolled in the study.

f. Patients, who suit the selection criteria has been identified .

g. A thorough history has been obtained and complete examination of the ear, nose and throat has been performed.

h. Pure tone audiometry, Distortion product otoacoustic emission (DPOAE) has been performed and assessed for sensorineural hearing loss

i. Data analysis: The collected data was input into an Excel spreadsheet, and analysis was performed using Epi Info 7.2 software.

j. Statistical method: The data was cleaned, validated, and analyzed with Epi Info 7 software.

k. Descriptive Statistics: For continuous variables, the range, mean, and standard deviation were computed, while proportions and percentages were determined for categorical variables.

l. Bi-Variate analysis: To assess the relationship between dependent and independent variables, chi-square tests and Student's t-tests were applied as appropriate.

3. **Ethical issue:-** Institutional Ethics Committee permitted to carry out study. All the information collected was strictly used for study purpose and confidentiality was strictly maintained. This was also ensured to study participants before starting study. The Consent Form and Participant Information Sheet attached as annexures.

RESULTS

TOTAL SAMPLE SIZE:-75

Table 1: Age distribution of study participants [N=75]

Age Group (in year)	Number	%
10-20	23	30.7
21-30	25	33.3
31-40	13	17.3
41-50	14	18.7
Mean ± SD	28.0 ± 11.2	

Figure 1: Age Distribution

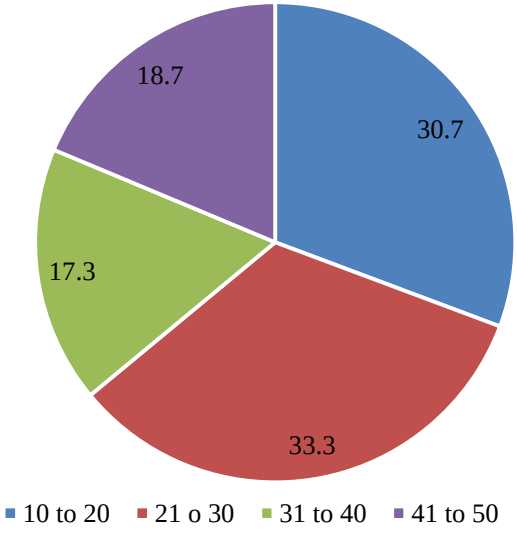

Table 1 and figure 1 shows that 30.7%, 33.3%, 17.3%, 18.7% study participants belonged to age group 10-20, 21-30, 31-40, 41-50 years respectively. Mean age was 28 years with 11.2 SD.

Table 2: Gender distribution of study participants [N=75]

Gender	Number	%
Male	48	64.0
Female	27	36.0

Figure 2: Gender Distribution

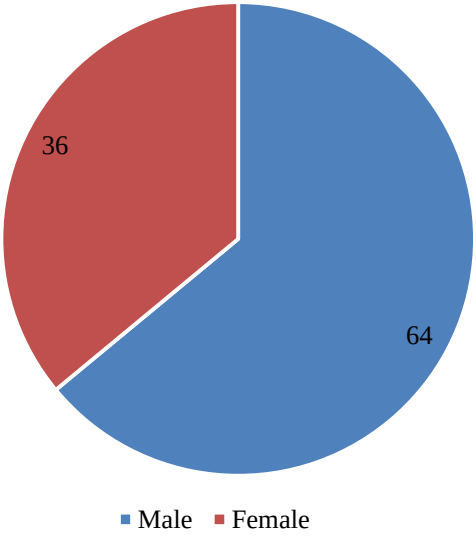

Table 2 and figure 2 shows that 64% & 36% study participants were male and female respectively.

Table 3: Duration of symptoms among study participants [N=75]

	Duration of Symptoms (in year)
Mean ± SD	2.82 ± 3.1

Table 3 shows that mean duration of symptoms was 2.82 years with 3.1 SD.

Table 4: Symptoms distribution of study participants [N=71]

Symptoms	Number	%
Nasal block	68	90.7
Recurrent Sneezing	46	61.3
Running nose	71	94.7
Itchy/watering eyes	28	37.3
Sleep disturbance	23	30.7
Impairment of daily activities	33	44.0

Figure 3: Symptoms Distribution

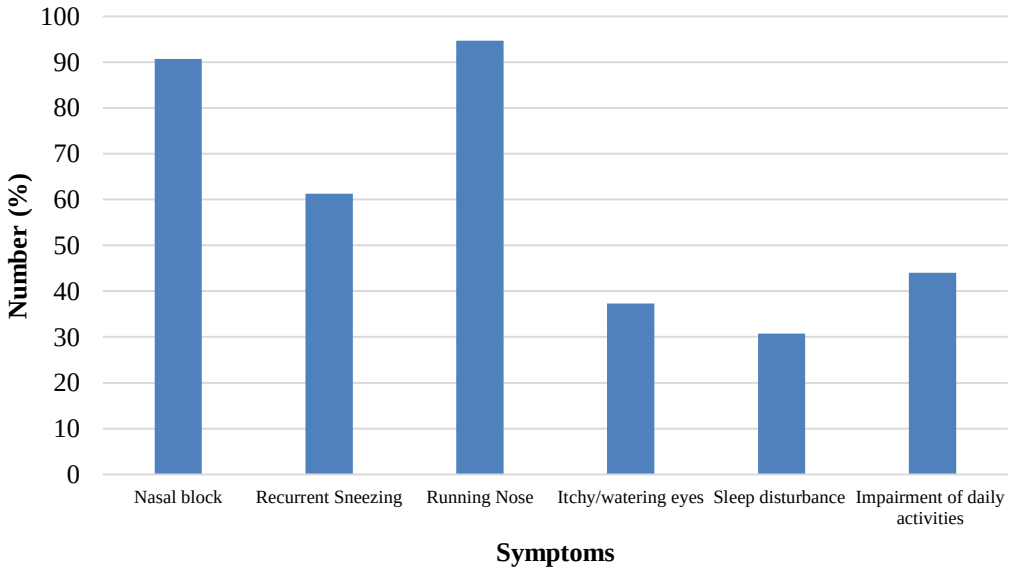

Table 4 and figure 3 shows that 90.7%, 61.3%, 94.7%, 37.3%, 30.7%, 44.0% study participants noted with symptoms like Nasal block, Recurrent Sneezing, Running Nose, Itchy/watering eyes, Sleep disturbance, Impairment of daily activities respectively.

Table 5: Hearing Loss Classification [N=75]

Hearing Loss Classification (in dB)	Right Ear		Left Ear		P value*
	Number	%	Number	%	
0-25 (normal)	39	52.0	46	61.3	0.25
26-40 (mild)	20	26.7	19	25.4	0.85
41-55 (moderate)	11	14.7	7	9.3	0.31
56-70 (moderately severe)	5	6.6	3	4.0	0.47
71-90 (severe)	0	0.0	0	0.0	--
>90 (profound)	0	0.0	0	0.0	--

* - Chi-square Test

Figure 4: Hearing Loss Classification

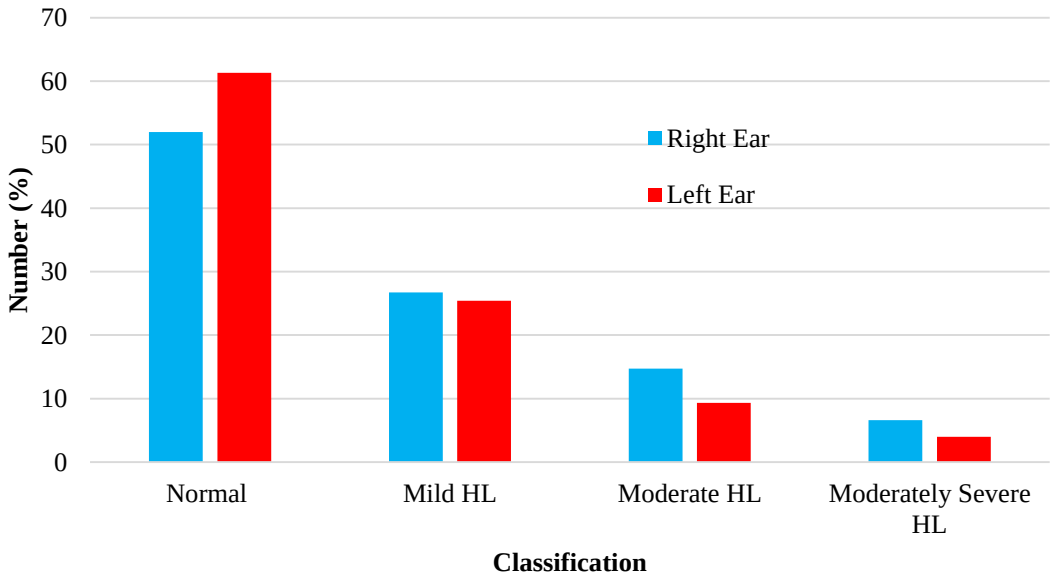

Table 5 and figure 4 shows that HL classification like 0-25 dB (normal), 26-40 dB (mild), 41-55 dB (moderate), 56-70 dB (moderately severe), 71-90 dB (severe), >90 dB (profound) noted in 52.0%, 26.7%, 14.7%, 6.6% cases in right side ear and 61.3%, 25.4%, 9.3%, 4.0% cases in left side ear respectively. The distribution of cases according to HL classification between right & left side ear was statistically not significant ($p>0.05$).

Table 6: Type of Hearing Loss [N=75]

Type Hearing Loss	Right Ear		Left Ear		P value*
	Number	%	Number	%	
Normal	34	45.3	45	60.0	0.07
SNHL	32	42.7	27	36.0	0.4
CHL	6	8.0	2	2.7	0.12
Mixed	3	4.0	1	1.3	0.31

* - Chi-square Test

Figure 5: Type of Hearing Loss

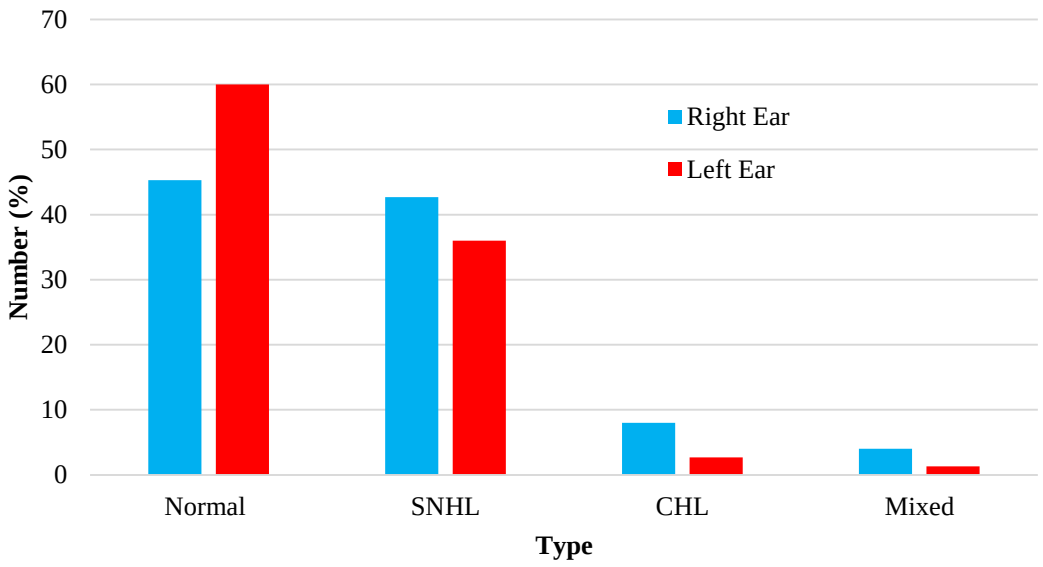

Table 6 and figure 5 shows that Normal, SNHL, CHL, Mixed type of HL noted in 45.3%, 42.7%, 8.0%, 4.0% cases in right side ear and 60.0%, 36.0%, 2.7%, 1.3% cases in left side ear respectively. The distribution of cases according to type of HL between right & left side ear was statistically not significant (p>0.05).

Table 7: Observations of Distortion Product Otoacoustic Emissions (DPOAEs) among study participants [N=75]

DPOAE	Right Ear		Left Ear		P value*
	Number	%	Number	%	
Present	33	44.0	41	54.7	0.19
Abnormal/absent	42	56.0	34	45.3	

* - Chi-square Test

Figure 6: Observations DPOAE

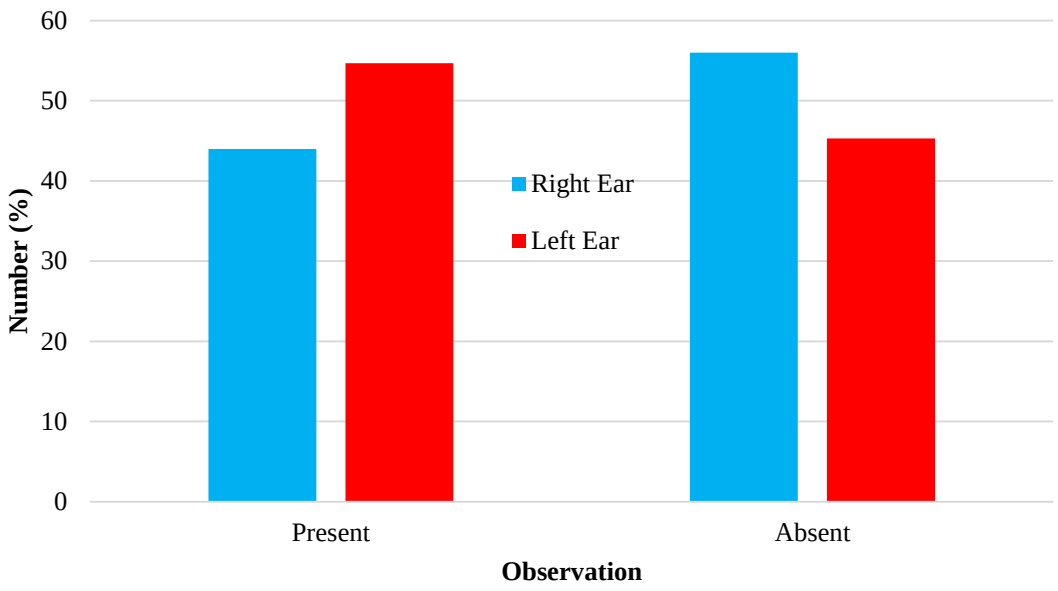

Table 7 and figure 6 shows that 56% cases in right side ear and 45.3% cases in left side ear were noted with abnormal DPOAEs.

Figure 7: Observations of B/L absent DPOAE

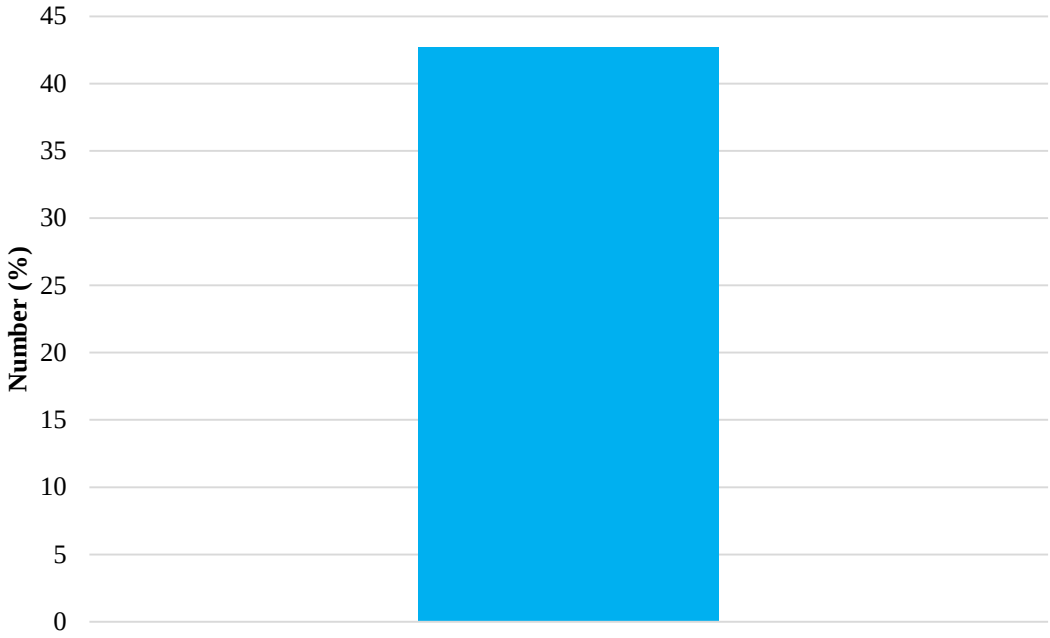

Figure 7 shows that 42.6% patients in left side shows B/L abnormal or absent DPOAEs.

Table 8: Association between duration of disease and type of HL (Right side) [N=75]

Type Hearing Loss	Duration of AR (in year)	P value*
Normal (n=34)	0.97 ± 1.1	**0.0001**
CHL (n=32)	2.47 ± 2.8	
SNHL (n=6)	4.68 ± 3.5	
Mixed (n=3)	4.66 ± 0.6	

* - One-way ANNOVA Test

Figure 8: Duration of disease Vs Type of HL (Right side)

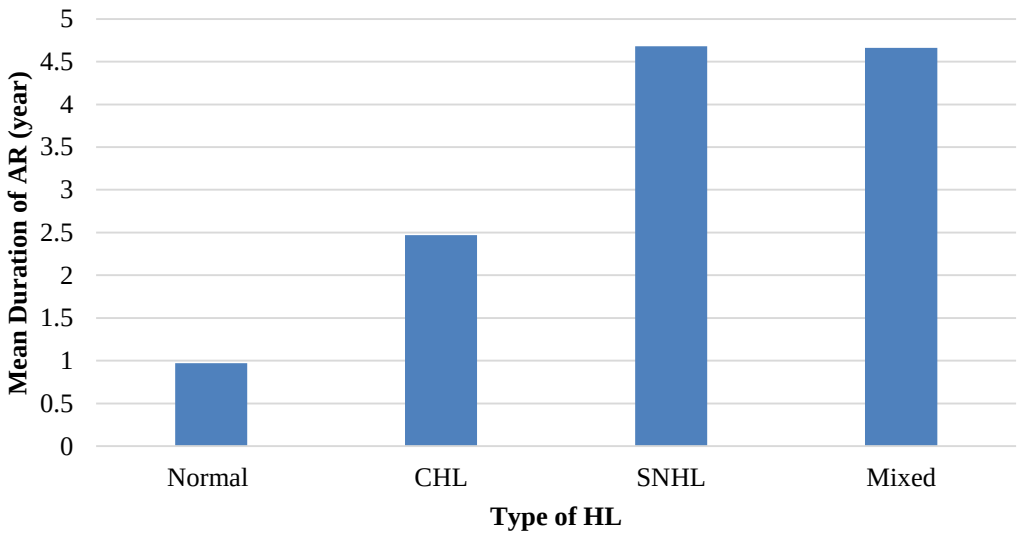

Table 8 and figure 8 shows that on right side ear cases, mean duration of AR was 0.97 years ± 1.1 SD, 2.47 years ± 2.8 SD, 4.68 years ± 3.5 SD, 4.66 years ± 0.6 SD noted in cases with normal, CHL, SNHL, mixed hearing loss respectively. The difference in mean duration of AR according to type of HL was statistically significant ($p<0.05$).

Table 9: Association between duration of disease and SNHL incidence (left side) [N=75]

Type Hearing Loss	Duration of AR in months	P value*
Normal (n=45)	1.29 ± 1.4	**0.0001**
CHL (n=27)	3.85 ± 4.5	
SNHL (n=2)	5.26 ± 3.5	
Mixed (n=1)	4.0	

* - One-way ANNOVA Test

Figure 9: Duration of disease Vs Type of HL (Left side)

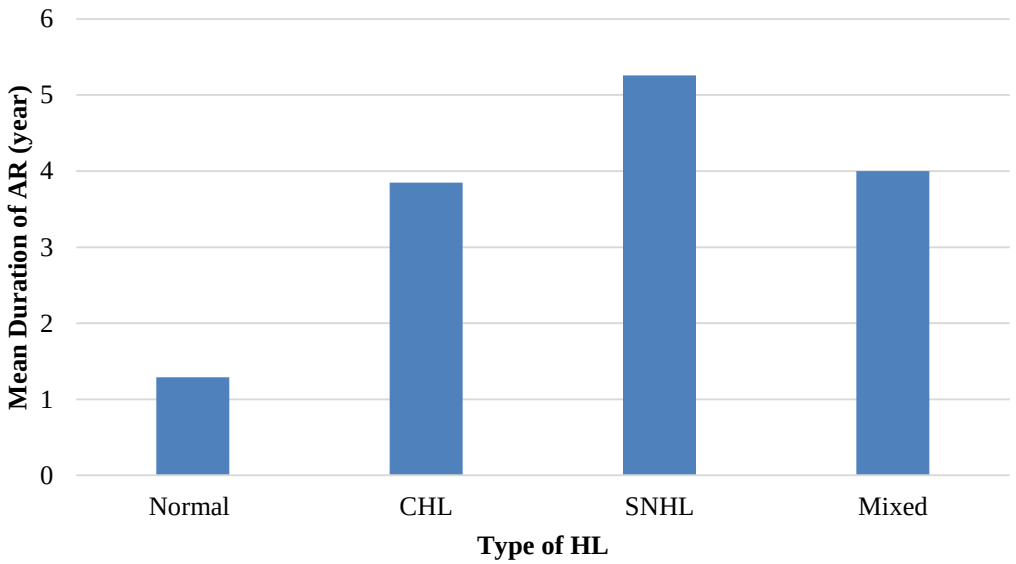

Table 9 and figure 9 shows that on left side ear cases, mean duration of AR was 1.29 years ± 1.4 SD, 3.85 years ± 4.5 SD, 5.26 years ± 3.5 SD, 4 years noted in cases with normal, CHL, SNHL, mixed hearing loss respectively. The difference in mean duration of AR according to type of HL was statistically significant ($p<0.05$).

DISCUSSION

We carried out cross-sectional study in 75 cases of clinically diagnosed allergic rhinitis with symptoms >4 to 6 weeks at ENT & HNS at KLES Dr Prabhakar Kore Charitable hospital, Belagavi with aim to know the incidence of SNHL in patients with allergic rhinitis. Inclusion criteria was Age 10 to 50 years, symptoms duration more than 4 to 6 weeks.

In table 1 present study shows that highest number of participants belonged to age group 21 to 30 years (33.3%) followed by 10 to 20 years (30.7%). Mean age was 28 years. In table 2 present study shows male:female ratio was 1:0.6 ,in table 3 mean duration of symptoms was 2.82 years noted among study participants.

These observations correlate with the study done by *Acharya S et al*[28]. A study done by *Karabulut H et al*[35] noted the mean age 27.7 years and male/female ratio was 1:2.9. A study done by *Mishra S et al*[36] noted the mean age 25 years and male/female ratio was 1:1.3. *HemlataKatiyar VM et al*[43] also found more number of female patient In their study Among the 111 patients studied 45 were males and 66 were females. However the *Yadav SPS* et al[44] shows the incidence of AR to be more in males.

In table 4 present study shows that running nose (94.7%) was the most common symptom noted among study participants followed by nasal block (90.7%) which correlates with studies done by, *Karabulut H et al*[35],*Acharya S et al*[28]

In table 5,6 present study found no statistical significant difference between level of hearing loss and side of ear. These findings matches with *Karabulut H et al*[35].

"Definitive cause for sensorineural hearing loss in allergic rhinitis is not known. However, endolymphatic sac has been hypothesised to be the likely seat of

immunoreactivity in inner ear. The endolymphatic sac has been shown to be capable of both processing antigen and producing its own local antibody response (*Harris JP et al*[48]) "[48]. "The resulting inflammatory mediators and accumulation of toxic metabolic products may interfere with hair cell function leading to sensorineural hearing loss (*Sahni D et al*[29]). "[29]

In table 6 Present study noted that incidence of hearing loss noted in 54.7% cases on right side year and 40% on left side ear. SNHL was the most common type of hearing loss and incidence noted was 42.7% on right side ear & 36% on left side ear noted among study participants.

A study done by *Acharya S et al*[28] stated that "the incidence of SNHL is 40% in their study which is correlate with the present study. "[28]

A study done by *Lasisi AO et al*[49] noted "the incidence of HL in 58% cases and SNHL in 23.3% cases. "[49]

In table 8,9 Present study observe statistical significant association between duration of AR symptoms and type of hearing loss in both the sides of ears. According to that, cases with SNHL noted with longest duration of AR symptoms followed by mixed type of HL. This indicate that severity of HL increased with increase in duration of AR symptoms. These observations are correlate with the study done by Sahni D et al.

In table 7 Present study shows that DPOAE noted absent in 56% cases in right ear,45.3% cases left ear statistically significant in higher number of both the ears of study participants ;In figure 8 present study shows that bilateral abnormal or absent distortion product otoacoustic emissions are note 42.6% of study population. A study

done by *Sahni D et al*[29] noted DPOAE absent in statistically significant in higher number of both the ears of study participants which is correlating with the present study.

"These DPOAE results clearly suggests dysfunctional outer hair cell in patients of allergic rhinitis. It has been proposed that the endolymphatic sac can process antigens and produce its own local antibody response; the resulting inflammatory mediators and toxic products may interfere with hair cell function leading to sensorineural hearing loss. In addition, the sac's fenestrated blood vessels are vulnerable to the effects of vasoactive mediators such as histamine, when released due to allergic reactions elsewhere in the body. Also, higher prevalence of absent otoacoustic emission reflects upon the possibility of higher incidence or higher intensity of hearing loss with greater duration of allergic symptoms. Additional research is required in this area to explore exact relation between duration of allergy and hearing deficit."[29]

Singh S et al[33] also found that, "abnormal DPOAE results in 27(90 per cent) of his study population. "[33]

Dwarkanath et al[30] also showed, "abnormal oae in allergic rhinitis cases than controls. "[30] *Sekhon GS et al*[50] also "showed statistically significant difference in the signal noise ratio (SNR) in most frequencies among patients of allergic rhinitis when compared with controls indicating outer hair cell dysfunction in them."[50]

In one study done by *Nursoy M et*[51], "no statistically significant difference was detected between the study group and control group in terms of their signal noise ratios in all frequencies of DPOAE."

The results showed that the incidence of sensorineural hearing loss was 36% in the left ear and 42.7% in the right ear; DPOAE tests show that 42.6% of study population had bilateral poorer signal-to-noise ratios and diminished or non existent distortion product amplitudes at higher frequencies which means that there is outer hair cells dysfunction. Consequently, sensorineural hearing loss is more susceptible in patients of allergic rhinitis.

CONCLUSION

A cross-sectional study was carried out involving 75 individuals diagnosed with allergic rhinitis, who had symptoms persisting for more than 4 to 6 weeks, at the ENT & HNS department of KLES Dr. Prabhakar Kore Charitable Hospital in Belagavi. The objective was to investigate the occurrence of sensorineural hearing loss in patients with allergic rhinitis, and the findings indicated that the incidence of sensorineural hearing loss was 42.7% in the right ear and 36% in the left ear. The current research indicates that the occurrence of sensorineural hearing loss (SNHL) is more prevalent among individuals with allergic rhinitis.

DPOAE tests demonstrated reduced or absent distortion product amplitudes and a lower signal-to-noise ratio in higher frequencies of bilateral ears in 42.6% of the study population . Therefore, it can be summarized that allergic rhinitis increases the vulnerability to hearing loss, affecting both conductive and sensorineural types.

It is a well known fact that conductive hearing loss is known to occur in allergic rhinitis but our study aimed to find the incidence of sensorineural hearing loss though the exact mechanism is not known .Allergens might affect the inner ear function, particularly outer hair cells can cause sensorineural hearing loss which does not reverse once occurred. Present study indicates that individuals with AR are more prone to hearing loss and severity of hearing loss is increased with duration of symptoms of AR. We would like to recommend a bigger study with large sample to confirm the findings.

Hence, we conclude that there is a higher incidence of sensorineural hearing loss in patients with allergic rhinitis. This should be kept in mind by otorhinolaryngologists while treating Allergic rhinitis. We recommend a routine

screening and hearing evaluation in the patients with symptoms of allergic rhinitis for more than 4-6 months and it should be treated at the earliest, so that adequate measures can be taken to prevent sensorineural hearing loss.

Prevention and control of Allergic rhinitis can go in a long way in controlling the occurrence of Sensorineural hearing loss there by increasing the quality of life.

SUMMARY

Allergic rhinitis is common type-I hypersensitivity reaction where nasal mucosa is affected and mediated by IgE. It manifests through a spectrum of clinical symptoms, including nasal obstruction, watery discharge, sneezing, and itching in the nasal and nasopharyngeal regions. The relationship between allergic rhinitis and otological complications has garnered increasing attention in recent years. Numerous studies suggest that allergic rhinitis leads to otitis media with effusion (OME). While the auditory manifestations of allergic rhinitis are increasingly recognized. There are a few studies indicating the occurrence of SNHL in patients of AR wherein the underlying mechanisms is poorly understood.

Present cross-sectional study conducted among 75 cases of clinically diagnosed allergic rhinitis with symptoms more than 4 to 6 weeks at ENT & HNS department of KLES Dr Prabhakar Kore Charitable hospital, Belagavi with aim to study the incidence of sensorineural hearing loss in patients with allergic rhinitis. Inclusion criteria was patients Aged betweeen 10 to 50 years, symptoms duration more than 4 to 6 weeks, who gave consent to participate in the study. Exclusion criteria was patients with a history of factors causing hearing loss like use of ototoxic agents, metabolic and systemic diseases, noise exposure, history of neurological factors causing hearing loss, ear diseases causing hearing loss like tympanic membrane perforation, cholesteatoma, tumours of middle ear and mastoid, acoustic neuroma.

Present study summarised that

- Highest number of participants belonged to Age group 21 to 30 years (33.3%) followed by 10 to 20 years (30.7%).
- Mean age was 28 years.
- The male: female ratio was 1:0.6
- Mean duration of symptoms was 2.82 years
- Running nose (94.7%) was the most common symptom noted among study participants followed by nasal block (90.7%).
- It is noted that incidence of hearing loss noted in 54.7% cases on right side year and 40% on left side ear.
- SNHL was the most common type of hearing loss noted with incidence of 42.7% in right ear and 36% on left side ear noted among study participants.
- The study observed that there is statistical significance associated between duration of AR symptoms and sensorineural hearing loss, this indicates that severity of SNHL increased with increase in duration of AR symptoms.
- Distorting product otoacoustic emissions (DPOAE) are absent or abnormal in 42% cases of right ear and 34% cases of left ear with reduced or absent distortion product amplitude and a lower signal-to-noise ratio in higher frequency ranges , which shows outer hair cells dysfunction.
- Distorting product otoacoustic emissions (DPOAE) are absent or abnormal in 42.6% cases in bilateral ears with reduced or absent distortion product amplitude and a lower signal-to-noise ratio in higher frequency ranges , which shows outer hair cells dysfunction in both the ears.

This suggests that allergic rhinitis increases the risk of hearing loss, as allergens impact the function of the inner ear, especially affecting the outer hair cells, resulting in irreversible sensorineural hearing loss.

Our findings indicate that individuals with allergic rhinitis have a higher likelihood of developing hearing loss, with the severity increasing as the duration of symptoms prolongs. To confirm these results, we strongly advocate for larger studies with an expanded sample size.

In summary, we suggest there is a greater prevalence of sensorineural hearing loss in patients with allergic rhinitis. Therefore, early diagnosis and treatment of allergic rhinitis are essential. Additionally, in patients experiencing symptoms for more than 4–6 months we recommend hearing assessments to enable timely intervention and reduce the risk of permanent hearing loss.

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ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

Incidence of sensorineural hearing loss in patients with Allergic Rhinitis' – A Case control study in KLES Dr . Prabhakar Kore Hospital, Belagavi"

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

Introduction: The present study is conducted among patients with Allergic rhinitis, controls of Same age and sex in equal number attending outpatient department of ENT and HNS in KLE's Dr Prabhakar Kore Charitable Hospital and Medical Research Centre, Belagavi and they will be investigated for Pure tone audiometry, Distortion product otoacoustic emission (DPOAE), Tympanometry. Your requested to participate in the study and your participation is completely Voluntary.

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

After getting inducted in the study, You will be evaluated for hearing with Pure tone audiometry, Distortion product otoacoustic emission (DPOAE), Tympanometry

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the principal investigator

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "Incidence of sensorineural hearing loss in patients with Allergic Rhinitis' – A Case control study in KLES Dr. Prabhakar Kore Hospital, Belagavi".My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant: Name of the witness:

Signature or left thumb impression of the witness: Name of the investigator:

Signature of the investigator:

ANNEXURE II: PROFORMA

'Incidence of sensorineural hearing loss in patients with Allergic Rhinitis' – A one year cross sectional study in KLES Dr . Prabhakar Kore Hospital, Belagavi

Date:

Name of participant:

Age: OP/IP no:

Sex: Date of assessment:

Address: Date of discharge:

Name of Parent/Guardian:

Relation:

CLINICAL PROFILE:

Chief Complaints:

S.NO.	COMPLAINTS	TICK IF PRESENT	IF PRESENT, DURATION OF SYMPTOMS
1)	NASAL BLOCK		
2)	RECURRENT SNEEZING		
3)	RUNNING NOSE		
4)	ITCHY/WATERING EYES		
5)	SLEEP DISTURBANCE		
6)	IMPAIRMENT OF DAILY ACTIVITIES		

History of Presenting Illness:

Past History:

Personal History:

Family History:

Treatment history:

I) **General Physical Examination** -

Built:

Nourishment:

Vitals:

Temperature:

Pulse rate:

Blood Pressure:

Respiratory Rate:

Pallor/Icterus/Clubbing/Cyanosis/Lymphadenopathy/Edema

ENT Examination

1) NOSE EXAMINATION

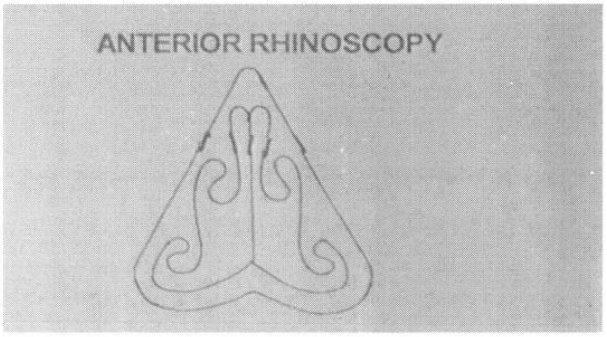
External appearance

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

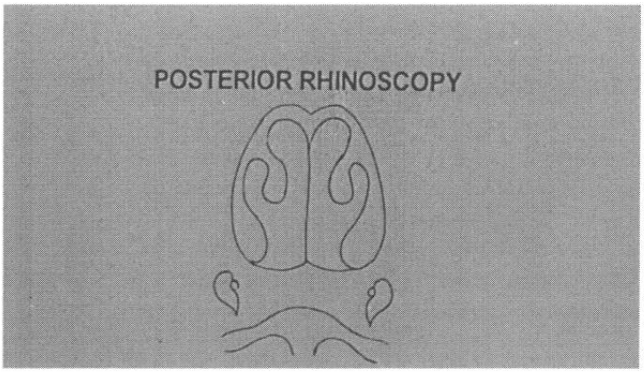
Cold spatula test:

On tip elevation:

Anterior Rhinoscopy:



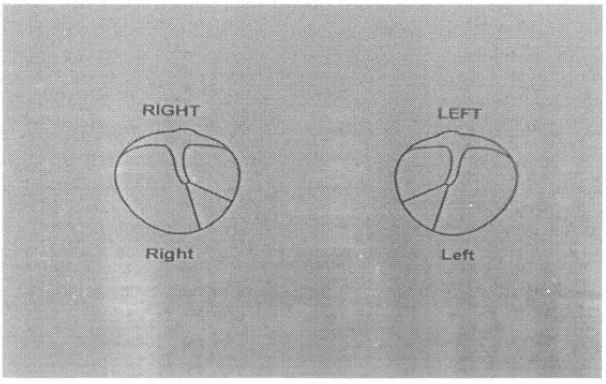
Posterior Rhinoscopy:



Paranasal Sinus Examination:

2) EAR EXAMINATION

	Right	Left
Pinna		
Pre auricular area		
Post auricular area		
Tragal tenderness		
Mastoid tenderness		
External Auditory canal		
Tympanic membrane		



TUNING FORK TESTS:

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

FACIAL NERVE EXAMINATION:

VESTIBULAR FUNCTION:

3) THROAT EXAMINATION:

Oral cavity:

Oropharynx:-

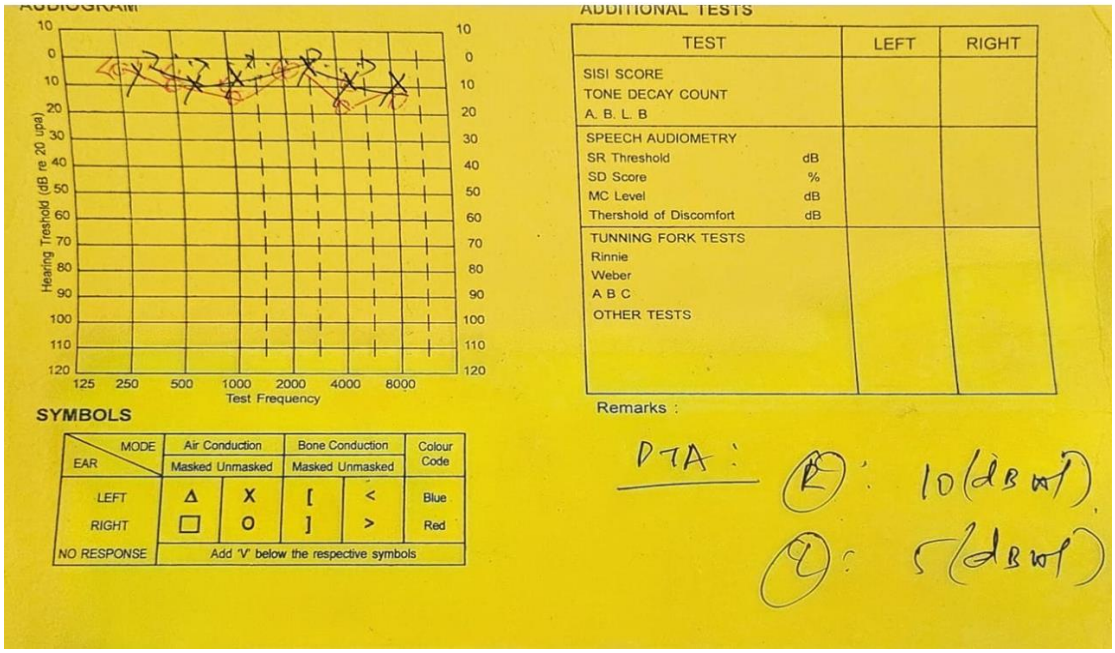
NECK EXAMINATION:

DIAGNOSIS:

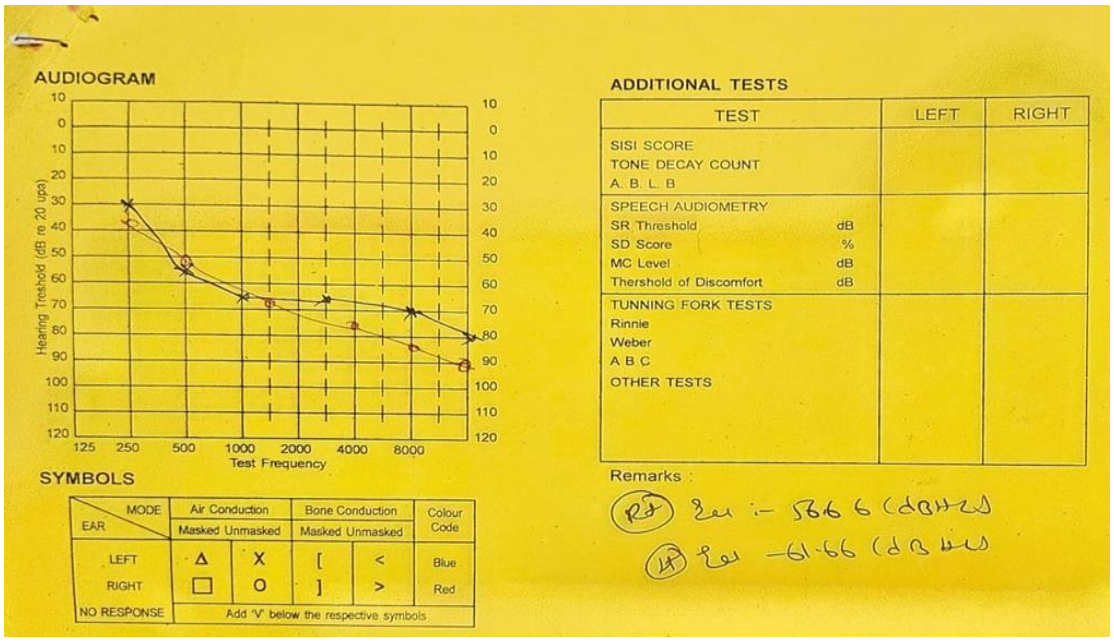
PURE TONE AUDIOMETRY:

DISTORTION PRODUCT OTOACOUSTIC EMISSION(DPOAE):

ANNEXURE III: PHOTOGRAPHS



Photograph 1: Showing normal PTA report



Photograph 2: Showing B/L Moderately severe sensorineural hearing loss

DPOAE: **L1/L2:**55/65 **F2/F1:**1.22

	Right						Left					
Frequencies	**1.5K**	**2K**	**3k**	**4k**	**6k**	**8k**	**1.5k**	**2k**	**3k**	**4k**	**6k**	**8k**
SNR	-6.5	6.3	9.4	10.3	16.8	18.2	8.6	15.6	23.4	25.2	12.5	25.1
DP	5.3	7.1	3.7	0.5	9.2	8.0	19.2	18.4	19.3	17.2	8.9	13.2
Noise	5.8	0.8	-5.7	-9.8	-7.6	-10.2	10.6	2.8	-4.1	-7.5	-3.4	-11.9

Impression:

Right: B/L Indication of Normal OHC's functioning

Left:

Signature

Photograph 3: DPOAE report showing B/L normal outer hair cells function

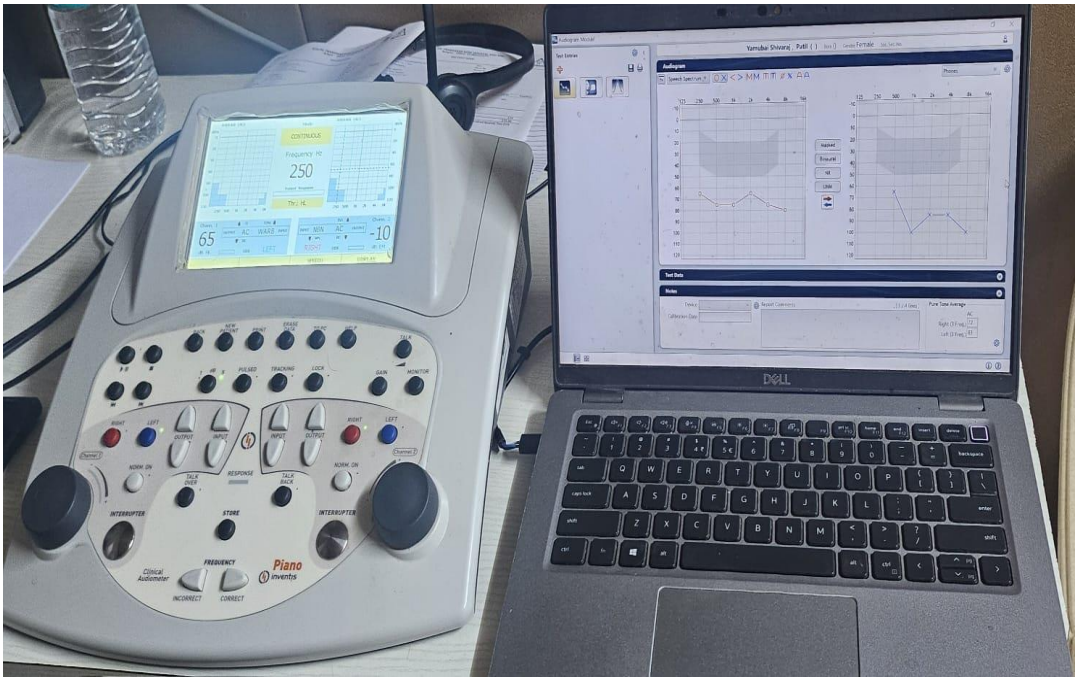
DPOAE: L1/L2: 55/65 F2/F1: 1.22

	Right						Left					
Frequencies	1K	1.5K	2k	3k	4k	6k	1k	1.5k	2k	3k	4k	6k
SNR	-	-	-	-	-	-	-	-	-	-	-	-
DP	-9	-6	-9	-20	-20	-19	-6	-11	4	-2	2	-14
Noise	-10	-8	-7	-20	-20	-20	-9	-10	5	-6	-10	-20

Impression: B/L (9) Indication of OHC's Dysgunction.

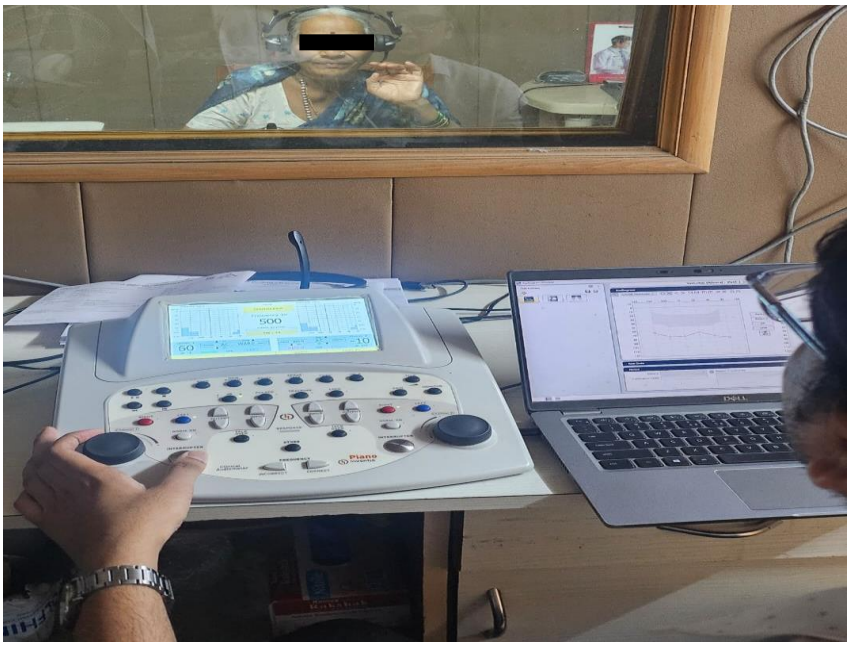
Signature

Photograph 4 : DPOAE report showing B/L outer hair cells Dysfunction

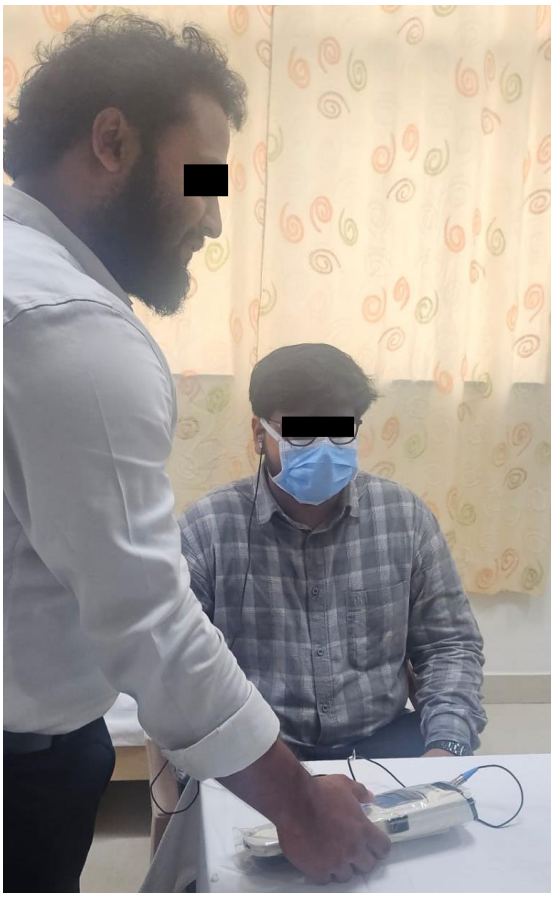


Photograph 5: Showing PTA machine

Photograph 6: showing OAE machine



Photograph 7 : Showing patient undergoing PTA



Photograph 8 : Showing patient undergoing DPOAE

ANNEXURE IV: KEY TO MASTER CHART

AGE	IN YEARS
GENDER	MALE&FEMALE
SYMPTOMS	PRESENT&ABSENT
DURATION OF SYMPTOMS	IN YEARS/MONTHS
PTA	PURE TONE AUDIOMETRY
DPOAE	DISTORTION PRODUCT OTOACOUSTIC EMISSIONS
CROS	Contralateral Routing Of Signal
DPOAE	Distortion Product Otoacoustic Emissions
SNHL	SENSORINEURAL HEARING LOSS
CHL	CONDUCTIVE HEARING LOSS
MHL	MIXED HEARING LOSS
dB	DECIBEL
HL	HEARING LOSS

ANNEXURE V: MASTER CHART

Sr. No.	ip/opdno	Age	Gender	**Nasal block**	**Recurrent Sneezing**	**Running Nose**	**Itchy/watering eyes**	**Sleep disturbance**	**Impairment of daily activities**	**Duration of symptoms**	**PTA (RIGHT)**	**PTA(LEFT)**	**DPOAE (RIGHT)**	**DPOAE(LEFT)**
1	7106211	23	Male	Present	Present	Present	Absent	Present	Present	5 years	55dBhl mhl	15dBhl normal	Absent	present
2	6234784	36	Female	Absent	Absent	Present	Present	Absent	Present	6years	33.3dBhl snhl	28.33 dBhl snhl	Absent	Absent
3	6696574	28	Male	Present	Present	Absent	Absent	Absent	Absent	4years	26.5dBhl snhl	28.7dBhl snhl	Absent	Absent
4	2274932	48	Female	Present	Present	Present	Absent	Absent	Present	3 years	53 dBhl snhl	58.5 dBhl snhl	Absent	Absent
5	6438383	18	Male	Present	Absent	Present	Present	Present	Absent	6months	15dBhl normal	10dBhl normal	Present	Present
6	7166309	34	Male	Absent	Present	Present	Absent	Absent	Present	2years	35.6dBhl snhl	40 dBhl snhl	Absent	Absent
7	7162295	46	Female	Present	Present	Present	Absent	Absent	Absent	6years	55.8 bBhl snhl	45.5dBhl snhl	Absent	Absent
8	6220213	21	Male	Present	Absent	Present	Present	Absent	Present	5months	12dBhl normal	10dBhl normal	Present	Present
9	7178808	16	Male	Present	Present	Present	Absent	Absent	Absent	1 year	28.5 dbhl chl	15 Dbhl normal	Absent	Present
10	7181575	38	Female	Present	Absent	Present	Present	Absent	Present	2year	20dbHL normal	22dbhl normal	present	present
11	6693510	26	Female	Absent	Present	Present	Absent	Present	Present	9months	18.5dBhl normal	15 Dbhl normal	Present	Absent
12	7186397	44	Male	Present	Present	Present	Present	Absent	Absent	5 years	55dBhl snhl	35.8 Dbhl snhl	Absent	Absent
13	7130958	16	Male	Present	Present	Present	Absent	Absent	Present	2years	15dBhl normal	12dbhl normal	Present	Present
14	7183183	49	Male	Present	Absent	Present	Absent	Absent	Absent	3 years	35dbhl snhl	28.3 dbhl snhl	Absent	Absent
15	5767823	31	Female	Present	Present	Present	Absent	Present	Absent	2years	28.5 dbhlsnhl	29dbhl snhl	Absent	Absent

16	72371211	24	Male	Present	Absent	Present	Present	Absent	Present	9months	27.5dbhl snhl	15dbhl normal	Absent	Absent
17	7247053	11	Female	Present	Present	Present	Absent	Absent	Absent	3months	15dbhl normal	10dbhl normal	Present	Present
18	6248657	47	Male	Present	Absent	Present	Present	Present	Present	10 years	48.3dbhl snhl	53.3dbhl snhl	Absent	Absent
19	7250366	39	Male	Present	Present	Present	Absent	Absent	Present	2years	32.3dbhl snhl	23dbhl normal	Absent	present
20	7250232	28	Female	Present	Absent	Present	Absent	Absent	Absent	6months	15dBhl normal	15dBhl normal	Absent	Absent
21	7250397	14	Male	Absent	Present	Present	Absent	Absent	Present	8months	15dBhl normal	15dBhl normal	Present	Present
22	7250236	35	Female	Present	Absent	Present	Absent	Present	Absent	2years	25.3dbhl snhl	32.5dbhl snhl	Absent	Absent
23	7224390	44	Male	Present	Present	Present	Present	Absent	Present	8years	45.5dBhl snhl	55.dbhl snhl	Absent	Absent
24	7274737	23	Male	Present	Present	Present	Absent	Absent	Absent	7months	45dbhl chl	35.5dbhl chl	Absent	Absent
25	7221730	40	Female	Absent	Absent	Present	Present	Present	Absent	4years	10dBHL normal	15dBhl normal	present	present
26	7257168	12	Male	Present	Present	Present	Absent	Absent	Present	5 years	10dBHL normal	15dBhl normal	Present	Present
27	7361780	42	Male	Present	Absent	Present	Absent	Absent	Present	3months	11dbhl normal	12bdhl normal	present	present
28	11006950	29	Female	Present	Absent	Present	Absent	Present	Absent	6months	10dBHL normal	10bdhl normal	present	present
29	118656	11	Female	Present	Present	Present	Present	Present	Absent	5months	15dBhl normal	15dBhl normal	Present	Present
30	1189430	24	Female	Present	Absent	Present	Present	Absent	Absent	6months	15dBhl normal	10dBhl normal	presnet	presnet
31	1189810	38	Male	Absent	Present	Present	Absent	Absent	Present	8years	55.5 dbhl snhl	42.3dbhl snhl	Absent	Absent
32	1193637	15	Male	Present	Present	Present	Absent	Present	Absent	1year	20dbHL normal	23.5 dbhl normal	Present	Present
33	1200192	28	Female	Present	Present	Absent	Present	Absent	Present	3months	15dBhl normal	10dBhl normal	Present	Present
34	1199101	15	Male	Present	Absent	Present	Absent	Absent	Absent	6months	15 dbhl normal	10dBhl normal	Present	Present
35	1199466	48	Female	Present	Present	Present	Present	Absent	Present	8years	28.3 dbhl snhl	33dbhlsnhl	Absent	Absent
36	1199551	25	Male	Present	Absent	Present	Absent	Absent	Absent	3years	25.3 dbhl snhl	15dbhlsnhl	Absent	Absent
37	1201585	18	Female	Present	Present	Present	Absent	Present	Present	3months	10dBHL normal	10 Dbhl normal	Present	Present
38	7282508	33	Male	Present	Present	Present	Present	Absent	Absent	2years	32DBHL SNHL	26.3dbhl snhl	Absent	Absent
39	1207791	29	Male	Present	Present	Present	Absent	Present	Present	5 years	45.5dBhl snhl	29.5 dbhl snhl	Absent	Absent
40	1198761	41	Male	Present	Present	Present	Absent	Absent	Absent	1year	25.3dbhl snhl	28.5 dbhl snhl	Absent	Absent

41	6802921	13	Female	Present	Present	Present	Absent	Absent	Absent	2months	10dBHL normal	10dBhl normal	Present	Present
42	1223444	21	Male	Present	Absent	Present	Present	Absent	Absent	5 years	49.5 mixed	28.3 snhl	Absent	Absent
43	3569326	30	Male	Present	Present	Present	Absent	Present	Absent	3 years	23.5 dbhl normal	32.5 dbhl snhl	present	Absent
44	1172528	25	Female	Present	Absent	Present	Absent	Absent	Present	2months	10 dbhl normal	15 Dbhl normal	Present	present
45	1166605	50	Male	Present	Present	Present	Absent	Absent	Absent	9years	35.8 dbhl snhl	70dbhl snhl	Absent	Absent
46	1166599	20	Male	Absent	Present	Present	Present	Absent	Absent	3months	15dBhl normal	10dBhl normal	Present	Present
47	1157179	36	Female	Present	Absent	Present	Absent	Absent	Present	9months	15 dbhl normal	10dBhl normal	present	present
48	1152489	26	Male	Present	Present	Present	Present	Present	Absent	2years	25.5dbhl snhl	25.5dbhl snl	Absent	Absent
49	1151767	24	Female	Present	Absent	Present	Present	Absent	Absent	8months	25dBhl chl	10 Dbhl normal	Absent	present
50	1165535	17	Male	Present	Absent	Present	Absent	Absent	Absent	5months	10dBHL normal	15dBhl normal	Present	Present
51	1161143	48	Female	Present	Present	Absent	Present	Present	Absent	15 years	65dBhl snhl	55dBhl snhl	Absent	Absent
52	1162195	28	Female	Present	Present	Present	Absent	Absent	Absent	2years	26.5 Dbhl snhl	15 Dbhl normal	Absent	present
53	1162077	18	Male	Present	Absent	Present	Present	Present	Present	4 years	32 dBhl mixed	35 dbhl mixed	Absent	Absent
54	1159857	27	Female	Present	Absent	Present	Absent	Absent	Absent	2years	36.5dbhl snhl	20.3dBHL Normal	Absent	present
55	1164212	18	Male	Present	Present	Present	Absent	Absent	Present	2months	15dBhl normal	10dBhl normal	Present	Present
56	10026408	42	Female	Present	Present	Present	Present	Absent	Absent	10 years	56dBhl snhl	45.5dBhl snhl	Absent	Absent
57	7169756	23	Male	Present	Present	Present	Absent	Present	Absent	3months	35.5dBhl chl	10dBhl normal	Absent	present
58	7821636	15	Male	Present	Present	Present	Absent	Absent	Present	3months	10dBHL normal	10dBhl normal	Present	Present
59	12062051	20	Male	Present	Absent	Present	Present	Absent	Absent	7years	48dBhl chl	45.5dBhl chl	Absent	Absent
60	4868010	34	Male	Present	Present	Present	Absent	Present	Absent	2years	32dBhl snhl	25dBhl normal	Absent	present
61	7302671	12	Male	Present	Present	Present	Present	Absent	Present	7months	15dBhl normal	10dBhl normal	Present	Present
62	7243687	25	Male	Present	Absent	Present	Absent	Present	Present	2years	25.2dBhl snhl	24.8dBhl normal	Absent	Absent
63	7391680	16	Female	Present	Present	Present	Present	Absent	Present	5 years	45dbhl chl	12 dBhl normal	Absent	present
64	1162067	46	Male	Present	Absent	Present	Absent	Absent	Absent	5years	35 dBhl snhl	28.5dBhl snhl	Absent	Absent
65	1159757	12	Male	Present	Present	Present	Absent	Present	Absent	8months	10dBHL normal	10dBhl normal	Present	Present

66	1164212	21	Male	Present	Present	Absent	Present	Absent	Present	2months	18dBhl normal	15.5 dBhl normal	Present	Present
67	1026308	38	Male	Present	Absent	Present	Absent	Absent	Present	10years	45.5dBhl snhl	33.3dBhl snhl	Absent	Absent
68	7169746	14	Male	Present	Present	Present	Present	Absent	Present	6months	10dBHL normal	10dBhl normal	Present	Present
69	7721636	28	Female	Present	Present	Present	Absent	Absent	Present	2years	25.5 dBhl snhl	28.dBhl snhl	Absent	Absent
70	12062041	41	Male	Present	Present	Present	Present	Present	Absent	4years	57.8dBhl snhl	55.5 dBhl snhl	Absent	Absent
71	4768010	18	Male	Present	Absent	Present	Present	Absent	Absent	1month	12dBhl normal	15dBhl normal	Present	Present
72	73025571	18	Male	Present	Absent	Present	Absent	Absent	Present	2years	15dBhl normal	15dBhl normal	present	present
73	7243587	28	Female	Present	Absent	Present	Absent	Present	Absent	1year	18dBhl normal	10dBhl normal	present	present
74	7395680	32	Male	Present	Present	Present	Absent	Absent	Absent	5 years	28dbhl snhl	15 dbhl normal	Absent	present
75	6437383	25	Male	Present	Present	Present	Absent	Present	Absent	1year	10dBHLnormal	10dBhl normal	present	Present