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A RETROSPECTIVE STUDY OF INCIDENCE, ETIOLOGY AND MANAGEMENT OF SENSORINEURAL HEARING LOSS IN ADULTS

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ABSTRACT

Hearing impairment is the most frequent sensory deficit in human population, affecting more than 250 million people in the world. Consequences of hearing impairment include inability to interpret speech sounds, often producing a reduced ability to communicate, delay in language acquisition, economic and educational disadvantage, social isolation and stigmata. In this study, 100 adult patients who came with hearing impairment and diagnosed with sensorineural hearing loss were reviewed retrospectively to study the various etiological factors and effective management strategies for sensorineural hearing loss (SNHL). The study was conducted at Dr. B.R. Ambedkar Medical College & Hospital over a period of 2 years (October 2012 to September 2014) in patients with hearing impairment. It was noted that males were predominantly involved and noise- induced hearing loss turned out to be the most common cause of SNHL in this study. Since the hearing impairment either worsened or remained the same, it was concluded that once the cochlea is damaged secondary to any etiological agent, the damage is irreversible.

KEYWORDS Sensorineural hearing loss (SNHL), Noise-induced hearing loss (NIHL)

INTRODUCTION:

From the developmental point of view, it is clear that hearing is a vital sense for human beings as the fetus's cochlea is developed at about mid-pregnancy and there is evidence that human babies can hear sound inside the uterus[1]. Hearing plays an essential role in the development of speech and spoken language. It also plays a role in cognitive aspects like memory and learning, warning signals and protective reflexes, interpretation of space and postural balance among other things[2]. The adverse effect of hearing loss on an individual's well being and quality of life can lead to depression, anxiety, loneliness, social isolation and dementia[3].

In humans, the inability to hear sound frequencies in the normal range of hearing (20- 20,000 Hz) is called hearing impairment[4]. The World Health Organization (WHO) defines disabling hearing impairment in adults as a permanent unaided hearing threshold level for better ear as 41 dB or greater in adults and 31 dB or greater in children less than 15 years of age. WHO classifies hearing impairment into five grades:

Table 1: WHO grades of hearing impairment

Grades of impairment	Audiometric ISO value (Average of 500, 1000, 2000, 4000 Hz) in the better ear	Impairment description
0 (no impairment)	25 dBHL or less	No or very slight hearing problems. Able to hear whispers
1 (slight impairment)	26-40 dBHL	Able to hear and repeat words using normal voice at 1 metre
2 (moderate impairment)	41-60 dBHL	Able to hear and repeat words using raised voice at 1 metre
3 (severe impairment)	61-80 dBHL	Able to hear some words when shouted into better ear
4 (profound impa irment including deafness)	81 dBHL or more	Unable to hear and understand even a shouted voice`

Sensorineural Hearing Loss (SNHL) occurs when there is damage to the inner ear (cochlea), or to the nerve pathways from the inner ear to the brain. SNHL is the most common type of hearing loss and cannot be medically treated so far. Those who are affected have difficulties in hearing faint sounds even when the speech is loud enough. The etiology of SNHL in adults can be either cochlear or retrocochlear Cochlear causes can be further divided into genetic or non-genetic:

A. Genetic Causes [5-13]: Familial progressive SNHL, Cochlear otosclerosis, Presbyacusis. Syndromes like Alport's, Refsum's, Hurler's, Klippel Feil, Paget's disease, Von Reckling Hasusen's disease, Crouzon's disease
B. Non-genetic causes: Infection, ototoxicity[14],NIHL, trauma, metabolic disorders, vascular insufficiency, degeneration

Retrocochlear causes include congenital (Usher's and Pendred's s syndrome), infections (meningitis, encephalomyelitis, brain abscess), tumors (acoustic neuroma, meningioma, glioma), vascular disorders (hypertension, vertebrobasilar insufficiency), trauma, metabolic and drug induced, demyelinating and degenerative disorders (multiple sclerosis, subacute combined degeneration, Friedrich's ataxia, syringomyelia) and psychogenic disorders.

AIMS AND OBJECTIVES:

1. Assessment of incidence of SNHL in patients with hearing impairment
2. Study of various etiological factors of SNHL
3. Effective management strategies of SNHL

MATERIALS AND METHODS:

The study was carried out in the Department of ENT of Dr. B.R. Ambedkar Medical College, Bangalore. Over a 2-year period, a total of 100 patients were admitted with complaints of decreased hearing and were diagnosed with pure SNHL after clinical and audiometric evaluation.

Inclusion criteria:

1. All patients with hearing impairment diagnosed with pure SNHL
2. Aged 20-80 years
3. Both males and females

Exclusion criteria:

1. Age below 20 years
2. Conductive hearing loss
3. Mixed hearing loss
4. Congenital hearing loss

At first, a detailed history was taken and diagnosis was made on the basis on the clinical (tuning fork tests), laboratory (complete

blood count, FBS&PPBS, renal function tests, VDRL, HIV, lipid profile), audiometric (pure tone audiometry, speech audiometry, brainstem evoked response audiometry) and radiological (X-ray mastoid, CT Brain including mastoid) investigations.

After confirmation of diagnosis and treating the patient accordingly, the patients were followed up for recording the benefit of hearing aid and PTA was repeated on all patients once in 3 months for first two visits and later once in 6 months for 2 years.

RESULTS AND OBSERVATION:

Etiology: The commonest cause was found to be noise induced hearing loss accounting for 32% followed by presbyacusis (24%), Infection (14%), ototoxicity (12%), hypertension (6%), Meniere's disease (6%), metabolic disorders (4%) and trauma (2%).

Table 2: Percentage of causes of SNHL in adults

Causes	No. of cases	Percentage (%)
NIHL	32	32
Presbyacusis	24	24
Infections	14	14
Ototoxicity	12	12
Hypertension	6	6
Meniere's Disease	6	6
Metabolic disorder	4	4
Trauma	2	2

Age: NIHL occurred mainly in the age group of 40-60 years. SNHL due to infection was common in between 20-29 years as infections (typhoid, mumps, meningitis) mainly affect the younger age group. Metabolic disorders were usually seen in the age group of 40-60 years. Presbyacusis being a disease of old age was seen in patients above 50 years of age and so was hypertension.

Table 3: Age distribution according to causes of SNHL

Causes	20-29 years	30-39 years	40-49 years	50-59 years	> 59 years
NIHL	2	9	12	7	2
Presbyacusis	0	0	0	14	10
Infections	8	4	1	1	0
Ototoxicity	3	2	2	2	3
Hypertension	0	0	2	2	2
Meniere's Disease	0	3	2	1	0
Metabolic Disorder	0	0	1	2	1
Trauma	1	0	1	0	0

Sex: SNHL was seen in 68 males and 32 females giving preponderance in males with a ratio of 2:1.

Table 4: Sex distribution of SNHL

Sex	No. of cases	Percentage (%)
Males	68	68
Females	32	32

Table 5: Sex distribution according to causes of SNHL

Causes	Males	Females
NIHL	22	10
Presbyacusis	14	10
Infections	10	4
Ototoxicity	8	4
Hypertension	4	2
Meniere's Disease	5	1
Metabolic Disorder	3	1
Trauma	2	0

Pure Tone Audiometry: PTA revealed mild loss in 16%, moderate in 6%, moderately severe in 30%, severe in 32% and profound loss in 16% of cases of various etiologies.

Table 6: Audiometric findings according to causes of SNHL (M- Mild, MD- Moderate, MS- Moderately Severe, S- Severe, P- Profound)

Causes	Pure Tone Audiometry					Total SD Score			BERA	
	M	MD	MS	S	P	GOOD	MOD	LOW	IND	PEAK
NIHL	6	0	8	16	2	16	12	4	0	0
Presbyacusis	2	0	6	6	10	12	4	8	0	0
Infections	2	2	4	4	2	7	4	3	0	1
Ototoxicity	0	0	8	4	0	6	4	2	0	0
Hypertension	0	0	4	2	0	3	2	1	0	0
Meniere's Disease	4	2	0	0	0	3	2	1	0	0
Metabolic Disorder	2	2	0	0	0	2	1	1	0	0
Trauma	0	0	0	0	2	2	0	0	1	1
Total	16	6	30	32	16	51	29	20		

DISCUSSION:

The commonest cause was found to be noise induced hearing loss accounting for 32%. The study also showed that NIHL occurred mainly in the age group of 40-60 years. This is because the patients in this age group were working in compression chambers in NAL, stone quarries, power looms, etc. for the past 10-15 years. They are exposed continuously to a noise level of 95 dB for 8-10 hours daily. According to earlier experiments on NIHL, an exposure 95 dB for 8-10 hours period for 10-15 years will induce permanent threshold shift. Most patients are males as they are the breadwinners of the family. In the post-typhoid SNHL, it was observed that the patients had leukopenia of 4000-5000 cells/ mm^3 during typhoid fever and hearing loss started during the third week of typhoid fever.

Of the presenting symptoms, all the patients presented with decreased hearing in both ears except those with traumatic SNHL and 1 case of post mumps SNHL. The hearing loss was insidious in onset and progressed gradually in all cases.. About 34% of the patients had tinnitus. In Meniere's disease, all the 6 patients had low pitched tinnitus. Vertigo was present in 6 patients Meniere's disease and 2 patients with trauma, accounting for 8% of the cases. In Meniere's, it was the rotatory movement of the surroundings while in SNHL it was unsteadiness. In NIHL, there were complaints of sound irritability, inability to have telephonic conversations and headache.

Pure tone audiometry revealed mild loss in 16%, moderate loss in in 6%, moderately severe loss in 30%, severe loss in 32% and profound loss in 16% cases of various etiologies. Speech discrimination score was high in 60%, moderate in 36% and a low score was seen in 4%. BERA revealed indistinct peaks bilaterally in the patient of post-meningitis SNHL and unilaterally in traumatic SNHL.

The patients were treated with Inj. Optineuron/ Neurobion (B_1- 33mg, B_6- 33mg, B_{12}- 33.3mcg/ml), 3ml on alternate days. A total of 10 injections were given. Tab. Neurobion/ Sclerobion (B_1- 10mg, B_6- 50mg, B_{12}- 50mcg) once daily for 30-60 days. Most patients were also empirically put on steroids (prednisolone) and vasodilators (depending on the merits of the case). Patients who had tinnitus were given Tab. Cinnarizine 25mg thrice daily for 10-15 days or more if necessary. In Meniere's disease, in addition to the above there was restriction to salt and water intake. Diuretics (Tab. Lasix 40mgOD for 10 days), Tab Dimenhydrinate 50mg TID for 15 days along with Inj. Stemetil SOS were also given.

Patients suffering from NIHL were advised to use ear protection, work is shifts and use hearing aids. Fifty percent of the patients were followed up for two years, once in every three months. Tinnitus subsided in all patients except one case of intractable Meniere's disease. Patients put on hearing aids did well. Repeat PTA revealed improvement in hearing by 5-15 dB in one case of post- typhoid SNHL. Worsening of hearing by 10-15 dB were seen in 16 patients. The remaining patients had the same levels of hearing.

CONCLUSION:

- In this study of SNHL in adults, it was noted that it predominantly involves males, the male to female sex ratio was approximately 2:1
- NIHL was the most common cause of SNHL in this study. The least incidence was found in traumatic cases
- SNHL secondary to infections (viral fever) was commonly seen in lower age groups (3rd and 4th decades)
- SNHL secondary to metabolic disorders and presbyacusis was commonly seen in higher age groups (5th, 6th and 7th decades)
- In the various above mentioned etiologies, cochlear symptoms (deafness and tinnitus) predominated over the vestibular symptoms (vertigo) which indicates that they are more cochleotoxic rather than vestibulotoxic
- With ototoxicity, SNHL can occur either during treatment or after the cessation of treatment
- In infections (viral fever and meningitis), both cochlear and retrocochlear loss can occur. It is also seen in Diabetes Mellitus
- In presbyacusis, cochlear deafness is seen
- An exposure to noise levels of 90-95 dB continuously for 8-10 hours a day for a period of 15 years will induce a permanent threshold shift
- It was observed that SNHL due to the etiologies outlined above either worsens or remains the same, but no improvement is seen
- Finally it can be concluded that once the cochlea is damaged secondary to any of the above etiologies, the damage is irreversible

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