Original Article

Assessment Of Pure Tone Hearing Threshold In Niddm & Its Comparison With Healthy Individual

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Abstract

Aim: To assess whether patients of NIDDM have a higher incidence of sensorineural hearing loss.

Methodology: A cross-sectional study was conducted included 100 participants such that 50 were diabetics and another were nondiabetics. Pure Tone Audiometry was performed on the patient, and the results were assessed by comparing them to the patient's diabetes duration. The IBM SPSS version 20 software was used for all data analysis. The tables were created using cross tabulation and frequency distribution.

Results: On comparing the degree of hearing loss between both the groups and the mean hearing threshold of both the ears with duration of diabetes it was found that distribution of degree of hearing loss between the cases and control was significantly different.

Conclusion: The study concluded that age had the direct association with the pure tone threshold level which means by increasing the age of the diabetes patients, hearing loss also increases as revealed by the significant p value of <0. 005.

INTRODUCTION:

The first quantitative hearing test used to evaluate the type and severity of hearing loss in adults and children is typically pure tone audiometry. Other tests for assessing middle ear functions include the immittance test and speech audiometry. The degree, kind, laterality, and frequency configuration of hearing loss are assessed using pure tone air conduction and bone conduction tests. Hearing thresholds (dB) are shown using pure tone audiometry for various frequencies (Hz). A pure tone audiology threshold is the decibel level at which a sound is perceived 50% of the time at a given frequency. Decibel scale is mainly used to record decibel hearing level in pure tone. The intensity of this scale is basically based on normal human being with 0 dB HL means for median threshold for adult.

Worldwide, diabetes mellitus type II is a common metabolic condition. Younger age groups are also affected by it, in addition to older age groups. However, each person may likely experience diabetes differently. Diabetes is also known as Diabetes Mellitus. It is a type of set of metabolic illness indicates by persistent high BP and sugar levels. It is frequently linked to low insulin synthesis or a lack of cell responsiveness to the released insulin. They exhibit a variety of symptoms and have an impact on proper metabolism. Weight loss, increased urination, increased thirst, and polydipsia are the common signs of diabetes (increased hunger). Additional signs of diabetes include weariness, exhaustion, blurred vision, headaches, sluggish wound and cut healing, and itchy skin in rare cases.

Zwislocki (2002) made the initial claim that diabetes and hearing loss are related in 1857. Following this, other research was conducted to look into the connections between DM and hearing loss. Diabetes causes changes in the glucose concentration in the inner ear, which can affect hearing. Changes in glucose concentration caused by changes in systemic insulin concentration can change the endolymphatic potential.

Regarding the relationship between hearing loss and diabetic symptoms, there are many inconsistencies in the data and studies currently available. The hearing loss typically characterised has a bilateral, progressive, and gradual start. Higher frequencies and elderly people are primarily affected by the sensorineural form of deafness. Although there is a decline in auditory acuity similar to presbycusis, people who are affected have a hearing loss that is more than what is typical for their age (Hoth, 2009).

Histological studies of the temporal bones revealed altered tiny blood vessels in the stria vascularis and modiolus. The inner ear is expected to have the most dramatic vascular changes in diabetes. The constriction of the blood vessels in the inner ear may be caused by arteriolosclerosis or intimal hypertrophy (Rodríguez, 2016).
This study examines the relationship between diabetes and hearing damage because it has been shown in numerous studies that persons with long-term diabetes have hearing issues.

AIM

To assess whether patients of NIDDM have a higher incidence of sensorineural hearing loss.

OBJECTIVES

1. Assess the influence of duration of NIDDM on hearing threshold.
2. To compare Hearing threshold in NIDDM with healthy individual in different age group.

1.1 Diabetes-Related Changes In Hearing

A metabolic disorder called diabetes mellitus (DM) causes vascular and neurological issues in persons who have it. We have demonstrated that, particularly among younger Veterans, those with diabetes have a worse time understanding speech than those of a comparable age without diabetes. According to statistics, people with diabetes experience higher hearing loss than people without the condition. A recent study by Bainbridge et al. (2004) reported that among 5,742 individuals in the National Health and Nutrition Examination survey, those with diabetes had more hearing loss than those without, which is the greatest evidence for these claims. The prevalence of mild to severe high-frequency impairment in the better ear varied most between participants in the 20 to 49 age group.

The prevalence of sensor neural hearing loss was higher in patients with DM and appeared to be correlated with the degree of elevated serum creatinine, according to a study that compared the electronic medical records of over 12,000 DM patients and over 53,000 age-matched controls from a Veterans Affairs database (Kakarlapudi et al. 2003). Additionally, there have been theories linking hearing loss to the severity of diabetes or the level of serum glucose. Only a few studies have looked at the possible relationship between cochlear dysfunction and the severity of diabetes, as determined by the type of medication patients need to manage their condition (insulin vs. oral hypoglycaemic agents).

Numerous research have tried to pinpoint the cause of hearing loss in people with DM, but as of right now, neither the site of the lesions nor the mechanism of impairment is known. The aetiology of this sensorineural hearing loss associated with DM has been theorised to include cochlear microangiopathy, hyperglycaemia of the cerebrospinal fluid or perilymph, auditory neuropathy, and diabetes encephalopathy. According to Fukushima et al. (2006), DM-related pathological alterations in the cochlea include thicker stria vascularis vessels, stria vascularis atrophy, and loss of outer hair cells, although there was no difference in spiral ganglion cell loss from controls. In contrast to those with less severe non-insulin-dependent diabetes, those with insulin-dependent diabetes (IDDM) showed more profound cochlear changes (NIDDM). People with diabetes have also seen modifications in their core auditory and cognitive processing (Lisowska et al. 2001). Most investigations on diabetes and hearing have used comparatively few subjects. Some earlier investigations focused mostly on older participants, whereas trials with relatively young participants virtually entirely included type I DM patients. Large-scale studies with DM patients of all ages did not include any test of proper auditory and determining pure tone for typical medical frequencies of audiogram.

1.2 Association Of NIDDM And Hearing Loss

Age-related hearing loss and NIDDM both significantly impair the health and quality of life of older people. Numerous microvascular problems, most frequently affecting diabetics' eyes and kidneys, are linked to NIDDM. It has been suggested that diabetics' ears and hearing may also be impacted by microvascular problems. Studies on diabetic mice have shown thickening of the stria vascularis capillaries' basement membrane. According to histopathological investigations, people with diabetes have damaged neurons and blood vessels in their inner ears. It has been hypothesised that these vascular lesions have a significant role in the neuronal degeneration of the auditory system.

Numerous clinical research have looked into the potential link between diabetes and hearing loss. The majority of these earlier investigations were quite small, involving 20–50 participants, which would have made it more difficult to find any associations. Only one of these studies specifically evaluated NIDDM, and many others did not make a distinction between IDDM and NIDDM. Studies using a comparison group may lack information regarding the comparison group's selection, have medical professionals, or have groups with high noise exposure. The contradictory results in the literature have been a result of these restrictions. Nonetheless, in case of these investigation, loss of hearing was generally discovered to be connected with nephropathy and critical retinopathy.

The larger trials, with 99–200 participants, were unable to identify an overall association between diabetes and hearing loss.
The relationship between diabetes and hearing loss has only been examined in one population-based investigation, the Framingham Heart Study. Pure tone averages (PTAs) were determined for the frequencies of 0.25, 0.5, and 1 kHz (PTA

lo) and 4, 6, and 8 kHz using audiometric data from the biennial evaluation 18. (PTA hi). There was no association between low glucose tolerance, diabetes, or both, and hearing thresholds. But in women, PTA lo was associated with blood sugar, meaning that as blood sugar levels increased, hearing thresholds did as well (or got worse).

1.3 Management Of Type 2 Diabetes (Niddm)

Over the past ten years, type 2 diabetes in particular has become a significant clinical and health issue. As a result of their medical care with better patient education, the availability of expertise, persistent follow-up, and screening for diabetes complications, people with diabetes are anticipated to have a number of benefits. After the start of the historic UKPDS experiment, a number of new pharmacological classes were available, allowing for increased treatment flexibility and advancing the attainment of glycaemic control.

In underdeveloped and recently industrialised countries, the epidemic is real. There will be a 35 percent rise in the prevalence of diabetes globally between 1995 and 2025. India will experience the second-highest increase in prevalence, at 59%. India will experience the most increase—a 195 percent rise, or 19–57 million people (King, 1998). India will have the highest proportion of diabetes at both points. What's more, by 2025, India will be home to one fifth of all diabetics worldwide, up from its current position of housing one tenth of all diabetics worldwide. Furthermore, diabetes prevalence in India is probably going to rise over time, making the estimations even worse.

The foundation upon which CHD and CAD build is hyperglycemia. Patients with diabetes have a higher chance of developing CAD, having cardiovascular problems, and dying from those problems. The risk of having CHD was approximately four times higher in diabetic females and 212 times higher in diabetic males (Folsom et al. 1997). This is the scene of the devastation that will follow the diabetes epidemic and the CHD epidemic. With a value of 4.9 for men and 3.2 for women, the age group of 25 to 44 years had the highest ratio of mortality rates for diabetics compared to nondiabetics. At the age range of 65 to 74 years, the ratio decreased with ageing but remained approximately 112 times higher (Gu, 1998).

Morbidity associated with the duration and glycaemic management of diabetes is increased by microvascular consequences. About 80% of diabetics with poor glycaemic control have any sort of retinopathy within 20 years, compared to 20% of those with excellent glycaemic control. Proliferative retinopathy is present in 40% and 5% of people, respectively. After diabetes has been established for 15-20 years, there is a 10-15% chance of developing clinically significant retinal edoema (Ohkuba, 1995). About 80% of diabetics with good glycaemic control continue to be free of microalbuminuria. The evolution of diabetic nephropathy over time in a patient with poor glycaemic control, however, is determined by genetic predisposition (Malmberg et al. 1996).

The main objective is to enhance the quality and quantity of life while also preventing, delaying, arresting, or reversing diabetic consequences. There is no question that type-2 diabetics can avoid, halt, or delay different consequences of diabetes, as demonstrated by the Kumamoto trial (Saini, 199), UKPDS study, and DIGAMI study (Garg, 1998). Additionally, life quality and quantity can be increased. Despite having a limited patient population, Kumamoto's trial was just as successful in treating IDDM patients as DCCT was. It has been demonstrated that numerous insulin injections used in conjunction with increased glycaemic control can postpone the development and progression of diabetic microvascular problems. Patients assigned to intensive treatment in the UKPDS Study had a 25% lower probability of microvascular end goals than those assigned to standard treatment. The removal of cataracts and retinal photocoagulation both significantly decreased, but the vitreous haemorrhage remained unaffected. At the end of nine years, there were considerably fewer patients in the intense group who had microalbuminuria, proteinuria, and impaired biothesiometer perception. In the UKPDS, there was a 16 percent reduction in acute MI risk, however it did not achieve statistical significance. This may show that either the glycaemic management for macrovascular disease was probably not tight enough or that the glycaemic threshold for progression is unknown (Bailey, 1996).

1.3.1 Stages In Management

Three stages in the management of diabetes with suitable opportunities and strategies can be characterised in light of the natural history of type II diabetes. These could be referred to as primary care, specialty care, or public health care. In order to prevent or delay the onset of diabetes, public health care entails raising knowledge about the disease, establishing screening programmes for an early diagnosis, and changing societal lifestyles. Primary medical care does not maintain adequate glycaemic control or the intervention needed for problems associated to diabetes up until standard treatment methods. To reduce morbidity and mortality, specialised care offers comprehensive treatment for problems and related risk factors.

1.3.2 Screening Of Diabetes [9]

Screening should be taken into consideration in high-risk populations, such as age >45, family history of diabetes (parent or sibling), obesity, hypertension, dyslipidemia, or prior history of impaired glucose tolerance (IGT) or gestational diabetes mellitus, in order to increase the cost-effectiveness of testing undiagnosed diabetics (GDM). Fasting plasma glucose (FPG) is the most popular screening test in order In order to be considered fasting, you must go at least eight hours without

consuming anything but water. It should be emphasised that capillary blood glucose testing using a glucometer is not advised here for screening. Three-yearly intervals should be thought about for screening high-risk individuals.

1.3.3 Treatment Modalities

There are four treatment options: oral medications, exercise, medical nutrition therapy (MNT), and insulin. Although practically every diabetic in India uses the fifth modality—Ayurveda, Homeopathy, advise from family and friends, etc.—it seldom has a solid scientific foundation. Furthermore, as diabetes is a chronic, progressing condition, short-term experimental treatments should be avoided.

Oral hypoglycaemic medications (OHA) should thereafter be added either alone or in combination, following a tiered and stepwise approach to type 2 diabetes management (Bloom garden, 1998). To achieve proper glycaemic control, pure insulin therapy (Garg, 1998) and coalition therapy with OHA should be administered last (Table-1).

TABLE 1

Metabolic control (ADA recommendation)

Biochemical index	Good	Acceptable	Poor
Fasting/preprandial plasma glucose	80-120	≤ 140	> 140
Postprandial plasma glucose	80-160	< 180	> 180
Glycosylated Hb	< 8.5%	8.5 – 9.5%	> 9.5%

1.3.4 Lifestyle Modifications

Any strategy for treating diabetes mellitus must include MNT and exercise. It is important to emphasise that ethnicity of diet should be maintained rather than delivering ready-made advice without getting into the complexities of MNT. Dietary changes should include reducing calorie intake, promoting complex carbohydrates and fibre, and avoiding free and refined sugar. After determining the underlying heart condition, all should be urged to go for a daily 30-minute brisk walk.

1.3.5 Single Oral Agents

Sulphonylurea, metformin, acarbose, thiazolidinediones and repaglinide are 5 groups of drugs.

Sulphonylureas : Sulphonylureas bind to a region of the beta cell plasma membrane known as the sulphonylurea receptor that is connected to ATP-dependent K+ channels (SUR). Because it depolarizes the membrane and activates voltage-dependent Ca+2 channels, insulin is exocytosed when the K+ channel is closed. Each pharmaceutical manufacturer strives to differentiate its sulphonylurea in order to increase market share; nevertheless, these variations are modest, and there is little evidence that they have a major impact on therapy success. Sulphonylurea therapy should start at the lowest dose suggested, with the exception of patients who have significant symptoms and high blood sugar. If taken 30 minutes before meals, gliclazide, glipizide, and glibenclamide are more absorbed and more effective. Gaining weight and hypoglycemia are frequent side effects.

TABLE 2

Drug	Daily dose	Duration	Renal excretion	Cost (Rs)
Glibenclamide	2.5- 20	16 – 24	50	0.50
Glipizide	2.5- 20	12 – 16	85	0.50
Gliclazide	80 – 320	10 – 20	70	3.00 (6.50)
Glimepiride	1 – 8	24	60	*11.00*

In light of exaggerated claims made by the pharmaceutical industry, two more recent sulphonylureas demand special. Although the azabicyclo-octyl ring of the gliclazide molecule is thought to improve platelet function and take into account vascular issues caused by atherosclerosis in individuals with diabetes, a better and more clinically validated impact can be obtained by taking half a tablet of the much less expensive aspirin. In contrast to other sulphonylureas, glimepiride (Bailey, 1996), a novel drug on the Indian market, attaches to a 65-kDa protein on the beta cell. Glimepiride is more pancreas-specific than other sulphonylureas because it interacts with the cardiovascular ATP dependent K+ channel less than glibenclamide. Glimepiride is thought to have a higher insulin-mimetic impact in peripheral tissue since it causes lower levels of insulin and C-peptide with identical glucose control. Without interruption from contemporaneous meals, gastrointestinal absorption is fully completed, and medication buildup is not observed with impaired renal function. It is necessary to assess the significance of these distinctions in terms of general clinical application.

Metformin (500 mg pill, Rs.0.65): Metformin decreases hunger and intestinal mucosal glucose transfer. Because of improved hepatic insulin sensitivity and suppression of gluconeogenesis, it primarily reduces endogenous hepatic glucose production. It also increases peripheral insulin-mediated glucose absorption, decreases fatty acid oxidation, and reduces fatty acid synthesis (Saitiel, 1996). In addition, there is an antiatherogenic impact that doesn't seem to be connected to glycemia alterations. This benefit includes improved lipid profiles with 10–20% lower triglyceride levels, 5–10% lower total and LDL cholesterol, and higher HDL levels. It also includes increased fibrinolytic activity (which reduces PAI-1), which had a 39 percent decreased risk (p=0.01) of myocardial infarction (MI) (UK, 1998). Although it is chosen as the first line of treatment for type 2 diabetics who are obese, non-obese diabetics have also found it to be just as effective. The recommended starting dose of metformin is 500 mg twice or three times a day with meals, gradually increasing to 2-3 gm. Nausea, a metallic taste in the mouth, and increased stool frequency are typical gastrointestinal side effects. Metformin medication should be avoided in those with renal, hepatic, and cardiac impairment due to the elevated risk of lactic acidosis. Almost all brands cost 65 paise for a 500-mg tablet.

Acarbose ($5.50 for a 50 mg tablet) competes with alpha-glucosidase enzymes like sucrase, maltase, dextrinase, and glucomylase for the carbohydrate binding domain. it prevents the breakdown of oligosaccharides. Gas, diarrhoea, and abdominal pain are brought on by the leakage of unabsorbed carbohydrates into the large intestine, where bacteria transform them into short-chain fatty acids. At a maximum dose of 100 mg three times during meals, a mean drop in FPG of around 20 mg/dl, PPPG of approximately 50 mg/dl, and HbAlc of approximately 0.86 percent are to be predicted. With a diet strong in carbohydrates, it works best. To avoid adverse effects, it should be titrated in accordance with Table 3. When given to patients with type 2 diabetes and cirrhosis, acarbose also significantly reduces blood ammonia, increases stomach peristalsis, and improves the clinical indications of encephalopathy (Bloom garden, 1998).

TABLE 3

Acarbose dosing schedule [10]

Week	BF	Lunch	Dinner
1	—	—	1/2 tab
2	1/2 tab	—	1/2 tab
3-4	1/2 tab	1/2 tab	1/2 tab
5	1/2 tab	1/2 tab	1 tab
6	1 tab	1/2 tab	1 tab
7-8	1 tab	1 tab	1 tab
9	1 tab	1 tab	2 tab
10	2 tab	1 tab	2 tab
11-12	2 lab	2 tab	2 tab

Thiazolidinedione (TZD): The TZD are a special class of medications that increase insulin activity and lower hyperinsulinemia by directly altering the mechanism of insulin resistance. Peroxisome proliferator activator receptor, or PPAR-, is found in adipocytes, skeletal muscle, and the liver. By binding to PPAR-, TZD controls the transcription of several molecules crucial for insulin action and lipid metabolism. The biological activity and receptor binding of many TZDs are interdependent. Rosiglitazone (RST) binds ten times stronger than pioglitazone, which in turn binds ten times stronger than triglitazone, similar to the clinical dosages of 2-8 mg, 30-50 mg, and 200-800 mg. TNF-, a putative mechanism in the state of insulin resistance, is likewise inhibited by TZDs (Saitiel, 1996). Due to its hepatotoxicity, troglitazone has been taken off the market globally. Currently, rosiglitazone and pioglitazone are utilized all over the world. RST is probably about to be released to the Indian market. With a decrease in FPG of around 75 mg/dl at eight weeks, RST monotherapy produces the best results, almost matching sulphonylureas in effectiveness. In addition, it raises HDL and LDL cholesterol while lowering triglycerides and free fatty acids. However, the size, buoyancy, and oxidative sensitivity of LDL particles do increase. Its preventive effects on the development of atherosclerosis remain undetermined. RST does not undergo hepatic cytochrome oxidation, hence abnormal liver chemistry is seen in clinical studies at a rate that is around one-tenth that of troglitazone (Bloom garden, 1999).

Repaglinide: (Rs3.50 per 0.5 mg tablet) Repaglinide is a short acting non-sulfonylurea insulin secretagogue and a carbamoylmethyl benzoic acid derivative. It works differently from sulphonyl urea in that it interacts with the ATP-sensitive potassium channel on pancreatic beta cells to stimulate insulin production. Because of its fast (5–10 min) absorption, it is suitable for a dose before meals. Due to the short half-life (one hour), maximum plasma levels are obtained in 30 to 60 minutes, allowing for quick action and lowering the risk of postprandial hypoglycaemic episodes. It can be given successfully to patients with mild to severe renal failure because it is metabolised predominantly by the liver (98 percent) and removed primarily by bile (90 percent). A pre-prandial dose of 0.5-2 mg is given to begin. (Gomis, 1999).

1.3.6 Coalition Therapy (Oha And Insulin)

Combination therapy using insulin and OHA has gained popularity over the past 20 years. Coalition therapy has two components (Saini, 1999). If a patient's glycemic control with OHA is unacceptably poor, extra nighttime insulin therapy may be recommended. Patients who are uncontrolled with or require large doses of insulin monotherapy can also receive supplementary OHA therapy in the hopes of improving glycemic control and lowering insulin doses. More patients accept additional insulin therapy, which also improves glycemic control, accelerates glycemic control, improves lipid profiles, reduces the need for insulin, prevents weight gain, and is cost-effective (Pugh, 1992; Jarvinen, 1992; Riddle, 1998; Romano, 1997)

INDICATION FOR COALITION

Secondary failure from sulfonylurea therapy occurs at a rate of 5–20% each year. As a result, treatment fails after 5 to 20 years. Therefore, patients on OHA medication at full dosages who are unable to keep their glycemic control at a tolerable level for 3-6 months should begin coalition therapy.

COALITION REGIMES

Sulfonylurea and insulin therapy: It lowers glycosylated hemoglobin by 1-2 percent and fasting serum glucose by 50-80 mg/dl (Johnson, 1996; Edelman, 1995). Except in cases when the fasting blood glucose level is greater than 300 mg/dl and in cases where the patient is severely obese, glycemic control improves in patients with the lowest glycemic control. Metformin and Insulin Therapy: In a recently completed trial, obese and non-obese type-2 diabetic patients received either metformin plus insulin or sulfonylurea plus insulin (Taskenen, 1989). Metformin treatment provided comparable glycemic control in the non-obese group, but with lower insulin dosages and with less weight gain. Glycemic control was better and there was higher weight reduction in the metformin group in obese patients (Lebovitz, 1997).

MATERIALS AND METHODS: -

Study Area:

The Present study was conducted in Department of Otorhinolaryngology at Index Medical College Hospital & Research Centre, Indore (M.P.)

Study Population:

This cross-sectional, observational study was conducted in the Department of ENT, IMCHRC, Indore, India. The study included 100 patients.

Study Groups:

Cases = 50 (Diabetic Patients)
Control = 50 (Non-Diabetic Patients)

INCLUSION CRITERIA:

CASES	CONTROLS
Age range 25 to 75 years	Age range 25 to 75 years
Known cases of Type 2 DM	Non-Diabetes Mellitus with Hba1c <6.5
Patients giving consent	Patients giving consent

EXCLUSION CRITERIA:

1. Subjects below 30 and above 75 years of age.
2. Patients with history of noise induced HL
3. Patients with history of CSOM
4. Patients with history of perforation of Tympanic Membrane
5. Patient unwilling for participating in study
6. Patient with known history of hyperthyroidism.
7. Patients with prolonged use of ototoxic medication.

METHODOLOGY

Utilizing an MAICO MA 42 audiometer, pure-tone audiometry was carried out using bone conduction at 500-4,000 Hz and air conduction at octave frequencies from 250-8,000 Hz. A room that was soundproof was used for the test. The audiometer was calibrated so that there is no A-B gap and normal human hearing is at 0 dB for both bone conduction and air conduction. The manual pure tone audiometry method was based on the American Society for Speech and Hearing Association [ASHA] 1978 recommendations (PTA).

STATISTICAL ANALYSIS

The IBM SPSS version 20 software was used for all data analysis. The tables were created using cross tabulation and frequency distribution. The mean and standard deviation were used to express quantitative variables. Data that was categorical was reported as a percentage. The graphs were made using PRISM and Microsoft Office. To compare the means, ANOVA and the student t-test were employed. The Chi Square test was used to compare the category data. Significant P values are those with a value less than 0.05.

OBSERVATIONS:

Table 1: Comparing Degree Of Hearing Loss In Both Ears

DEGREE OF HEARING LOSS	RIGHT EAR		LEFT EAR	
	CASE	CONTROL	CASE	CONTROL
NORMAL HEARING	12 (24)	29 (58)	10 (20)	34 (68)
MILD HEARING LOSS	6 (12)	3 (6)	15 (30)	2 (4)
MODERATE LOSS	16 (32)	8 (16)	12 (24)	4 (8)
MODERATELY SEVERE	10 (20)	7 (14)	6 (12)	3 (6)
SEVERE	6 (12)	3 (6)	6 (12)	7 (14)
P value	0.016		<0.001	

Table 2: Diabetes Duration In Cases

DIABETES DURATION (Years)	FREQUENCY	PERCENT
<5	7	14.0
6 - 10	22	44.0
>10	21	42.0
Total	50	100.0

Table 3: Comparing Degree Of Hearing Loss Of Right Ear With Duration Of Diabetes.

DEGREE OF HEARING LOSS	DIABTES DURATION (Years)			P value
	<5	6 - 10	>10	
NORMAL HEARING	6	5	1	0.006
MILD HEARING LOSS	0	4	2	
MODERATE LOSS	1	7	8	
MODERATELY SEVERE	0	3	7	
SEVERE	0	3	3	

In present study, on comparing the degree of hearing loss of right ear with the duration of diabetes it was found that among the mild hearing loss patients, majority had diabetes duration between 6-10 years (18.2%). Of the moderate hearing loss patients, majority had diabetes duration of >10 years (38.1%) followed by 31.6% patients who had diabetes duration between 6-10 years. Among the moderately severe patients, majority had diabetes duration of >10 years (33.3%) followed by 13.6% patients who had diabetes duration between 6-10 years. Similarly, majority of the patients with severe hearing loss had diabetes duration of >10 years (14.3%). This proved that degree of hearing loss of right had direct association with the diabetes duration. Hearing loss increases with increasing the diabetes duration as revealed by the present study findings.

Table 4: Comparing Degree Of Hearing Loss Of Left Ear With Duration Of Diabetes.

DEGREE OF HEARING LOSS	DIABTES DURATION (Years)			P value
	<5	6 - 10	>10	
NORMAL HEARING	1	9	5	0.001
MILD HEARING LOSS	0	2	10	
MODERATE LOSS	0	4	2	
MODERATELY SEVERE	5	5	0	
SEVERE	1	2	4	

In present study, on comparing the degree of hearing loss of left ear with the duration of diabetes it was found that among the mild hearing loss patients, majority had diabetes duration between 6-10 years (40.9%). Of the moderate hearing loss patients, majority had diabetes duration of >10 years (47.6%) followed by 9.1% patients who had diabetes duration between 6-10 years. Among the moderately severe patients, majority had diabetes duration of 6-10 years (18.2%) followed by 9.5% patients who had diabetes duration between >10 years. Similarly, majority of the patients with severe hearing loss had diabetes duration of >10 years (19%). This proved that degree of hearing loss of left ear had direct association with the diabetes duration. Hearing loss increases with increasing the diabetes duration as revealed by the present study findings.

Table 5: Comparison Of Age With Mean Hearing Threshold Between Groups

GROUP	Age Of Patients		PTA Rt.	PTA Lt.
CASES	31 - 40	Mean	39.28	38.45
		Std. Deviation	15.2061	13.8206
	41 - 50	Mean	50.16	52.27
		Std. Deviation	29.1545	14.12
	51 - 60	Mean	43.62	44.42
		Std. Deviation	15.1273	16.7591
	61 - 70	Mean	53.23	49.3
		Std. Deviation	21.1666	24.1433
	71 - 75	Mean	60.75	60.3
		Std. Deviation	3.8891	6.6468
CONTROL	20 - 30	Mean	30.36	12.91
		Std. Deviation	21.6577	6.1879
	31 - 40	Mean	27.3	14.5
		Std. Deviation	20.2233	4.9497
	41 - 50	Mean	40.02	28.37
		Std. Deviation	20.9981	15.9154
	51 - 60	Mean	22.29	21.42
		Std. Deviation	16.7334	19.4356
	61 - 70	Mean	40.3	54.05
		Std. Deviation	26.2947	25.7487
	71 - 75	Mean	16	15
		Std. Deviation	.	.
P value			<0.001	0.002

Age also had the direct association with the pure tone audiometry threshold level which means by increasing the age of the diabetes patients, hearing loss also increases as revealed by the significant p value of <0.005.

Table 6: Comparison Of Mean Hearing Loss With Duration Of Diabetes

Diabetes Duration (Years)		PTA Rt.	PTA Lt.
<5 years	Mean	23.91	32.94
	Std. Deviation	9.6423	9.62
6 - 10	Mean	46.51	43.63
	Std. Deviation	19.4413	20.0476
>10	Mean	56.71	53.83
	Std. Deviation	17.7887	16.8969
P value		<0.001	0.005

In Present study, with increasing the duration of diabetes, pure tone audiometry threshold of both the ears increases, which shows a direct association of diabetes duration with hearing loss.

RESULTS:

On comparing the degree of hearing loss between both the groups and the mean hearing threshold of both the ears with duration of diabetes it was found that distribution of degree of hearing loss between the cases and control was significantly different.

DISCUSSION:

Diabetes mellitus (DM) is a metabolic illness that results in elevated blood glucose levels and long-term vascular as well as neurological consequences. It is caused by a relative or absolute insulin shortage. Type-2 diabetes is caused by a combination of reduced beta pancreatic cell function and insulin resistance at the receptor level, whereas type-1 diabetes is caused by the autoimmune destruction of insulin-producing beta cells of the pancreas.
The current study evaluates the prevalence of sensori-neural hearing loss among type-2 [noninsulin-dependent (NIDDM)] patients.

In present study in Case group majority of the patients were in the age group between 51-60 years (34%) followed by 61-70 years (26%) and 41-50 years (22%). In control group, majority of the subjects had age between 51-60 years (34%) followed by 61-70 years (26%) and 20-30 years (16%). The age distribution between both the groups was significant with p value of 0.046. Similarly, Sachdeva K et al in their study enrolled 184 subjects, 92 were in each group case and control group. Maximum numbers of patients were found in the age group of51-60 years in both group. Age was matched between groups. (Carhart et al, 1959)

In current study on comparing the degree of hearing loss between both the groups it was found that in right ear, majority of the patients in cases had moderate hearing loss (32%) followed by moderately severe hearing loss (20%) mild hearing loss (12%) and 12% had severe hearing loss. In Control group, majority of the subjects had no hearing loss (58%) while 16% had moderate loss, 14% had moderately severe hearing loss and 6% had severe hearing loss. The Distribution of degree of hearing loss on right ear between the groups was significantly different. That means cases had more severe hearing loss of right ear as compared to Control group as revealed by the significant p value of 0.016. On comparing the degree of hearing loss between both the groups it was found that in left ear, majority of the patients in cases had mild hearing loss (30%) followed by moderately hearing loss (24%), and 12% had severe hearing loss. In Control group, majority of the subjects had no hearing loss.(68%) while 8% had moderate loss, 6% had moderately severe hearing loss and 14% had severe hearing loss. The Distribution of degree of hearing loss on right ear between the groups was significantly different. That means cases had more severe hearing loss of right ear as compared to Control group as revealed by the significant p value of <0.001. In the Sachdeva K et al. study, it was discovered that 33.7 percent (31) of the diabetes patients had mild to moderate sensorineural hearing loss, compared to 13 percent (12) in the control group. This difference, with a p value of 0.009, is significant. Significant differences were discovered between the case and control groups in terms of many auditory parameters in both the right and left ears.

In present study on comparing the mean pure tone threshold of right ear with duration of the disease, in group where duration of disease is < 5 year was 23.9 dB hearing loss, in group with duration of disease 5 - 10 year was 46.5 dB hearing loss and in >10-year age 56.7 dB hearing loss. This is significant as denotes by significant P value of <0.001. Similar trend was followed in pure tone threshold of the left ear with duration of disease. This was in line with the study done by Bressler , et al (1992).

On comparison between the different age group and pure tone threshold of right ear, there is progressive trend in hearing loss is seen in the cases with increases age of the subjects.

CONCLUSION:

The present study concluded that Age had the direct association with the pure tone threshold level which means by increasing the age of the diabetes patients, hearing loss also increases as revealed by the significant p value of <0. 005.Hearing loss increases as the age of the diabetic person progresses, there might be bias in geriatric age group as chances of presbycusis also increases with the age. Although it has been observed that diabetic patients do witness early onset of higher degree of hearing loss as compared to general population.

Current study concludes that the diabetic patients should undergo periodic audiometry evaluation and regular monitoring of their blood sugar levels to avoid early or untimely severe hearing impairment. And also, the study had some limitation in terms of small sample size cross sectional and single centers, which do not replicate the actual population behaviour. This study aimed to find prevalence of hearing loss and only a single PTA test was done, but serial monitoring of hearing ability in the diabetic patients would be more helpful in prevention of the auditory impairment.

REFERENCES:

1. Anindita R, Adnan A, Munir D, Syafril S, Ashar T. Comparison of Sensorineural Hearing Loss Degree between Controlled and Uncontrolled Type 2 Diabetes Mellitus Patients. Int J Sci Stud 201
2. Bailey CJ, Turner RC. Metformin. N Engl J Med. 1996; 334:574–579.

3. Bainbridge KE, Hoffman HJ, Cowie CC. Diabetes and hearing impairment in the United States: audiometric evidence from the National Health and Nutrition Examination Survey, 1999 to 2004. Ann Intern Med. 2008; 149:1–10.
4. Bhaskar KN, Chalihadan S, Vaswani R, Rehaman CP. Childhood and adolescent overweight and obesity—a public health challenge in India. *Int J Sci Stud.* 2014;2(4):1–16.
5. Bloomgarden ZT. New and traditional treatment of glycaemia in NIDDM. Diabetic Care. 1996;19:295–299.
6. Bloomgarden ZT. Treatment of type-2 diabetes. Diabetes Care. 1999; 22:170–174.
7. Bloomgarden ZT. Type-2 diabetics: its prevalence, causes and treatment. Diabetes Care. 1998; 21:860–865.
8. Bressler R, Johnson D. New pharmacological approaches to therapy of NIDDM. Diabetes Care. 1992; 15:792–802.
9. Cooke DW, Plotnick L. Type 1 diabetes mellitus in paediatrics. Pediatr Rev 2008,29(11):374-84
10. Diabetes and Hearing loss. Centres for Disease Control and Prevention 2021. Available from: Diabetes and Hearing Loss | Diabetes | CDC
11. Edelman SV, White D, Henry RR. Intensive insulin therapy or patients with type-II diabetes. International Diabetes Monitor. 1995; 7:1–9.
12. Ferris FL, Davis MD, Aiello LM. Treatment of diabetic retinopathy. N Engl J Med. 1999; 341:667–668.
13. Folsom AR, Szklo S, Stevens P, Liao F, Smith R, Eckfeldt JH. A perspective study of coronary heart disease in relation to fasting insulin, glucose, and diabetes: The atherosclerosis risk in community (ARIC) study. Diabetes Care. 1997; 20:935–942.
14. Fukushima H, Cureoglu S, Schachern PA, Paparella MM, Harada T, Oktay MF, et al. Effects of type 2 diabetes mellitus on cochlear structure in humans. Arch Otolaryngol Head Neck Surg2006; 132:934 8.
15. Fukushima J, Cureolglu S, Schachern PA, Paparella MM, Harada T, Mehet OF. Effects of type 2 diabetes on cochlear structure in humans. Arch Otolaryngol Head Neck Surg. 2006; 132:934–938.
16. Garg MK, Saini JS. Insulin therapy in noninsulin dependent diabetes mellitus. Golden Jubilee National Medical Update. 1998:39–51.
17. Gomis R. Repaglinide as monotherapy in type-2 diabetes. Exp Clin Endocrinol Diabetes. 1999;107(suppl 4):S133–S135.
18. Gu K, Cowie CC, Harris MI. Mortality in adults with and without diabetes in a national cohort of the US population. 1971–1993. Diabetes Care. 1998; 21:1138–1145.
19. Guidelines for Manual Pure Tone Threshold Audiometry. American Speech-Hearing Language Association 2005. Available from: Guidelines for Manual Pure-Tone Threshold Audiometry (asha.org)
20. Hirsch IB, Riddle MC. Current therapies for diabetes. Endocrinol MetabClin North America. 1997; 26:511–678.
21. Johnson JL, Wolf SL, Kabadi UM. Efficacy of insulin and sulfonylurea combination therapy in type II diabetes: a meta-analysis of randomised placebo-controlled trials. Archh Intern Med. 1996; 156:259–264.
22. Jordao AMD. Consideration sur un cas du diabete. Union medical du Paris. 1857; 11:446.
23. Kakarlapudi V, Sawyer R, Staecker H. The effect of diabetes on sensorineural hearing loss. Otol Neurotol. 2003;24:382–386.
24. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995–2025. Diabetes Care. 1998; 21:1414–1431.
25. Lebovitz HE. Alpha-glucosida.se inhibitors. Endocr Metab Clin North Am. 1997; 26:539–552.
26. Lisowska G, Namyslowski G, Morawski K, Strojek K. Cochlear dysfunction and diabetic microangiopathy. Scand Audiol. 2001;30(suppl 52):199–203.
27. Malmberg K, Ryden L, Hamsien A, Herlitz J, Waidenstrom A, Wedel H. Effects of insulin treatment on cause specific one year mortality and morbidity in diabetic patients with acute myocardial infarction. DIGAMI study group. Eur Heart J. 1996; 17:1337–1344.
28. Ohkuba Y, Kishikawa H, Araki E, Miyata T, Isami S, Motoyosyi S. Intensive insulin therapy prevents the progression of diabetic microvascular complications in Japanese patients with non-insulin dependent diabetes mellitus. A randomized prospective 6-year study. Diabetes Res Clin Pract. 1995; 28:103–117.
29. Pugh JA, Wagner ML, Sawyer J, Ramirez G, Tuley M, Friedberg SJ. Is combination sulfonylurea and insulin therapy useful in NIDDM patients? A meta-analysis. Diabetes Care. 1992; 15:953–959.
30. Riddle MC, Scheider J, The Glimeperide Combination Group Beginning insulin treatment of obese patients with evening 70/30 insulin plus glimeperide versus insulin alone. Diabetes Care. 1998; 21:1052–1057.
31. Ritz ER, Orth SR. Nephropathy in patients with type-2 diabetes mellitus. N Engl J Med. 1999; 341:1127–1133.
32. Robinson AC, Burki J, Robinson S, Johnston DG, Elkells RS. The effect of metformin on glycemie control on serum lipids in insulin treated NIDDM patients with suboptimal metabolic control. Diabetes Care. 1998; 21:701–705.
33. Romano G, Patti L, Inneli F, Di-Marino L, Annuzzi G, Lavicoli M, Coronel GA. Insulin and sulfonylurea therapy in NIDDM patients: Are the effects on lipoprotein metabolism different even with similar blood glucose control. Diabetes. 1997; 46:1601–1606.
34. Sachdeva K et al. Int J Otorhinolaryngol Head Neck Surg. 2018 Mar;4(2): 499-507
35. Saini JS, Garg MK. Insulin and OHA: Days of coalition therapy. API Medicine Update. 1999;9(II):287–299.
36. Saitiel AR, Olefsky JM. Thiazolidinediones in the treatment of insulin resistance and type-2 diabetes. Diabetes. 1996; 45:1661–1669.
37. Stach BA. Clinical Audiology: An introduction. 2nd edition (2010) Singular Publishing Group: USA
38. Taskenen MR, Sane T, Helve E, Karone SL, Nikkila EA, Yki-Jarvinen H. Bedtime insulin for suppression of overnight free fatty acid, blood glucose, and glucose production in NIDDM. Diabetes. 1989; 38:580–588.
39. Thiago Hernandes Diniz and Heraldo Lorena Guida, Hearing Loss in Patients with Diabetes Mellitus. Braz J Otorhinolaryngol. 2009;75(4):573-8
40. Tiwari A, Mudhol RS. Prevalence of sensorineural hearing loss among type-II diabetes mellitus patients attending KLES Dr. Prabhakar Kore Hospital and MRC: A cross-sectional study. Indian J Health Sci Biomed Res 2018; 11:165-9.
41. Tomisawa H. Diabetic changes in the stria vascularis in humans—a study of PAS- stained temporal bone sections. Nippon Jibiinkoka Gakkai Kaiho 103(11):1227-37,2000.
42. UK Prospective Diabetes Study Group Effect of intensive blood-glucose control with metformin on complications in overweight patients with type-2 diabetes (UKPDS 34) Lancet. 1998; 352:854–865.
43. Yki-Jarvinen H, Kauppila M, Kujansuu E, Lahti J, Marjanen T, Niskanen L. Comparison of insulin regimens in patients with non-insulin dependent diabetes mellitus. N Engl J Med. 1992; 327:1426–3143.