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ORIGINAL ARTICLE

Otological and Visual Implications of Diabetes Mellitus in North Indian Population

Prateek Roy[1] · Manish Chandra[1] · Anupam Mishra[1] · Veerendra Verma[1] · K. K. Sawlani[3] · H. P. Singh[1] · Sunil Kumar[1] · Sandeep Saxena[2] · A. B. Singh[1] · Shivani Pandey[4] · Kauser Usman[3] · Pramod Kumar[2] · D. Himanshu[3]

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Abstract The worldwide population of diabetic patients is increasing alarmingly with India claiming number one position. It causes irreversible damage to cochlear hair cells, vestibular apparatus, visual pathway, nephrons, nerves, if not checked in time. A total of 188 patients of diabetes mellitus were included in this prospective study. The patients underwent routine anamnesis, hearing handicap inventory and dizziness handicap inventory assessment along with clinical examination for audiological, vestibular, neurological and ophthalmological (fundoscopy) status. In our study a sensorineural hearing loss, retinopathy, neuropathy, vestibulopathy was seen in diabetic patients.

Keywords Hearing loss · Vestibulopathy · Retinopathy · Nephropathy · Diabetes mellitus

Introduction

Diabetes mellitus (DM) characterized by hyperglycemia, glycosuria, polyuria, polyphagia, polydypsia, ketonaemia and negative nitrogen balance, is the major life threat of present times [1, 2]. Irrespective of its types (type 1 and type 2) it can cause irreversible damage or complications if not checked in time; complications may arise in the form of neuropathy, retinopathy, nephropathy, vascular damage, erectile dysfunction, non-alcoholic fatty liver disease and many more. Research has hinted at an increased risk for hearing loss in diabetic patients [3–5], but confounders of noise exposure, ototoxic drug exposure, presbycusis, and known syndromes that affect both glucose metabolism and cochlear function make it difficult to establish this association. Currently, there are no formal recommendations for screening for hearing loss in diabetic patients. The existence of diabetes-related hearing loss increases the possibility of a corresponding condition: diabetic vestibulopathy. Physicians have attributed impaired balance and gait in diabetic patients to impaired proprioception. It is possible that diabetic patients also have impaired vestibular input because the microcirculation that maintains cochlear function is entangled with the circulation of the vestibular organs. Diabetic retinopathy (DR) is a well-recognized complication and has been shown that nearly all type 1 and 75% of type 2 diabetes will develop DR after 15 years' duration of diabetes [6, 7]. DR is classified into non proliferative diabetic retinopathy (NPDR) characterized by microaneurysms, retinal hemorrhages, cotton-wool spots, or venous beading and proliferative diabetic retinopathy (PDR) which occurs with further retinal ischemia is characterized by growth of new blood vessels on retina and posterior surface of vitreous. Nephropathy is the leading cause of chronic renal failure, the initial marker being microalbuminuria. A greater proportion of patient with type 2 DM compared with type 1 DM develop microalbuminuria [7, 8]. It has been postulated that fluctuations (increase in HbA1C i.e. glycosylated haemoglobin of more than 2% between two consecutive measurements within 3 months' interval ± 2 weeks) in HbA1C is the predictor of nephropathy in type1 DM. Diabetes induced neuropathies are classified into symmetrical or asymmetrical (focal or multifocal) forms, the symmetrical form is primarily sensory and autonomic whereas the asymmetric form can be

✉ Manish Chandra
drchandramanish@yahoo.co.in

[1] Department of ENT Head Neck Surgery, KGMU, Lucknow, India

[2] Department of Ophthalmology, KGMU, Lucknow, India

[3] Department of Medicine, KGMU, Lucknow, India

[4] Department of Biochemistry, KGMU, Lucknow, India

sensory, motor or both as well as affecting the individual cranial or peripheral nerves. Diabetic peripheral neuropathy is defined as stocking-glove neuropathy or somatic and/or autonomic neuropathy, which affects the longest nerve first before progressing proximally. Distal symmetrical form of diabetic peripheral neuropathy otherwise known as diabetic sensorimotor peripheral neuropathy is the primary risk factor for the development of diabetic foot ulcer, responsible for 85% of lower extremity amputation in diabetes patient. Thus it is necessary to monitor neuropathy if at all present and to find its significance level to take the proper treatment strategy.

The worldwide population of diabetic patients is increasing alarmingly with India claiming number one position in the chart with 50.8 million diabetics; a prediction made by the WHO stated that developing countries would face the impact of the epidemic in the 21st century; the population which was predicted to come under this syndrome by 2010 was 285 million people (6.4% of world's adult population) and expected to exceed to 438 million by 2030 (7.8% of the world's adult population). Data from the 2011 National Diabetes Fact Sheet (released Jan. 26, 2011) reveals that 25.8 million children and adults in the United States have diabetes which is 8.3% of the population.

Materials and Methods

A total of 188 serial patients of diabetes mellitus were included in this prospective study that attended the outpatient department of Otorhinolaryngology and Head and neck Surgery of a tertiary care university hospital of North India (Lucknow). The excluded set of patients included those with mental retardation, pregnancy, and those on drugs causing neuropathy, retinopathy, audiopathy. (such as isoniazid, dapsone, thalidomide, nitrofurantoin, metrogyl, amiodarone, cisplatin, phenytoin, disufiram, chloroquine, ethambutol, thioridazine, tamoxifen, chlorpromazine, Amphotericin-B, aminoglycosides, rifampin, vancomycin, acyclovir etc.).

These patients underwent routine anamnesis, hearing handicap inventory (HHI) and dizziness handicap inventory (DHI) assessment along with clinical examination for audiological, vestibular, neurological and ophthalmological (fundoscopy) status. The vestibular examination protocol included: Gait assessment (patient asked to walk along a straight line to a fixed target with eyes open and then closed), Rhomberg test (patient asked to stand with feet together and arm by the side with eyes open first, then with eyes closed); Untenberger test (patient standing with outstretched hands and eyes closed was asked to make 50 steps on the spot inside the circle), Posturometry test (patient standing on special platform conditions with eyes open/closed) and Bithermal caloric test (patient supine with head end elevated to 30° and each ear irrigated for 40 s with hot (44 °C) and cold (30 °C) to stimulate vestibular labyrinth and then patient is observe for nystagmus). The audiological examination included: classical pure tone audiometry (PTA) including SISI (short increment sensitivity index), TDT (tone decay test), Tympanometry, Speech reception threshold (SRT). The retinal examination by fundoscopy was undertaken to classify retinopathy as Non proliferative diabetic retinopathy (NPDR) or Proliferative diabetic retinopathy (DPR). The neurological examination consisted of sensory examination testing for pain sensation (pin prick), light touch sensation (brush), position sense, stereognosis, graphesthesia, and extinction and cranial nerve examination along with cerebellar function testing. In addition a few investigations were also undertaken such as blood sugar (FBS, PP, RBS), HbA1c, serum creatinine, serum urea, urine protein, microalbuminuria, urine routine/microscopy, lipid profile, serum cholesterol, triglyceride, LDL, HDL.

Statistical Analysis

Continuous data were summarized as Mean ± SE (standard error of the mean) while discrete (categorical) in no and %. Association between variables was done by Chi square (χ^2) test. A two-tailed ($\alpha = 2$) p value less than 0.05 ($p < 0.05$) was considered statistically significant. Analysis was performed on SPSS software (Windows version 17.0).

The most widely used measure of central tendency is arithmetic mean, usually referred to simply as the mean, is calculated as

$$\overline{X} = \frac{\sum_{i=1}^{n} X_i}{n}$$

The median is generally defined as the middle measurement in an ordered set of data. That is, there are just as many observations larger than the median as there are smaller. The median (M) of a sample of data may be found by first arranging the measurements in order of magnitude (preferably ascending). For even and odd number of measurements, the median is evaluated as

$M = [(n + 1)/2]$th observation: odd number

Find the value at position n/2

Find the value at position n+1/2

Find the average of the two values to get the median

The standard deviation (SD) is the positive square root of the variance, and is calculated as

$$SD = \sqrt{\frac{\Sigma X_i^2 - \frac{(\Sigma X_i)^2}{n}}{n-1}}$$

and SE (standard error of the mean) is calculated as

$$SE = \frac{SD}{\sqrt{n}}$$

where n = no. of observations

The Chi square (χ^2) test is used to compare the categorical data as

$$\chi^2 = \Sigma\Sigma \frac{(F_{ij} - f_{ij})^2}{f_{ij}}$$

where, F_{ij} is the observed frequency while f_{ij} the expected frequency. The degrees of freedom (DF) is calculated as: DF = (r − 1) (c − 1)

Level of significance "*p*" is the probability signifies level of significance as is as follows:

$p > 0.05$ Not significant (ns).
$p < 0.05$ Just significant (*).
$p < 0.01$ Moderate significant (**).
$p < 0.001$ Highly significant (***).

Results

Demographics

The age of patients ranged from 25 to 75 years with mean (± SE) 51.59 ± 0.76 years and median 50 years. The patients were mostly 41–50 years old (37.2%) in which females (59.6%) predominated. Further, the duration of diabetes mellitus (DM) of patients ranged from 0.1 to 30.0 years with mean (± SE) 7.16 ± 0.0.48 years and median 6 years. The duration of DM of most of patients were < 5 years (42.6%) as shown in Table 1.

Table 1 Demographic characteristics of patients

Demographic characteristics	No. of patients N (%)
Age (years):	
Mean ± SE, range, median	51.59 ± 0.76, 25–70, 50
20–30	8 (4.3)
31–40	20 (10.6)
41–50	70 (37.2)
51–60	58 (30.9)
61–70	32 (17.0)
Sex	
Female	112 (59.6)
Male	76 (40.4)
Duration of DM (years):	
Mean ± SE, range, median	7.16 ± 0.48, 0.1–30.0, 6
< 5	80 (42.6)
5–10	50 (26.6)
≥ 10	58 (30.9)

Biochemical Profile

The biochemical profile of patients at presentation are summarized in Table 2. The FBS, PP, HbA1c, S. creatinine, albuminuria, S. cholesterol and triglyceride of patients ranged from 53 to 565 mg/dl, 102–587 mg/dl, 5.3–14.7%, 0.5–6.4 mg/dl, 4–2480 mg/dl, 65–354 mg/dl and 54–601 mg/dl respectively with mean (± SE) 207.52 ± 6.37 mg/dl, 262.89 ± 7.08 mg/dl, 9.15 ± 0.18%, 1.77 ± 0.10 mg/dl, 303.03 ± 40.89 mg/dl, 151.60 ± 3.45 and 157.19 ± 6.81 mg/dl respectively and median 196 mg/dl, 244 mg/dl, 8%, 1 mg/dl, 60 mg/dl, 152 mg/dl and 133 mg/dl respectively. The FBS, PP, HbA1c, S. creatinine, albuminuria, S. cholesterol and

Table 2 Biochemical profile of patients

Biochemical profile	No. of patients N (%)
FBS (mg/dl):	
Mean ± SE, range, median	207.52 ± 6.37, 53–565, 196
≤ 126	30 (16.0)
127–200	82 (43.6)
201–300	52 (27.7)
≥ 301	24 (12.8)
PP (mg/dl):	
Mean ± SE, range, median	262.89 ± 7.08, 102–587, 244
≤ 200	50 (26.6)
> 200	138 (73.4)
HbA1c (%):	
Mean ± SE, range, median	9.15 ± 0.18, 5.3–14.7, 8
< 7	34 (18.1)
7–8	38 (20.2)
8–9	38 (20.2)
≥ 10	78 (41.5)
S. creatinine (mg/dl):	
Mean ± SE, range, median	1.77 ± 0.10, 0.5–6.4, 1
≤ 1.5	110 (58.5)
> 1.5	78 (41.5)
Albuminuria (mg/dl):	
Mean ± SE, range, median	303.03 ± 40.89, 4–2480, 60
< 30	86 (45.7)
30–300	62 (33.0)
> 300	40 (21.3)
S. cholesterol (mg/dl):	
Mean ± SE, range, median	151.60 ± 3.45, 65–354, 152
≤ 180	136 (72.3)
> 180	52 (27.7)
Triglyceride (mg/dl):	
Mean ± SE, range, median	157.19 ± 6.81, 54–601, 133
≤ 150	108 (57.4)
> 150	80 (42.6)

Table 3 Otorhinological profile of patients

Examination profile	No. of patients N (%)
Sensory abnormality	
Absent	106 (56.4)
Present	82 (43.6)
Cranial nerve abnormality	
Absent	170 (90.4)
Present	18 (9.6)
Cerebellar examination	
Normal	162 (86.2)
Abnormal	26 (13.8)
PTA	
Normal	78 (41.5)
Mild	64 (34.0)
Moderate	46 (24.5)
Vestibular examination	
Normal	26 (13.8)
Abnormal	162 (86.2)
Retinal examination	
No change	88 (46.0)
Mild change NPDR	66 (35.1)
Moderate change NPDR	14 (7.4)
Hypertensive retinopathy	12 (6.4)
PDR	8 (4.3)

triglyceride of most of the patients were in the range of 127–200 mg/dl (43.6%), $>$ 200 mg/dl (73.4%), $\geq$ 9% (41.5%), $\leq$ 1.5 mg/dl, $<$ 30 mg/dl, $\leq$ 180 mg/dl and $\leq$ 150 mg/dl respectively.

Otorhinolaryngological and Opthalmological Profile

The Otorhinological profile of patients at presentation are summarized in Table 3. The retinal examination of most of the patients showed no change (46.0%) followed by mild change NPDR (35.1%), moderate change NPDR (7.4%), hypertensive retinopathy (6.4%) and PDR (4.3%). Further, sensory and cranial abnormality was present in 82 (43.6%) and 18 (9.6%) cases respectively. Furthermore, 26 (13.8%) cases had cerebellar signs. The PTA of 78 (41.5%) cases was normal, 64 (34.0%) were with mild and 46 (24.5%) with moderate loss. Moreover, the vestibular abnormality was present in 162 (86.2%) cases.

The correlation of retinal examination with other variables is summarized in Table 4. On correlating, χ^2 test showed significant association of retinal examination (no change/mild change NPDR/moderate change NPDR/hypertensive retinopathy/PDR) with all demographic characteristics and few biochemical profiles viz. age ($\chi^2 = 76.69$, $p < 0.001$), sex ($\chi^2 = 12.33$, $p = 0.015$), duration of DM ($\chi^2 = 26.90$, $p = 0.001$), FBS ($\chi^2 = 34.29$, $p = 0.001$), PP ($\chi^2 = 23.91$, $p < 0.001$), HbA1c ($\chi^2 = 36.05$, $p < 0.001$), S. cholesterol ($\chi^2 = 13.82$, $p = 0.008$) and triglyceride ($\chi^2 = 30.45$, $p < 0.001$).

Diabetes mellitus is well known to affect the various body systems and Indians being genetically more prone to such effects may show some different pattern as compared to the western world. It is worthwhile correlating the various system-involvements with the diabetic profile per se. Accordingly the correlation of abnormal sensory examination with variables is summarized in Table 5. On correlating, χ^2 test showed significant association of abnormal sensory examination (absent/present) with age ($\chi^2 = 21.52$, $p < 0.001$), duration of DM ($\chi^2 = 7.65$, $p = 0.022$), FBS ($\chi^2 = 24.16$, $p < 0.001$), PP ($\chi^2 = 6.76$, $p = 0.009$), and S. cholesterol ($\chi^2 = 9.39$, $p = 0.002$). Hence association of abnormal sensations in diabetic may reflect an underlying hypercholesterolemia that needs to be investigated. However, abnormal sensory examination was not ($p > 0.05$) associated with sex, albuminuria and triglyceride.

The association of abnormal cranial examination with demographic and biochemical profile of patients is summarized in Table 6. On correlating, χ^2 test showed significant association of abnormal cranial examination (absent/present) with FBS ($\chi^2 = 27.61$, $p < 0.001$), PP ($\chi^2 = 16.37$, $p < 0.001$). However abnormal cranial examination was not ($p > 0.05$) associated with age, sex, duration of DM, HbA1c, S. cholesterol and triglyceride. Hence presence of cranial nerve involvement may reflect the severity of diabetes in terms of blood sugar status of short term only.

The association of abnormal cerebellar examination with demographic and biochemical profile of patients is summarized in Table 7. On correlating, χ^2 test showed significant association of abnormal cerebellar examination with FBS ($\chi^2 = 9.49$, $p = 0.023$) and Albuminura ($\chi^2 = 11.96$, $p = 0.003$). However, abnormal cerebellar examination did not ($p > 0.05$) found to be associated with age, sex, duration of DM, PP, HbA1c, S. creatinine, S. cholesterol and triglyceride. No specific conclusions can be drawn in this regard except that this may reflect the role of hyperosmolar state in general.

The association of PTA with demographic and biochemical profile of patients is summarized in Table 8. On correlating, χ^2 test showed significant association of PTA thresholds with FBS ($\chi^2 = 14.51$, $p = 0.024$). However, PTA did not ($p > 0.05$) found to be associated with age, sex, duration of DM, PP, HbA1c, S. cholesterol and triglyceride. It seems that the hyperosmolarity may have some effect over fluid dynamics of endolymph/perilymph, thus affecting the PTA thresholds. This may be unrelated to the age per se or the even long-term control of blood sugar.

Table 4 Association of retinal examination with demographic and biochemical profile of patients (n = 188)

Variables	Retinal examination				
	No change (n = 88) (%)	Mild change NPDR (n = 66)(%)	Moderate change NPDR (n = 14) (%)	Hypertensive retinopathy (n = 12) (%)	PDR (n = 8) (%)
Age (years)					
20–30	4 (4.5)	4 (6.1)	0 (0.0)	0 (0.0)	0 (0.0)
31–40	8 (9.1)	2 (3.0)	2 (14.3)	2 (16.7)	6 (75.0)
41–50	48 (54.5)	18 (27.3)	2 (14.3)	2 (16.7)	0 (0.0)
51–60	14 (15.9)	28 (42.4)	10 (71.4)	6 (50.0)	0 (0.0)
61–70	14 (15.9)	14 (21.2)	0 (0.0)	2 (16.7)	2 (25.0)
Sex					
Female	52 (59.1)	46 (69.7)	8 (57.1)	2 (16.7)	4 (50.0)
Male	36 (40.9)	20 (30.3)	6 (42.9)	10 (83.3)	4 (50.0)
Duration of DM (years)					
< 5	46 (52.3)	18 (27.3)	2 (14.3)	10 (83.3)	4 (50.0)
5–10	18 (20.5)	24 (36.4)	6 (42.9)	2 (16.7)	0 (0.0)
≥ 10	24 (27.3)	24 (36.4)	6 (42.9)	0 (0.0)	4 (50.0)
FBS (mg/dl)					
≤ 126	16 (18.2)	6 (9.1)	2 (14.3)	6 (50.0)	0 (0.0)
127–200	36 (40.9)	24 (36.4)	8 (57.1)	6 (50.0)	8 (100.0)
201–300	26 (29.5)	22 (33.3)	4 (28.6)	0 (0.0)	0 (0.0)
≥ 301	10 (11.4)	14 (21.2)	0 (0.0)	0 (0.0)	0 (0.0)
PP (mg/dl)					
≤ 200	24 (27.3)	10 (15.2)	8 (57.1)	8 (66.7)	0 (0.0)
> 200	64 (72.7)	56 (84.8)	6 (42.9)	4 (33.3)	8 (100.0)
HbA1c (%)					
< 7	18 (20.5)	10 (15.2)	2 (14.3)	4 (33.3)	0 (0.0)
7–8	20 (22.7)	6 (9.1)	6 (42.9)	6 (50.0)	0 (0.0)
8–9	10 (11.4)	20 (30.3)	4 (28.6)	2 (16.7)	2 (25.0)
≥ 10	40 (45.5)	30 (45.5)	2 (14.3)	0 (0.0)	6 (75.0)
S. cholesterol (mg/dl)					
≤ 180	70 (79.5)	46 (69.7)	8 (57.1)	10 (83.3)	2 (25.0)
> 180	18 (20.5)	20 (30.3)	6 (42.9)	2 (16.7)	6 (75.0)
Triglyceride (mg/dl)					
≤ 150	56 (63.6)	34 (51.5)	0 (0.0)	10 (83.3)	8 (100.0)
> 150	32 (36.4)	32 (48.5)	14 (100.0)	2 (16.7)	0 (0.0)

Although hyperlipidemia is associated with hearing impairment REF but this was not evident in our series.

The association of abnormal vestibular status with demographic and biochemical profile of patients is summarized in Table 9. On correlating, χ^2 test showed significant association of abnormal vestibular examination (normal/abnormal) with age ($\chi^2 = 19.76$, $p = 0.001$), sex ($\chi^2 = 5.59$, $p = 0.018$), duration of DM ($\chi^2 = 15.05$, $p = 0.001$), FBS ($\chi^2 = 8.68$, $p = 0.034$), HbA1c ($\chi^2 = 14.32$, $p = 0.003$). However, abnormal vestibular examination did not ($p > 0.05$) correlate well with PP, S. cholesterol and triglyceride.

Discussion

In our Indian population it is surprising to note that the prevalence of hearing impairment is less than 60% as opposed to vestibulopathy that is seen in more than 85% of patients. This suggests that diabetes mellitus is more likely to involve vestibular than cochlear functions. The vestibular compensation is probably responsible for minimal balance complaints unless the level of dysfunction is far too much. The retinal findings are more consistently associated with age, sex, diabetic variables and lipid

Table 5 Association of abnormal sensory examination with demographic and biochemical profile of patients (n = 188)

Variables	Sensory examination		χ^2 value	*p* value
	Absent (n = 106) (%)	Present (n = 82) (%)		
Age (years)				
20–30	8 (7.5)	0 (0.0)	21.52	< 0.001
31–40	10 (9.4)	10 (12.2)		
41–50	50 (47.2)	20 (24.4)		
51–60	22 (20.8)	36 (43.9)		
61–70	16 (15.1)	16 (19.5)		
Sex				
Female	58 (54.7)	54 (65.9)	2.38	0.123
Male	48 (45.3)	28 (34.1)		
Duration of DM (years)				
< 5	54 (50.9)	26 (31.7)	7.65	0.022
5–10	22 (20.8)	28 (34.1)		
≥ 10	30 (28.3)	28 (34.1)		
FBS (mg/dl)				
≤ 126	24 (22.6)	6 (7.3)	24.16	< 0.001
127–200	44 (41.5)	38 (46.3)		
201–300	34 (32.1)	18 (22.0)		
≥ 301	4 (3.8)	20 (24.4)		
PP (mg/dl)				
≤ 200	36 (34.0)	14 (17.1)	6.76	0.009
> 200	70 (66.0)	68 (82.9)		
HbA1c (%)				
< 7	22 (20.8)	12 (14.6)	1.42	0.702
7–8	20 (18.9)	18 (22.0)		
8–9	22 (20.8)	16 (19.5)		
≥ 10	42 (39.6)	36 (43.9)		
Albuminuria (mg/dl)				
< 30	54 (50.9)	32 (39.0)	5.87	0.053
30–300	36 (34.0)	26 (31.7)		
> 300	16 (15.1)	24 (29.3)		
S. cholesterol (mg/dl)				
≤ 180	86 (81.1)	50 (61.0)	9.39	0.002
> 180	20 (18.9)	32 (39.0)		
Triglyceride (mg/dl)				
≤ 150	64 (60.4)	44 (53.7)	0.85	0.355
> 150	42 (39.6)	38 (46.3)		

profile, and hence possibly reflects a better picture of degree of diabetic morbidity than auditory or vestibular involvement. However, the degree of audio vestibular involvement is also quite substantial in Indian population. Further research is needed to study other (olfactory, renal) morbidities in diabetes as well and also establish a correlation of all the morbidities amongst one another. The prevalence of raised serum creatinine of 42% in this series is worth noting despite the fact that 45% of patients showing normal albuminuria. This may reflect an ongoing initiation of stress on kidneys and a great potential of future nephropathy. A comparatively reduced number of elderly patients may have biased our observation towards middle age effects. Similarly, the predominance of cases with duration of DM of less than 5 years may have biased the conclusions against adequate reflections for longer duration of disease.

Table 6 Association of abnormal cranial examination with demographic and biochemical profile of patients (n = 188)

Variables	Cranial examination		χ^2 value	*p* value
	Absent (n = 170) (%)	Present (n = 18) (%)		
Age (years)				
20–30	8 (4.7)	0 (0.0)	4.53	0.340
31–40	16 (9.4)	4 (22.2)		
41–50	62 (36.5)	8 (44.4)		
51–60	54 (31.8)	4 (22.2)		
61–70	30 (17.6)	2 (11.1)		
Sex				
Female	102 (60.0)	10 (55.6)	0.13	0.715
Male	68 (40.0)	8 (44.4)		
Duration of DM (years)				
< 5	68 (40.0)	12 (66.7)	5.38	0.068
5–10	46 (27.1)	4 (22.2)		
≥ 10	56 (32.9)	2 (11.1)		
FBS (mg/dl)				
≤ 126	20 (11.8)	10 (55.6)	27.61	< 0.001
127–200	74 (43.5)	8 (44.4)		
201–300	52 (30.6)	0 (0.0)		
≥ 301	24 (14.1)	0 (0.0)		
PP (mg/dl)				
≤ 200	38 (22.4)	12 (66.7)	16.37	< 0.001
> 200	132 (77.6)	6 (33.3)		
HbA1c (%)				
< 7	32 (18.8)	2 (11.1)	2.59	0.459
7–8	32 (18.8)	6 (33.3)		
8–9	34 (20.0)	4 (22.2)		
≥ 10	72 (42.4)	6 (33.3)		
S. cholesterol (mg/dl)				
≤ 180	122 (71.8)	14 (77.8)	0.29	0.588
> 180	48 (28.2)	4 (22.2)		
Triglyceride (mg/dl)				
≤ 150	94 (55.3)	14 (77.8)	3.37	0.067
> 150	76 (44.7)	4 (22.2)		

It is evident from a review of otorhinolaryngology literature that the relationship between diabetes and sensorineural hearing loss is complex. Weng et al. noted that 44.8% diabetic subjects had profound hearing loss, while Rajendran et al. showed 73% diabetic patients having sensorineural hearing loss, Agarwal et al. 64.86%, Shafeeq et al. showed a prevalence of sensorineural hearing loss in 62.65% of type II diabetics [9–12]. The wide variation in prevalence of sensorineural hearing loss in diabetics may be due to difference in methodology including inclusion and exclusion criteria's. In our study sensorineural hearing loss was found to be typically bilateral symmetrical, progressive and gradual in onset, although asymmetry was noted in some of the patients. This result was comparable with the studies by Cullen, Kurien, Tay [13–15].

Axelsson et al. showed that the incidence of pure tone sensorineural hearing loss increases with age in patients with diabetes, even after correction for senile deafness. In the present study also incidence of sensorineural hearing loss was increased with the age of diabetics [16].

Celik et al. observed that as the duration of diabetes increased the incidence of hearing loss also increased [17]. Shafeeq et al. showed patients with more than 15 years of diabetic duration had increased prevalence of hearing loss 94.4% when compared to younger age groups [11].

Table 7 Association of abnormal cerebellar examination with demographic and biochemical profile of patients (n = 188)

Variables	Cerebellar examination		χ^2 value	*p* value
	Normal (n = 162) (%)	Abnormal (n = 26) (%)		
Age (years)				
20–30	8 (4.9)	0 (0.0)	4.76	0.312
31–40	16 (9.9)	4 (15.4)		
41–50	64 (39.5)	6 (23.1)		
51–60	48 (29.6)	10 (38.5)		
61–70	26 (16.0)	6 (23.1)		
Sex				
Female	96 (59.3)	16 (61.5)	0.05	0.826
Male	66 (40.7)	10 (38.5)		
Duration of DM (years)				
< 5	70 (43.2)	10 (38.5)	0.32	0.854
5–10	42 (25.9)	8 (30.8)		
≥ 10	50 (30.9)	8 (30.8)		
FBS (mg/dl)				
≤ 126	26 (16.0)	4 (15.4)	9.49	0.023
127–200	72 (44.4)	10 (38.5)		
201–300	48 (29.6)	4 (15.4)		
≥ 301	16 (9.9)	8 (30.8)		
PP (mg/dl)				
≤ 200	44 (27.2)	6 (23.1)	0.19	0.662
> 200	118 (72.8)	20 (76.9)		
HbA1c (%)				
< 7	28 (17.3)	6 (23.1)	4.21	0.239
7–8	36 (22.2)	2 (7.7)		
8–9	34 (21.0)	4 (15.4)		
≥ 10	64 (39.5)	14 (53.8)		
S. creatinine (mg/dl)				
≤ 1.5	98 (60.5)	12 (46.2)	1.90	0.168
> 1.5	64 (39.5)	14 (53.8)		
Albuminuria (mg/dl)				
< 30	76 (46.9)	10 (38.5)	11.96	0.003
30–300	58 (35.8)	4 (15.4)		
> 300	28 (17.3)	12 (46.2)		
S. cholesterol (mg/dl)				
≤ 180	120 (74.1)	16 (61.5)	1.76	0.185
> 180	42 (25.9)	10 (38.5)		
Triglyceride (mg/dl)				
≤ 150	94 (58.0)	14 (53.8)	0.16	0.689
> 150	68 (42.0)	12 (46.2)		

There was no significant association between HbA1c level and sensorineural hearing loss (*p* value 0.121) in our study. There was significant association between FBS level and sensorineural hearing loss (*p* value 0.024) 40.7% of mild sensorineural hearing loss had FBS > 200 mg/dl. Kurien et al. found that maximum incidence of sensorineural hearing loss 88% was present when blood glucose level was abnormal [15]. Control of blood sugar levels and their association with sensorineural hearing loss has been debated since long. These results were comparable to that with studies published by Cullen, Kurien, Tay [13–15, 18]. Farooq et al. found increase in FBG levels and

Table 8 Association of PTA with demographic and biochemical profile of patients (n = 188) biochemical profile of patients (n = 188)

Variables	PTA			χ^2 value	*p* value
	Normal (n = 78) (%)	Mild (n = 64) (%)	Moderate (n = 46) (%)		
Age (years)					
20–30	4 (5.1)	4 (6.3)	0 (0.0)	0.260	0.260
31–40	6 (7.7)	10 (15.6)	4 (8.7)		
41–50	34 (43.6)	20 (31.3)	16 (34.8)		
51–60	22 (28.2)	22 (34.4)	14 (30.4)		
61–70	12 (15.4)	8 (12.5)	12 (26.1)		
Sex					
Female	44 (56.4)	38 (59.4)	30 (65.2)	0.93	0.627
Male	34 (43.6)	26 (40.6)	16 (34.8)		
Duration of DM (years)	32 (41.0)	26 (40.6)	22 (47.8)	1.14	0.888
< 5	22 (28.2)	16 (25.0)	12 (26.1)		
5–10	24 (30.8)	22 (34.4)	12 (26.1)		
≥ 10					
FBS (mg/dl)					
≤ 126	14 (17.9)	12 (18.8)	4 (8.7)	14.51	0.024
127–200	30 (38.5)	26 (40.6)	26 (56.5)		
201–300	28 (35.9)	12 (18.8)	12 (26.1)		
≥ 301	6 (7.7)	14 (21.9)	4 (8.7)		
PP (mg/dl)					
≤ 200	8 (23.1)	22 (34.4)	10 (21.7)	3.03	0.219
> 200	60 (76.9)	42 (65.6)	36 (78.3)		
HbA1c (%)					
< 7	14 (17.9)	12 (18.8)	8 (17.4)	10.10	0.121
7–8	10 (12.8)	14 (21.9)	14 (30.4)		
8–9	14 (17.9)	12 (18.8)	12 (26.1)		
≥ 10	40 (51.3)	26 (40.6)	12 (26.1)		
S. cholesterol (mg/dl)	60 (76.9)	42 (65.6)	34 (73.9)	2.32	0.314
≤ 180	18 (23.1)	22 (34.4)	12 (26.1)		
> 180					
Triglyceride (mg/dl)					
≤ 150	46 (59.0)	32 (50.0)	30 (65.2)	2.66	0.264
> 150	32 (41.0)	32 (50.0)	16 (34.8)		

high HbA1c correlate with more severe sensorineural hearing loss [19]. This is in contrast to the study done by Mozaffari et al. in which there was no association between severity of sensorineural hearing loss and FBG [20].

Most of the studies revealed that there was no significant difference between severity of sensorineural hearing loss and also between the genders. One of the limitations is that it could not create a relationship with diabetes and sensorineural hearing loss because there was no control group of non-diabetic patients.

In our study there was a significant association between duration of diabetes and retinopathy (*p* value 0.001) and 50% cases of PDR had duration of diabetes more than 10 years. Fong et al. found prevalence of retinopathy 8% at 3 years, 25% at 5 years, 60% at 10 years and 80% at 15 years [21].

There was a significant association between NPDR and PDR with PP (p value < 0.001). 84.8% cases of mild NPDR and 100% cases of PDR had PP > 200 mg/dl. Yamini Singh et al. in a study found the mean FBS levels in diabetics without complications and diabetics with complications were $186.54 + 38.42$ and $86 + 10.91$ respectively when compared with controls [22]. These differences were statistically significant ($p < 0.001$) and consistent with the reports given by Akinloye et al. and Verma et al. [23, 24].

Table 9 Association of abnormal vestibular examination with demographic and biochemical profile of patients (n = 188)

Variables	Vestibular examination		χ^2 value	p value
	Normal (n = 26) (%)	Abnormal (n = 162) (%)		
Age (years)				
20–30	2 (7.7)	6 (3.7)	19.76	0.001
31–40	8 (30.8)	12 (7.4)		
41–50	12 (46.2)	58 (35.8)		
51–60	2 (7.7)	56 (34.6)		
61–70	2 (7.7)	30 (18.5)		
Sex				
Female	10 (38.5)	102 (63.0)	5.59	0.018
Male	16 (61.5)	60 (37.0)		
Duration of DM (years)				
< 5	20 (76.9)	60 (37.0)	15.05	0.001
5–10	4 (15.4)	46 (28.4)		
≥ 10	2 (7.7)	56 (34.6)		
FBS (mg/dl)				
≤ 126	6 (23.1)	24 (14.8)	8.68	0.034
127–200	16 (61.5)	66 (40.7)		
201–300	4 (15.4)	48 (29.6)		
≥ 301	0 (0.0)	24 (14.8)		
PP (mg/dl)				
≤ 200	10 (38.5)	40 (24.7)	2.18	0.140
> 200	16 (61.5)	122 (75.3)		
HbA1c (%)				
< 7	8 (30.8)	26 (16.0)	14.32	0.003
7–8	8 (30.8)	30 (18.5)		
8–9	8 (30.8)	30 (18.5)		
≥ 10	2 (7.7)	76 (46.9)		
S. cholesterol (mg/dl)				
≤ 180	16 (61.5)	120 (74.1)	1.76	0.185
> 180	10 (38.5)	42 (25.9)		
Triglyceride (mg/dl)				
≤ 150	14 (53.8)	94 (58.0)	0.16	0.689
> 150	12 (46.2)	68 (42.0)		

There was a significant association between retinopathy and HbA1c (p value < 0.001). Randomized control trials have confirmed the predictive value of poor glycaemic control compared with good control in determining the risk of nephropathy and retinopathy. DCCT showed 76% reduction in the rate of development of any retinopathy and an 80% reduction in progression of established retinopathy in patients with strict control of diabetes. Wisconsin epidemiological study of diabetic retinopathy showed a positive correlation between severity of retinopathy and high levels of HbA1c after 10 years of diabetes mellitus [25]. In the CURES Eye study for every 2% elevation of HbA1c the risk of diabetic retinopathy increases by a factor of 1.7. in the UKPDS the risk reduction in eye complications for every 1% decrease in HbA1c was 195 [26, 27].

There was a significant association between retinopathy and serum cholesterol ($p = 0.008$). 42.9% cases of moderate NPDR and 75% cases of PDR had serum cholesterol > 180 mg/dl. There was a significant association between retinopathy and serum triglyceride ($p < 0.001$). 48.5% cases of mild NPDR and 100% cases of moderate NPDR had triglyceride level > 150 mg/dl. Jyothi Idiculla et al. found dyslipidemia was present in 45.5% patients [28]. Diabetic retinopathy of any severity was found in 42.7% patients, with mild to moderate NPDR in 26.1%, severe NPDR in 8.5% and PDR in 8.2%. retinal hard exudate

formation was found to have statistically significant correlation with the presence of dyslipidemia ($p = 0.02$), increase total cholesterol ($p = 0.002$) and LDL levels ($p = 0.001$) and the correlation with triglyceride levels showed a trend towards significance ($p = 0.07$). On multivariate analysis increased total cholesterol showed a statistically significant association with increased retinal hard exudates formation ($p = 0.02$), whilst the other variables did not. Association of total cholesterol levels with retinopathy has been clearly demonstrated, especially in type II diabetes patients. However, Khizar Niazi et al. observed no association for any type of retinopathy. This could be explained by low mean levels of total cholesterol (< 200 mg/dl) of their patients studied, and could reflect the major role of genetic factors in various stages of diabetic eye diseases [29].

There was a significant association between abnormal sensory examination and FBS ($p < 0.001$). 44.4% cases with abnormal sensory examination and FBS > 200 mg/dl, PP ($p = 0.009$). 82.9% cases with abnormal sensory examination had PP > 200 mg/dl. Serum creatinine ($p < 0.001$). 61% cases with abnormal sensory examination had serum creatinine > 1.5 mg/dl and serum cholesterol ($p = 0.0021$). 39% cases with abnormal sensory examination had cholesterol > 180 mg/dl.

In the San Luis valley Diabetes study (SLVDS), a population based study of type II diabetic patients where the diagnosis of neuropathy was based on history and examination; there was an overall prevalence of 28%. In another study, the prevalence of DPN in type II diabetes among hospital patients in Spain was 26.7% [30]. However, the study involved assessment of only symptoms and signs for a diagnosis of DPN against a predetermined score. In another hospital based study, which analyzed the long term complications of newly diagnosed type II diabetes, the prevalence of neuropathy was 25.2%. The higher prevalence of neuropathy in the present study may be due to the selection of patients who were more symptomatic and tended to have more complications. A 45% prevalence of neuropathy in type II diabetes was reported from a population based sample from Rochester and 42% from a sample of 811 diabetes subjects drawn from 37 UK general practice.

There was a significant association between abnormal sensory examination and duration of diabetes ($p = 0.022$). In a Spanish study, the prevalence increased from 14% at under 5-year duration to 44% at duration of more than 30 years. In Mythili et al. study, the prevalence was 63% in those with duration less than 5 years to 90% in those with duration more than 10 years [31]. These data probably relate to a bias inherent in a hospital based study where the more severely affected diabetic patients are taken care of. HbA1c was significantly higher in those with neuropathy in the present study. The risk of developing DPN has been calculated to rise by approximately 10–15% for every 1% rise in HbA1c.

In our study 88.7% cases with abnormal vestibular examination were between 40 and 70 years of age which was found to be significant ($p = 0.001$), and 63% cases had duration of diabetes > 5 years which was found to be significant ($p = 0.001$). 44.4% cases of abnormal vestibular examination had FBS > 200 mg/dl which was significant ($p = 0.034$). In our study 65.4% cases with abnormal vestibular examination had HbA1c > 8% which was significant ($p = 0.003$). Prakash and Sumathi did a study to predict subclinical vestibulopathy at the earlier stage of diabetes so as to take necessary action to prevent the occurrence of vestibulopathy by having a good glycemic control [32, 33].

The similar study was done in 40 years and above to diagnose prevalence of vestibulopathy in diabetes mellitus by Klagenberg et al. [34] and Gawron et al. [35]. Klagenberg et al. and Karlin Fabianne in their study reported 60% prevalence of vestibular disease in diabetics in contrast to 42% of diabetics having vestibular disease in Sumathi et al. [35–37]. In their study, diabetic population of age group greater than 35 years, 51% had vestibulopathy which is significant, whereas in diabetics of age group less than 35 years it was only 26% similar to the study done by Li et al. [38].

The pathophysiology of vestibulopathy in diabetics is well documented by many studies. The cause may be due to the effects of complex glycation end products in the inner ear. So the degree of control of hyperglycaemia is essential to prevent the occurrence of vestibulopathy at the earlier stage.

In our study abnormal cerebellar examination did not correlate with duration of diabetes, HbA1c levels, PP. Rafaele et al. compared the results of static and dynamic balance tests and cerebellar function between control and diabetics groups, no significant difference was found [39].

Irshad et al. found out association between complicated diabetes and sensorineural hearing loss [40]. 29 patients of case and 6 patients of control showed up with sensorineural hearing loss. When cross tabulated their study group (cases and control) with sensorineural hearing loss the result came out significant ($p = 0.001$). Odds ratio was 5.81. It implies that sensorineural hearing loss is associated with diabetes with complications. In previous study, researchers compared 50 patients who had diabetes with 50 matched controls and found that hearing loss was significantly higher in cases than in control (94% vs. 18%, $p = 0.001$). In their study of association between presence of diabetic complications and sensorineural hearing loss, it was found a six fold increased risk of high frequency hearing loss

associated with both peripheral neuropathy and coronary artery disease.

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Compliance with Ethical Standards

Conflict of interest All the authors declare that they have no conflict of interest.

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