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ORIGINAL ARTICLE

TO ASSESS THE VESTIBULAR AND AUDITORY FUNCTIONS IN PATIENTS WITH DIABETIC NEPHROPATHY IN COMPARISON WITH PATIENTS OF UNCOMPLICATED DIABETES MELLITUS

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ABSTRACT: The inner ear dysfunction occurs in diabetes mellitus. The typical hearing loss described is a progressive, bilateral sensori-neural deafness of gradual onset which affects predominantly the higher frequencies. The causes are an angiopathy of the inner ear, neuronal degeneration, and electrolyte imbalance. Although the relationship between diabetes and vestibulo-cochlear dysfunction has been studied by various investigators, the exact relationship between various complications of diabetes and inner ear dysfunction requires further detailed study. Surprisingly the incidence of hearing loss in diabetes with complications is on the rise, which creates an interest to study the vestibulo-cochlear functions in diabetic nephropathy. This was a prospective study to assess the vestibular and auditory functions in patients with diabetic nephropathy in comparison with patients of uncomplicated diabetes mellitus. The aim of this study is to assess the vestibular and auditory functions in patients with diabetic nephropathy in comparison with patients of uncomplicated diabetes mellitus. This study includes 60 patients, 30 patients with uncomplicated diabetes mellitus were categorized in the group I and 30 patients with diabetic nephropathy were categorized in group II.

KEYWORDS: Diabetic micro angiopathy, Diabetic nephropathy, Vestibulo-cochlear dysfunction.

INTRODUCTION: Diabetes mellitus is a clinical syndrome characterized by hyperglycemia, due to deficiency or diminished effectiveness of insulin. The long standing metabolic derangement of diabetes mellitus is frequently associated with permanent and irreversible functional and structural changes in the cells of the body, those of the vascular system being particularly susceptible. The micro vascular complications of the diabetes include retinopathy, nephropathy and neuropathy. The duration of diabetes is an important determination of Micro vascular disease.[1] Diabetic nephropathy is currently defined by the presence of persistently positive urinary dipstick test for albumin in a person with diabetes in absence of other renal disease. Diabetic nephropathy is histologically characterized by glomerulosclerosis, hyalinosis of afferent and efferent glomerular arterioles and sometimes pyelonephritis.[2]

Mongensen and Co-workers (1983), have Proposed a Five Stage Progression in the Natural History of Diabetic Nephropathy.[3]:

- Stage I: Renal hypertrophy and hypertension.
- Stage II: Renal lesion without clinical signs.
- Stage III: Incipient Nephropathy.
- Stage IV: Clinical diabetic nephropathy.
- Stage V: End stage renal failure.

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The inner ear dysfunction occurs in diabetes mellitus. The typical hearing loss described is a progressive, bilateral sensori-neural deafness of gradual onset which affects predominantly the higher frequencies.[4] The causes are an angiopathy of the inner ear;[5] neuronal degeneration;[6] and electrolyte imbalance.[7]

Although the relationship between diabetes and vestibulo-cochlear dysfunction has been studied by various investigators, the exact relationship between various complications of diabetes and inner ear dysfunction requires further detailed study. Surprisingly the incidence of hearing loss in diabetes with complications is on the rise, which creates an interest to study the vestibulo-cochlear functions in diabetic nephropathy.

The Relation between Inner Ear and Kidney: Anatomical and physiological similarities are seen in inner ear and kidney. From electron microscopic studies, both the stria vascularis and the glomerulus of the kidney are epithelial structures intimately associated with vascular system. The renal tubules also have comparable ultrastructural features with the stria vascularis. The marginal cells of the stria vascularis contain a similar rich amount of mitochondria as the tubular cells of the proximal loop of Henle.[8]

Biochemical investigations revealed that oxygen consumption in both cell complexes is extremely high. The stria vascularis seems to exchange sodium for potassium by using on active ionic pump which pumps potassium into the endolymph. The tubular epithelium in the thick ascending limb of Henle also actively reabsorbs sodium. For the inner ear, the normal function of this pump is necessary to maintain the high endolymphatic potassium concentration and the high endolymphatic potential. For the kidney, this pump regulates the reabsorption of certain ions, especially those of sodium chloride and water.[8]

Pathlogy and Pathogenesis: Almost all possible complications of diabetes may present in diabetic nephropathy. Elevation of blood pressure is usually present from the early microalbuminuric stage of diabetic nephropathy. Hypertension in diabetic nephropathy is exquisitely volume sensitive and this becomes more apparent as renal failure progresses.[9] Neuropathy affects most patients with advanced diabetic nephropathy to variable extent. Micro vascular disease notably retinopathy and nephropathy is frequently seen in patients with long standing diabetes.

In 1961, Jorgensen studied temporal bones of thirteen diabetics complicated by nephropathy and retinopathy.[5] A large number of the diabetics showed appreciable periodic acid Schiff (PAS) – positive precipitates in the capillary walls, which stood out in some of the preparations as thick cables having walls 10-20 times thicker than normal. In mild cases, these changes were observed in a few capillaries, while in the more severe cases, they had spread to practically the entire capillary system of the stria. Costa (1967) also found PAS-positive thickenings in the capillary walls of the stria vascularis and the modiolus and in addition, thickening of the basement membrane and partial collapse of Reissner's membrane just above the stria vascularis.[10] Similarly Kovar (1973) reported thickening of the vessel walls in the stria vascularis and spiral ligament as well as hemorrhage in the modiolus, perilymph and endolymph.[11]

Makisima and Tanaka (1971) confirmed the thickening of the capillary walls of stria vascularis and commented that this led to a narrowing of the lumina.[6] They also found thickened walls and a narrow lumen in the internal auditory artery, hair cell loss, marked atrophy of the spiral ganglion in the basal and middle turns of the cochlea, and demyelination and beading of the eight nerve sheath.

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Prior to 1960 three main theories existed as to the pathogenesis of diabetic hearing impairment and vestibular dysfunction, namely a neuropathy, an angiopathy and a combination of the two (Taylor, 1978), whereas, after that date the vast majority of workers agree that the primary lesion is angiopathic (Jorgensen, 1961). An angiopathy of inner ear can lead to vestibule-cochlear dysfunction either directly or by interfering with the blood supply to the cochlea and vestibule by diminution of transport through thickened capillary walls and/or by reduction in blood flow through the narrowed vasculature, or by causing secondary degeneration in the eighth cranial nerve.[4,5]

METHODS: This was a prospective study to assess the vestibular and auditory functions in patients with diabetic nephropathy in comparison with patients of uncomplicated diabetes mellitus. This study includes 60 patients, 30 patients with diabetic nephropathy were categorized in the group I and 30 patients with uncomplicated diabetes mellitus were categorized in group II. This study was conducted during September 1990 to August 1992.

Group I included 30 uncomplicated and well controlled diabetic patients; all were between 25 and 55 years of age. A detailed history was taken. Systemic examination, local examination of the ears, nose and throat, routine investigations including blood sugar level (Fasting and post prandial), blood urea level, serum creatinine, serum electrolytes, audiometry and caloric tests were carried out in all patients.

Thirty patients of diabetic nephropathy were studied in group II, who we admitted under Nephrology and Medicine Unit at Miraj Medical Centre, Miraj; during the period of Sept. 1990 to Aug. 1992. All patients were diagnosed by Dept. of Nephrology and Dept. of Medicine and criteria for the diagnosis were persistently positive urinary dipstick for albumin, high blood urea level, high serum creatinine level, small sized kidneys and normal sized kidneys with distorted parenchymal texture on abdominal ultrasonography.

Patients with Diabetic Nephropathy were chosen for this study on the basis of the following Criteria:

1. Normal mental status and no evidence of central nervous system disease.
2. Conservatively managed patients with diabetic nephropathy.
3. Patients treated with dialysis and intensive diuretic therapy were excluded from this study.
4. Diabetic nephropathy with ketoacidosis or uraemic coma were excluded.
5. History of vestibulo-cochlear symptoms prior to onset of diabetes, family h/o hearing loss, h/o noise exposure, nephrotoxic drugs and patients with middle ear pathology were excluded from this study.

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OBSERVATIONS AND RESULTS:

VESTIBULAR RESPONSE TO CALORIC TEST								COCHLEAR RESPOSE TO AUDIOMETRY						
Sr. No.	Groups	Normal Function	D.P		C.P		Dys function In %	Sr. No.	Groups	Normal Cochlear Function	S-N Hearing loss at speech Frequency			Cochlear Dys-function In %
			Rt	Lt	Rt	Lt					Mild	Mode rate	Severe	
1	Uncomplicated Diabetic (Group I)	16	5	5	2	2	46.66	1	Uncomplicated Diabetic Group I	14	13	3	--	53.34%
2	Diabetic Nephropathy (Group II)	11	6	5	3	5	63.33	2	Diabetic Nephropathy Group II	10	7	10	3	66.66%

Table 1

AIR CONDUCTION THRESHOD RT. EAR					BONE CONDUCTIN THRESHOLD RT.EAR				
Frequency Hz	uncomplicated Diabetic Group I		Diabetic Nephropathy Group II		Frequency Hz	Uncomplicated Diabetic Group I		Diabetic Nephropathy Group II	
	Mean	S.D.	Mean	S.D.		Mean	S.D.	Mean	S.D.
250	9	4	16	10.4	250	7.1	4.5	11.6	6.9
500	13	8.2	21.3	11.4	500	13.0	8.2	20.6	11.9
1000	15.6	8.7	24.6	12.6	1000	15.6	8.3	24	12.4
2000	16	10.6	25.6	12.6	2000	17.0	9.7	25.5	14.6
4000	16.8	12.4	27.5	17.8	4000	17.8	12.7	27.8	17.8
6000	18.33	12.5	30.8	19					
8000	22.5	13.9	34.15	19.1					

Table 2

AIR CONDUCTION THRESHOD LT. EAR					BONE CONDUCTIN THRESHOLD LT. EAR				
Frequency Hz	Uncomplicated Diabetic Group I		Diabetic Nephropathy Group II		Frequency Hz	Uncomplicated Diabetic Group I		Diabetic Nephropathy Group II	
	Mean	S.D.	Mean	S.D.		mean	S.D.	Mean	S.D.
250	7.0	4.7	13.5	8	250	7.1	4.5	10.6	6.5
500	13.6	6.1	19.3	11.2	500	12.1	6.9	19.1	11.9
1000	15.1	8.8	21.4	13.8	1000	16	9.1	24.3	13.8
2000	18.3	10.4	24.5	14.1	2000	18.5	11.8	26	14
4000	20.0	12.2	26.8	17.2	4000	19.5	13.4	28.3	17.5
6000	22.1	13.7	29.6	18.1					
8000	25.3	15.3	32.6	20.4					

Table 3

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TABLE 4: The Correlation between Vestibulo-cochlear Dysfuntion And Duration Of Diabetes In Diabetic Nephropathy.

Duration	3-7 yrs.	8-12 yrs.	Duration	3-7 yrs.	8-12 yrs.
Normal Hearing	8	2	Normal vestibular function	9	2
Reduced Hearing	10	10	Vestibular dysfunction	9	10
Total	18	12	Total	18	12

Table 4

N = 30 N = 30.

P <0.001: Significant P <0.001: Significant.

Table 5: Mean Air Conduction Threshold According to Duration of Diabetes in Diabetic Nephropathy.

Duration of Diabetes	No	Mean Air Conduction Threshold						
		250	500	1000	2000	4000	6000	8000
3 to 7 years	18	12.2	17.5	19	20.9	24.3	26.6	28.7
8 to 12 years	12	18.9	24.5	28.9	31.2	31.4	35.5	40.8

Table 5

DISCUSSION: In the present study, vestibulo - cochlear functions of 30 uncomplicated diabetics and 30 diabetic nephropathy patients were assessed.

Table I shows, 46.66% patients of uncomplicated diabetic group showed vestibular dysfunction, in which 10 patients had directional preponderance (D. P.) and 4 had unilateral canal paresis (C. P.); 63.33% of diabetic nephropathy patients showed vestibular dysfunction, in which 11 patients had D.P. and 8 patients had unilateral C.P. these findings are consistent with A. Misra (1989), who found vestibular dysfunction in 66% of renal failure patients, of which 58% patients had canal paresis.[12] Where 53.34% of uncomplicated diabetic patients showed bilateral sensori-neural hearing loss, in which 13 patients had mild type and 3 patients had moderate hearing loss; 66.66% of diabetic nephropathy patients showed bilateral S-N hearing loss, in which 7 patients had mild type, 10 had moderate type and 3 patients had severe hearing loss.

Table 2 and 3 shows, both uncomplicated diabetic and diabetic nephropathy patients showed significant hearing loss, but latter group showed more hearing loss as compared to uncomplicated diabetics.

Table 4 and 5 showed the direct correlation between vestibulo-cochlear affection and duration of diabetes in diabetic nephropathy.

These results were more consistent with J. Zelenka (1965). In his study, about 75% patients of diabetes showed reduced hearing and vestibular dysfunction which was roughly proportional to the duration of diabetes.[13] I.G. Taylor (1978) found 15db hearing loss for at least one frequency in 63.2% of diabetic patients.[4] Taylor (1978), Jorgensen (1961), and Zelenka (1965), concluded that the primary lesion is angiopathy of the inner ear in diabetics.[4,5,13]

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CONCLUSION: Thirty patients of diabetic nephropathy, thirty patients of uncomplicated diabetes mellitus were assessed regarding their vestibular and auditory functions by using pure tone audiometry and caloric test.

The following Conclusions are drawn from our study:

1. 63.33% of diabetic nephropathy patients showed statistically significant vestibular dysfunction in which 11 patients had D.P. and 8 patients had unilateral canal paresis; 46.66% patients of uncomplicated diabetes mellitus showed vestibular dysfunction, in which 10 patients had D. P. and 5 had unilateral canal paresis.
2. 66.66% patients with diabetic nephropathy showed bilateral sensori-neural hearing loss, in which 7 patients had mild type, 10 had moderate type and 3 patients had severe hearing loss; 53.34% of uncomplicated diabetic patients.
3. Patients of diabetic nephropathy showed significant vestibular affection and moderate to severe hearing loss at higher frequencies as compared to uncomplicated diabetics.
4. There is a direct correlation between vestibulo-cochlear affection and duration of diabetes in diabetic nephropathy ($p< 0.001$).

At the end it is concluded that the vestibular and auditory dysfunctions are associated with diabetic nephropathy and are more common in patients with diabetic nephropathy than uncomplicated diabetes.

A larger group of patients of diabetic nephropathy with regular follow-up, a larger group of controls and modern equipments to detect objectively the auditory thresholds and vestibular functions, without the need of patients' co-operation, are essential for further more detailed study

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