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STUDY OF HEARING LOSS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

Objectives:
To study the hearing loss in patients with chronic kidney disease.

Materials and methods:
This case control study of 50 CKD patients and 50 age (±5) matched population was conducted in the Department of Ototolaryngology Head and Neck Surgery and Department of Internal medicine, B. P. Koirala Institute of Health Sciences, Dharan, between July 2007 to December 2008. Otological examination was carried out in all the subjects. Middle ear pathology and conductive hearing loss excluded. Pure tone audiometry was carried out on each case and control (patient/participant) in a sound-proof room. Data was collected and analyzed. The sensory neural hearing loss of the CKD patients was compared with the healthy controls.

Results:
We found sensory neural hearing loss in 46% of CKD patients and 43% in normal population. High frequency hearing loss was the commonest abnormality found and was present in 31% of CKD patients and 29% in control group. Middle frequency hearing loss was found in 8% of CKD patients and 8% in control group. Low frequency hearing loss was found in 7% of CKD patients and 6% in control group. Average hearing loss (≤ 2000Hz) was present in 8% of CKD patients and 7% in control group. (P-value >0.05)

Conclusion:
The hearing loss in patients with chronic kidney disease was higher than the age matched normal population though not statistically significant.

Key words: Chronic kidney disease, Pure tone audiometry, SNHL,

INTRODUCTION:

Many similarities exist between the nephron and the stria vascularies of the cochlea and hearing loss have been reported in patients with renal failure[1]. Although the gross anatomy of the kidney and cochlea differ greatly, there are many similarities at the ultra-structural level. Both contain epithelial structures in close contact with their vascular supply. Basement membranes are found in Bowman's capsule, the proximal renal tubule of the kidney and around the capillaries of the stria vascularies. In addition, basement membrane-lined intercellular channels exist in both the glomerulus and the stria. Carbonic anhydrase is present in both the stria and nephron. The hereditary condition Alport's syndrome comprises both renal pathology and progressive hearing loss. Furthermore it has been shown that there is an immunological connection between the kidney and inner ear in that antibodies raised against the nephron also deposit in the stria vascularis[1]. Various pharmacological agents act both on the inner ear and kidney. Aminoglycoside antibiotics can be both nephrotoxic and ototoxic[2]. In 2000, The National kidney foundation (NKF) kidney disease outcome quality initiative advisory board approved development of clinical practice guidelines to define "chronic kidney disease" and to classify stages in the progression of chronic kidney diseases. The work group defined chronic kidney disease to include conditions that affect the kidney, with the potential to cause either progressive loss in the kidney function or complications resulting from decreased kidney function.

The work group developed the following operational definition of chronic kidney disease[3].

1. Kidney damage for <3 months as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifest by either pathological abnormalities or markers of kidney damage, including abnormalities in the composition of the blood or urine, or abnormalities in imaging tests.
2. GFR<60 ml/min/1.73m^2 for >3 months, with or without kidney damage.

Hearing threshold is generally defined as the lowest sound pressure at which under specified condition a person gives at least 50% correct detection of response. Pure tone audiometry is done to measure the hearing threshold for certain standardized stimuli via air conduction and bone conduction routes. Audiometric hearing loss may be defined as an average loss of greater than 25 decibels (dB) for the frequencies 0.5, 1 and 2 kHz[2]. If bone conduction level is normal (within 20 db) with AB gap 20 db or more, then the deafness is said to be of conductive type. If bone conduction level is more than 20 db and AB gap is 15 db or less, then the deafness is said to be of sensory neural type. If bone conduction level is more than 20 db and AB gap is 20 db or more the deafness is called mixed type. The result of mean hearing loss is calculated at speech frequencies i.e. between 500, 1000, 2000 Hz[4]. Sensory neural hearing loss results from lesions of the cochlea especially the hair cells of organ of corti, eighth nerve or central auditory pathways. It may be present at birth (congenital) or start later in life. (acquired)[5]. Disorders of metabolism as well as endocrine disorders are reported to results in Sensorineural hearing loss including the diabetes mellitus, hypothyroidism, hyperlipoproteinemia, renal failure, hyperuricemia, acromegaly, Addison's diseases and pheochromocytoma[6]. In adult patients with end stage renal disease, reported incidence of SNHL is about 20-87% which is much higher than the general population. Early detection of hearing loss makes the rehabilitation possible at the early stage[7]. No studies have been done in the past to assess the hearing loss in patients with CKD in Nepal. The present study was done to explore the hearing status between the chronic kidney disease patients and age matched healthy controls.

MATERIALS AND METHOD:

This was a case control study conducted in the department of Ototolaryngology and Head &Neck surgery and department of Internal Medicine, B. P. Koirala Institute of Health Sciences, Dharan, between July 2007 to December 2008. Fifty consecutive CKD patients between age of 15 to 73 years without history of ear disease prior to CKD, without conductive hearing loss, without history of sensory neural hearing loss prior to development of CKD (due to other causes e.g. noise, infection) were included. Similarly fifty age(+/-5) matched normal population or surgical cases without any ear problems, any systemic illness related to chronic Kidney diseases or hearing loss and any other known risk factors for hearing loss were taken in the study. Patients/participants (case and control group) below five years were not included in this study because of their inabilities to interpret audiometry tones appropriately. Informed consent was taken from all participants. Ethical approval was taken from the ethical committee of the institute. Chronic kidney disease (CKD) diagnosed according to NKF/DQLI criteria. Detail clinical history and physical examination including otological assessment was carried out in all the subjects according to Performa. Patients/participant's occupational history was taken to exclude risk factors for hearing loss e.g. noise exposure. Birth and developmental history was taken to exclude congenital and other causes of acquired hearing loss before the development of CKD.

Drug history was taken, especially the ototoxic drugs. Past history of ear trauma and head injury was taken to rule out the prior hearing loss. Otological examination was carried out to exclude middle ear pathology. All the subjects had normal ears at otoscopy. Tunning fork tests (Rinne's, Weber's) were carried out with 512 Hz tuning fork which gave Rinne's positive in sensory neural hearing loss. Weber test was done to detect the better hearing cochlea. We found positive Rinne's test and Weber's test was centralized in all the patients/participants (case and control group). Pure tone audiometry was done on each patient/participant in a sound-proof room. Calibrated Italian diagnostic audiometer (Amploid-460) was chosen for study and tests performed by a trained audiometrician. The audiometric testing was done by a single person to ensure test-retest reliability. No patients had air bone gap in the audiometric testing. The results are documented as low (250- 500 Hz), middle (1000 -2000KHz), and high (4000 -8000Hz). Threshold for hearing was determined separately for each ear. We calculated average of two responses in cases of doubt. Hearing threshold of cases were compared with those of control group. The collected data was entered into Microsoft Excel Spreadsheet and was analyzed using SPSS version 11.5. Mean and standard deviation of variables in both Group 1 and Group 2 were calculated. Student 't' test was used for comparing cases with controls.

RESULTS:

Total of 100 subjects were included in our study.(Table1) Mean age of CKD patients in Group1 was 41.36 years (Range: 17-73 years). Mean age of Group- 2 consisting of healthy controls was 43.26 years (Range: 20–77 years). The two groups were similar with respect to age. (No significant difference) (P-value 0.625). There were 24 (57%) females and 26 (43%) males in Group1 while Group2 consisted of 22 (53%) females and 28 (47%) males. The two groups were similar with respect to sex with no significant difference (p-value 0.688). There were ten patients (20%) in stage 2, seven (14%) patients in stage 3, seven patients (14%) in stage 4, twenty-six patients (52%) in stage 5 of chronic kidney disease. The mean duration of chronic kidney diseases since detection was 27.98 months. (SD= 33.43) (range: 1-152 months). Out of 50 CKD patients; seven patients had diabetes mellitus; twelve had hypertension and fourteen had both diabetes mellitus and hypertension both as a comorbid illness. Fifteen patients didn't have any known comorbid illness. Out of 50 patients; twenty one patients had history of atenolol administration, ten patients had history of atenolol and frusemide administration, one patient had history of frusemide and amikacin administration and eighteen patients did not have any history of ototoxic drug administration. Two (4%) patients had history of renal transplantation. Twenty seven (54%) patients had history of dialysis and 23 (46%) patients did not have any history of dialysis. Sensory neural hearing loss was present in 46% of CKD patients (group1) and 43% of normal population (group 2). High frequency hearing loss was the commonest abnormality found and was found in 31% of CKD patients (group1) and 29% of normal healthy population (group 2). Middle frequency sensory neural hearing loss was found in 8% of CKD patients (group1) and 8% normal population (group 2). Low frequency hearing loss was found in 7% of CKD patients (group1) and 6 % normal population. (group 2). Average hearing loss (≤ 2,000Hz) was present in 8% of CKD patients (group1) and 7% normal population (group 2) (Table 6). Mean hearing loss in ≤2,000 Hz, 250-500 Hz, 1,000-2,000 Hz, 4,000-8,000Hz frequency range were 18.93±13.91db, 18.25±13.74dB, 18.75±14.11dB and 39.66±28.96dB in CKD patients(group-1) and 18.46±11.17dB, 16.85±11.07dB, 19.60±14.38dB and 28.27±28.03dB in control (group 2). (Table 7) Hearing loss in different audiometric frequencies in CKD patients (group 1) was higher than it was in control group. But the difference could not reach to a statistically significant level (P-value >0.05)

DISCUSSION:

Hearing loss has been observed in patients with chronic kidney disease. With this background, we conducted the present study to assess the hearing loss in patients with chronic kidney disease. There were fifty patients of CKD in our study. The patients were between 17 to 73 years of age. Mean age of patients was 41.36 years (SD ±16.37 years; Range 17-73 years). There were 24 (48%) females and 26 (52%) males in patient group. There was similar age and sex distribution of the patients. Patients/participants (case and control group) below five years were not included in our study. This study was similar in age and sex distribution to the study done by LP. Morton[9] et al and D. Gatland[8] et al. We had taken all the CKD patients irrespective of the hemodialysis and renal transplantation. Our aim here was to assess

Tab. 1: Age distribution

Variable	Category	Group 1 CKD patients	Group 2 Control	Total
Age(in years)	15-30	19	10	29
	31-45	8	12	20
	46-60	17	16	33
	61-80	6	12	18
Total		50	50	100

Tab. 2: Sensory neural hearing loss in ≤ 2,000Hz frequency range

Variable	Category	Group 1 CKD Patients	Group 2 Control	Total	Percentage%
Hearing Loss	0-25dB	42	43	85	85%
	26-40dB	6	5	11	11%
	41-55dB	1	1	2	2%
	56-70dB	0	1	1	1%
	71-90dB	0	0	0	0%
	>90dB	1	0	1	1%
Total		50	50	100	100%

Tab. 3: Sensory neural hearing loss in 250-500Hz frequency range

Variable	Category	Group1 CKD Patients	Group2 Control	Total	Percentage%
Hearing Loss	0-25dB	43	43	86	86%
	26-40dB	7	6	13	13%
	41-55dB	0	0	0	0%
	56-70dB	0	0	0	0%
	71-90dB	0	0	0	0%
	>90dB	0	0	0	0%
Total		50	50	100	100%

Tab. 4: Sensory neural hearing loss in 1,000-2,000Hz frequency range

Variable	Category	Group1 CKD Patients	Group 2 Control	Total	Percentage%
Hearing Loss	0-25dB	42	42	84	84%
	26-40dB	6	4	10	10%
	41-55dB	1	0	1	1%
	56-70dB	0	2	2	2%
	71-90dB	0	1	1	1%
	>90dB	1	1	2	2%
Total		50	50	100	100%

Tab. 5: Sensory neural hearing loss in 4,000-8,000Hz frequency range

Variable	Category	Group1 CKD Patients	Group 2 Control	Total	Percentage%
Hearing Loss	0-25dB	19	21	40	40%
	26-40dB	10	10	20	20%
	41-55dB	8	8	16	16%
	56-70dB	6	6	12	12%
	71-90dB	6	5	11	11%
	>90dB	1	0	1	1%
Total		50	50	100	100%

Tab. 6: Sensory neural hearing loss in CKD patients and normal population

Frequency	CKD Patients	Control
4000-8000Hz	31%	29%
1000-2000Hz	8%	8%
250-500Hz	7%	6%
Total	46%	43%
Mean hearing loss (<2000Hz)	8%	7%

Tab. 7: Comparison of mean of hearing loss in CKD Patients (Group-1) with control (Group- 2)

Frequency	CKD Patients (n=50)	Control (n=50)	P=value
≤ 2,000 Means	18.93	18.46	0.853
(N=22) SD	13.91	11.17	
250-500 Means	18.25	16.85	0.576
(N=8) SD	13.74	18.25	
1,000-2,000 Means	18.75	19.60	0.766
(N=8) SD	14.11	14.38	
4,000-8,000 Means	39.66	38.27	0.808
(N=8) SD	28.96	28.03	

the hearing loss in all the cases of CKD irrespective of treatment modalities. We had also not taken gender as a study parameter. Study done by Bains (2007) et al, showed no correlation of hearing loss with gender[12]. Ozturan O, Lam S (1998) showed that hearing was unaffected by HD[11]. In our study, there were ten patients (20%) in stage 2, seven (14%) patients in stage 3, seven patients (14%) in stage 4 and twenty six patients (52%) in stage 5 of chronic kidney disease. Thirty seven patients had various comorbid diseases (diabetes mellitus, hypertension and both diabetes mellitus and hypertension) apart from CKD. We found hearing loss in 46% of CKD patients and 43% in control. High frequency hearing loss was the commonest abnormality found in our study and it was 31% in CKD patients. Hearing loss in CKD patients was higher. The difference was not statistically significant. (P-value was >0.05). In our study, the mean of hearing loss in 250-500, 1,000-2,000, 4,000-8,000Hz was higher in CKD patients than the control. Study done by Bains (2007) et al, compared with healthy controls, chronic kidney disease patients showed a highly significant bilateral sensorineural hearing loss at all frequencies of 0.25 to 8.0 kHz, which was more marked in higher frequencies[12]. Adler D. et al (1990) studied terminal renal failure and hearing loss for the frequencies 0.5, 1, 2 and 4 kHz. They found that there was 36% of hearing loss in patients with CRF. After excluding the patients with etiological factors known to predispose to hearing loss (congenital hearing loss, middle ear disorders noise induced hearing loss and cochlear ototoxicity), Adler found it to be 10%. Morton et al, (1992), study showed a high incidence of sensorineural hearing loss in chronic renal failure. The cause of the hearing loss was not established[9]. Study of kingerman AB (1981) showed a high incidence of high frequency impairment which could not be attributed to age, noise exposure, ototoxicity or hereditary. An association between this high-frequency impairment and both the renal disease and its treatment was suggested. Clinically significant sensorineural hearing loss did not appear associated with non-genetic kidney disease. Hutchison and klod (1982) assessed a series of 15 patients under the age of 60 suffering from chronic renal failure. These patients were being treated by haemodialysis.They eliminated from their study any patient who was diabetic or in whom the cause of renal failure was considered to be congenital. Each patient was tested once when the effects of the renal failure were most severe and they were about to undergo dialysis. They tested using pure tone audiometry, acoustic reflex thresholds, reflex decay test, elctronystagmography and brainstem auditory evoked responces. They concluded that when ototoxic drugs, noise exposure, diabetes, congenital nephrites and age above 60 years are eliminated and although individual abnormalities will occur; chronic renal failure does not in itself produce a clinically significant hearing loss; neither does it produce an abnormality of the peripheral or central vestibular function that is clinically significant nor did it produce an abnormality within the brain stem that affects the auditory or vestibular brain stem function from the clinical stand point[13]. In the study done by Lasisi AO et al. (2007) sensorineural hearing loss was found in 67% of CRF and 32% of controls. The mean hearing threshold of CRF was 47.42dB, while that of controls was 35dB. Pearson correlation showed the difference was significant. Stavraulaki et al[14] results was also similar to our study. Sensorineural hearing loss of unknown etiology was found in 55.5% of renal patients mainly in the higher frequencies. Changes in PTA thresholds were not significantly different than those in the control group[14] (P>0.05). The cause of the hearing loss was not established. The difference in the prevalence of sensory neural hearing loss in previous studies and the present one may be related to patient selection, due to the effect of ototoxic drug administration, comorbid illness, hemodialysis and renal transplantation, which were variably excluded from previous studies. It therefore appears that sensory neural hearing loss of unknown etiology commonly present in patients with chronic kidney disease. In our study the number of patients studied was less and it was conducted in a hospital setup. Patients who had actively sought medical advice, in general, are likely to be more health conscious. Therefore the results obtained by us cannot be applied to the entire chronic kidney disease population. We, therefore recommend that further studies need to be done in our setup on large population of chronic kidney disease patients

CONCLUSION:

The hearing loss in patients with chronic kidney disease was higher than the age matched normal population but was not statistically significant.

REFERENCES:

1 Quick CA, Fish A, Brown C. The relationship between cochlea and kidney. Laryngoscope 1973; 83,1469-82
2. Bergstrom L, Jenkins P, Sando I, English GM. Hearing loss in renal diseases. Clinical and pathological studies. Ann Ottol 1973; 82; 55576
3. Levey AS, Coresh J, Balk E. practice guidelines for chronic kidney disease: evaluation, classification, and stratification. National Kidney Foundation; Ann. Intern. Med. 2003; July15; 139(2): 137-47
4. Khanna SM, Tonndorf J. Tympanic membrane vibration in cats studied by time averaged holography. Journal of the acoustic society of America; 1972;51:1904-1920
5. A. Fitzgerald OC. Examination of the ear. Alan G. Kerr; Scott-Brown's Otolaryngology, volume-3, 6th Edition, Butterworth Heinemann, oxford, 1997, pp15.
6. Lam Hoe Yeoh. Causes of hearing disorders. Alan G. Kerr. Scott-Brown's Otolaryngology, volume-2, 6th Edition, Butterworth Heinemann, Oxford, 1997 pp15.
7. ST. Esfahani, A. Madani, N. Ataei, AN. Tehrani, P. Mohseni and Z. Ghanbari. Sensory neural hearing loss in children with end stage renal disease. Acta Medica Iranica. 2004; 42(5): 375-378.
8. D. Gatland, B. Tucker, S. Chalstrey, M. Kenne, L Baker. Hearing loss in chronic renal failure- hearing threshold changes following haemodialysis. Journal of Royal Society Medicine, 1984:587–589.
9. L.P. Morton, L. Reynolds, R. Zent, B.L. Rayner. Hearing Thresholds in CAPD Patient. Clinical Otolaryngology and Allied Sciences. 2001 Jun;26(3):235-42
10. Lasisi AO, Salako BL, Kodiya MA, Amusat MA, Osisanya WP. Hearing threshold in patients with chronic renal failure. Saudi Medical Journal. 2007 May; 28(5):744-6.
11. Ozturan O, Lam S. The effect of hemodialysis on hearing using puretone audiometry and distortion-product otoacoustic emissions. Journal of Otorhinolaryngology 1998 Nov-Dec; 60(6):306-313.
12. Bains H, Chopra J, Sandhu B. Cochlear Function in Chronic Kidney Disease and Renal Transplantation. Transplantation Proceedings; Volume 39, Issue 5, Pages 1465 - 1468
13. Grahe K, Horund G, Z Hals oharenhailled 1924; 8; 375. Oda M, Preacido MC, Quick CA, Paperella MM. Labyrinthine pathology of chronic renal failure patients treated with haemodialysis and kidney transplantation. Laryngoscope; 1976; 84:1489.
14. Stavroulaki P, Nikolopoulos TP, Psarommatis I, Apostolopoulos NC. Hearing evaluation with distortion-product otoacoustic emissions in young patients undergoing haemodialysis. Otolaryngology and allied sciences. 2001 Jun; 26(3):235-42.