

EVALUATION OF AUDITORY FUNCTIONS IN CHRONIC KIDNEY DISEASE

Nitin Kumar¹, *Avneesh Kumar², Ramesh Azad³, Sanjay Vikrant⁴

Received : 15/07/2025

Received in revised form : 03/09/2025

Accepted : 22/09/2025

Keywords: CKD, HT, HL.

Corresponding Author:

Dr. Avneesh Kumar,
Email: dravi1314@gmail.com

DOI: 10.47009/jamp.2025.7.5.149

Source of Support: Nil,
Conflict of Interest: None declaredInt J Acad Med Pharm
2025; 7 (5); 773-778

ABSTRACT

Background: We aimed to evaluate the association between hearing impairment in chronic kidney disease (CKD) and to find correlation of blood urea nitrogen and serum creatinine level with hearing threshold and evoked responses. We also evaluated the effect of dialysis on hearing threshold and evoked responses. **Materials and Methods:** Total 80 subjects were enrolled in this study which includes 40 CKD patients and 40 controls. Evaluation of hearing was done using Pure tone audiometry, tympanometry, Brainstem evoked response audiometry and speech audiometry. Various biochemical parameters of CKD patients were recorded. Various audiological parameters were compared in both groups and there correlations with various biochemical parameters were studied. **Result:** In summary there is increased hearing thresholds in CKD patients but the values are within normal range. There is prolongation of wave 3 and wave 5 in CKD patients which is statistically significant (P value < 0.001) when compared to controls. There is increased hearing thresholds with increased duration of dialysis. There is also increased speech reception threshold and decreased speech discrimination score in CKD patients. **Conclusion:** At last we concluded that there is increased hearing threshold in CKD patients. Hearing thresholds were also increased with duration of dialysis but there is no correlation of hearing threshold with other biochemical parameters.

INTRODUCTION

Chronic kidney disease (CKD) is a public health problem. Patient of CKD progresses gradually from various stages of CKD to end stage renal disease (ESRD). CKD causes the dispersion of nitrogen metabolic products, water, and electrolytes; homeostasis imbalance; and dysfunction of the calcium-phosphorus metabolism; as well as acid base equilibrium accompanied by anemia and hypertension. ESRD pathophysiology and biochemical dysfunction appear to cause auditory pathway damage, both at the sensory organ and neuronal levels. ESRD patients undergoing dialysis treatment exhibit some degree of sensorineural hearing impairment.^[1-3] Ototoxic drugs and associated conditions of renal failure, such as hypertension, electrolyte imbalance,^[3,4] alterations in blood urea nitrogen,^[4] and lipids,^[2] and hemodialysis treatment (HT),^[5,6] are some of the factors outlined in the literature responsible for hearing loss. There are contrasting results as to whether HT may be a significant factor for hearing impairment.^[1,5,6] Sensorineural hearing impairment has been reported in chronic kidney disease (CKD) patients with a

prevalence of 20–40%. The etiopathogenetic mechanisms reported included osmotic alteration resulting in loss of hair cells, collapse of the endolymphatic space, edema and atrophy of specialized auditory cells and in some, complications of hemodialysis.^[7-15] Many anatomical, physiological, pharmacological, and pathological similarities exist between the nephron and stria vascularis of the cochlea, and there are many similarities at the ultrastructural level. Both contain epithelial structures in close contact with their vascular supply. In addition to this basement membrane-lined intercellular channels exist both in the glomerulus and the stria vascularis. Both the inner ear and kidney are involved in body fluid homeostasis, namely, active transport of fluid and electrolytes accomplished by stria vascularis and glomerulus, respectively, and therefore have epithelium containing a Na⁺-K⁺ ATPase, carbonic anhydrase.

There is a general agreement on the most common audiometric profile in CKD patients: it is a high frequency HL, usually starting beyond 2 kHz, sometimes notching at 6 kHz.^[7] The objectives of this study were to assess auditory function in patients of

CKD so we can detect degree of hearing loss in different stages of CKD.

MATERIALS AND METHODS

This case control study was carried out in tertiary care hospital Indira Gandhi medical college shimla by the department of otorhinolaryngology and department of nephrology, over a period of one year, including the patients of chronic kidney disease(Stage 3-5).

Methodology: This study was carried out over a period of one year. Various tests were done like Pure tone audiometry, Speech audiometry, Tympanometry and BERA in cases as well as controls. Following ethical approval from the ethical committee of IGMC Shimla patient were enrolled for study after taking informed written consent detailed History and clinical Examination, patient were subjected to audiological tests as per the proforma attached.

Inclusion Criteria

- CKD (stage 3-5)
- Age of patient 18-50 years
- Normal tympanic membrane
- Normal middle ear function
- Stable patients on hemodialysis

Exclusion Criteria

- Patients having conductive hearing loss
 - Hospitalization in previous one month
 - History of noise exposure e.g. occupational as working in a factory
 - History of hearing loss of unknown etiology
- The Hearing assessment was done in following manner
- History and general physical examination
 - Otoscopic examination
 - Pure tone audiometry
 - Tympanometry
 - Speech audiometry
 - Brainstem evoked response audiometry

Otosopic Examination: Detailed otoscopic examination was done to look for any pathology in the ear which could exclude the patient from study or for wax or other lesions which could interfere with pure tone audiometry.

The examination was carried out in a audiometry room which is acoustically treated

Pure Tone Audiometry: Pure-tone audiometry was performed using audiometer AC-40 (INTERACOUSTIC) using TDH-39 earphones. The

auditory thresholds of each ear at frequencies of 0.5,1,2,3,4,6,8 kHz was measured.

Tympanometry-Tympanometry was conducted using the AT- 235 (INTERACOUSTIC) at a probe tone frequency of 220 Hz. In impedance audiometry type of Tympanogram, the tympanic cavity pressure is measured.

Speech Audiometry: Speech audiometry was performed using the speech reception threshold (SRT) and speech discrimination score (SDS) .

Brainstem Evoked Response Audiometry: The ABRs were recorded by using ECG electrodes. A single-channel recording was obtained with brainstem evoked response audiometer (Labat Epic Plus) system. Stimuli was condensation clicks, presented at the rates of 11.7 millise. The standard stimulus presentation level was 80 dB nHL (Normal hearing level). If waves I, III, and V could not be discerned, a higher intensity (maximum of 95 dB nHL) was used. The bandpass filters were set at 150 to 3,000 Hz. The analysis time was set at 10 ms. Two trials of 2,000 clicks were recorded to ensure reproducibility of the traces. The right and left ears were stimulated separately, and proper masking was applied to the ear not being tested.

RESULTS

The study was undertaken in the department of otorhinolaryngology and department of nephrology, Indira Gandhi Medical College, Shimla. We enrolled a total of 80 subjects in our study with 40 subjects in each group. The mean age of cases (CKD) was 37.4±8.53 years and that of controls was 34.68±1.26 years. The groups were comparable in terms of age with p value of 0.128.

On comparing cases and controls, right ear, a statistically significant difference was observed in PTA levels (p value 0.001) [Table 1]. There was a significant difference in wave 3 (p value <0.001) and wave 5 (p value 0.001) [Table 1].

In left ear, there was statistically significant difference in terms of cases and controls in PTA with a p value of <0.001 [Table 1]. A statistically significant difference was also observed in absolute latency of wave 3 (p value 0.004), wave 5 (p value <0.001) and interpeak latency of wave 3-5 (p value 0.019) and wave 1-5 (p value 0.009) [Table 1].

Table 1: Comparison of PTA, Absolute Peak Latencies and Interpeak Latencies of Right And Left Ear- Between CKD Patients and Controls

	Right EAR			Left EAR		
PTA, latencies and interpeak latencies	Case (CKD) MEAN±SD	Control MEAN±SD	P Value	Case (CKD) MEAN±SD	Control MEAN±SD	P Value
PTA	18.06±9.29	12.57±3.62	0.001	21.38±15.47	12.05±3.32	<0.001
WAVE I	1.79±0.3	1.72±0.17	0.198	1.77±0.38	1.69±0.23	0.288
WAVE III	3.88±0.2	3.72±0.19	<0.001	3.86±0.3	3.68±0.24	0.004
WAVE V	5.8±0.32	5.6±0.3	0.001	5.82±0.37	5.5±0.26	<0.001
WAVE I-III	2.07±0.33	1.99±0.23	0.272	2.08±0.31	1.97±0.32	0.118
WAVE III-V	1.93±0.26	1.87±0.29	0.364	1.98±0.33	1.82±0.25	0.019
WAVE I-V	4.03±0.42	3.87±0.36	0.062	4.05±0.4	3.82±0.39	0.009

The mean±SD values of blood parameters have been shown in table 2. The mean Hb observed in cases was 8.02±2.63. Creatinine levels observed were 8.3±2.86 while blood urea nitrogen (BUN) levels

were 93.1±30.88. Sodium, potassium and chloride levels were 133.9±6.19, 4.28±0.82 and 98.2±8.75 respectively.

Table 2: Mean of Blood Biochemical Parameters in CKD Patients

Biochemical parameters	Mean	SD
Hb (g/dl)	8.02	2.63
BUN (mg/dl)	93.1	30.88
Creatinine (mg/dl)	8.3	2.86
Uric acid (mg/dl)	10.86	5.14
Sodium (meq/l)	133.9	6.19
Potassium (meq/l)	4.28	0.82
Chloride (meq/l)	98.2	8.75
Calcium (mg/dl)	7.84	1.16
Phosphorus (mg/dl)	8.11	2.6

There was positive relation of PTA, absolute latencies and interpeak latencies of right ear with blood urea nitrogen but none was statistically significantly associated [Table 3].

PTA, absolute latencies and interpeak latencies of left ear were compared with BUN [Table 3]. There was

no statistically significant association in any of the parameters with blood urea nitrogen but wave 1 and wave 3-5 were negatively associated with BUN with pearson correlation value of -0.042 and -0.092.

Table 3: Relationship of PTA, Absolute Latencies and Interpeak Latencies of Right And Left Ear With BUN

	Right ear		Left ear	
PTA, absolute latencies and interpeak latencies	Pearson correlation (r)	P value	Pearson correlation (r)	P value
PTA	0.237	0.142	0.193	0.232
WAVE I	0.110	0.498	-0.042	0.799
WAVE III	0.064	0.693	0.1	0.539
WAVE V	0.171	0.291	0.05	0.761
WAVE I-III	0.007	0.964	0.13	0.425
WAVE III-V	0.221	0.17	-0.092	0.571
WAVE I-V	0.077	0.638	0.055	0.734

On comparing serum creatinine with PTA, absolute latencies and interpeak latencies, it was observed that wave 3, wave 1-3 and wave 1-5 were negatively associated with serum creatinine with pearson correlation value of -0.023, -0.193 and -0.056 respectively [Table 4]. However, there was no statistically significant association of any parameter with serum creatinine.

PTA, absolute latencies and interpeak latencies when measured in left ear in cases, there was no statistically significant association of any of these with serum creatinine. Wave 1-3, wave 3-5 and wave 1-5 of left ear were negatively associated with serum creatinine [Table 4].

Table 4: Relationship of PTA, Absolute Latencies and Interpeak Latencies of Right And Left Ear With Serum Creatinine

	Right ear		Left ear	
PTA, absolute latencies and interpeak latencies	Pearson correlation (r)	P value	Pearson correlation (r)	P value
PTA	0.066	0.685	0.259	0.107
WAVE I	0.152	0.349	0.187	0.248
WAVE III	-0.023	0.888	0.136	0.404
WAVE V	0.07	0.667	0.008	0.96
WAVE I-III	-0.193	0.233	-0.114	0.485
WAVE III-V	0.1	0.538	-0.096	0.557
WAVE I-V	-0.056	0.729	-0.205	0.204

There was a total of 29 cases who were on dialysis, and out of them 26 were on dialysis for <1 year and rest of the 3 patients were on dialysis for 1-2 years. On evaluating right ear of cases it was observed that PTA was significantly more in those who were on dialysis for 1-2 year (27.73±16.33) in comparison to

those on dialysis for <1 year (16.7±6.57) with a p value of 0.027 [Table 5].

On evaluating left ear of cases who were on dialysis, it was observed that PTA values were statistically significantly higher in those who were on dialysis for 1-2 years with p value of 0.027 [Table 5].

Table 5: Duration Of Dialysis wise Distribution Of PTA And BERA in Right and Left Ear

	RIGHT EAR			LEFT EAR		
	Dialysis ≤1 year (mean±SD) (n=26)	Dialysis 1-2 year (mean±SD) (n=3)	p value	Dialysis ≤1 year (mean±SD) (n=26)	Dialysis 1-2 year (mean±SD) (n=3)	p value
PTA	16.7±6.57	27.73±16.33	0.027	16.7±6.57	27.73±16.33	0.027
WAVE I	1.86±0.3	1.74±0.42	0.52	1.8±0.36	2.04±0.37	0.289
WAVE III	3.88±0.19	3.88±0.23	0.974	3.84±0.3	4.03±0.41	0.329
WAVE V	5.8±0.29	5.65±0.44	0.248	5.86±0.35	5.86±0.59	0.974
WAVE I-III	1.99±0.32	2.14±0.25	0.459	2.03±0.29	1.98±0.04	0.79
WAVE III-V	1.97±0.27	1.77±0.21	0.232	2.04±0.38	1.82±0.2	0.342
WAVE I-V	4.01±0.41	3.91±0.19	0.678	4.05±0.43	3.81±0.24	0.354

The speech reception threshold in right ear was statistically significantly higher in cases in comparison to controls with p value of <0.001 but on left side the difference was not statistically significant. Speech discrimination score in right ear in cases was 90.18±4.15 and in controls the score was 94.4±4.78 [Table 6]. There was statistically

significant difference between the mean speech discrimination score of right ear in cases and controls with p value of <0.001. Similarly, in left ear also the speech discrimination score was statistically significantly higher in controls when compared to cases with p value of <0.001 [Table 6].

Table 6: Speech reception threshold and speech discrimination scores of both ears in cases and controls

	CKD	Control	P value
Speech reception threshold right	23.53±2.64	20.78±3.37	<0.001
Speech reception threshold left	21.13±3.07	19.88±3.08	0.073
Speech discrimination score right	90.18±4.15	94.4±4.78	<0.001
Speech discrimination score left	89.9±4.26	94.98±5.16	<0.001

DISCUSSION

The auditory system per se is not severely involved in CRF. However slight impairment in hearing level in addition to disturbance of vestibular function has been reported in CRF. These mild abnormalities have been ascribed to the direct action of uraemic toxins or to physiochemical changes induced by the CRF in the inner ear fluids.^[16-23]

Physiologically, the kidney and cochlea play similarly important roles in fluid and electrolyte regulation and may share a common antigenicity. These factors may explain the similar effects of medications and immunological factors on both organs and strongly supports a relationship between hearing disorders and CKD.^[21] Several etiological factors, including uremia, ototoxic medication, electrolyte disturbances and hemodialysis treatment, have been linked to hearing disorders in patients with renal failure. Alder et al,^[7] reported an inverse correlation between serum creatinine levels and sodium-potassium activated ATPase levels and suggested that the inhibition of this enzyme system might cause inner ear dysfunction in terminal patients with uremia.

In the study by Amali Adekwu et al,^[22] there is hearing loss present at speech frequencies as well as high frequencies Whereas the studies by Gatland,^[3] and Nikolopoulos,^[13] reported hearing loss to be mainly in the high frequencies which is comparable to our study in which there is also presence of hearing loss mainly at higher frequencies (6 khz and 8 khz).

The brainstem evoked response audiometry studied was considered to be superior over pure tone audiometry for determining the site of lesion and

conduction study so the wave pattern of study was compared in cases and controls.

The two most common complications in end stage CRF are encephalopathy and peripheral neuropathy, characterized by premature demyelination and axonal degeneration of neural fibers.

The present study indicates that patients of CKD have delayed BAER latencies of wave III and V. The results of prolonged BAER latencies are in accordance with previous studies,^[6,19,20] which showed delayed prolongation mostly of the later waves III, V and interpeak I-V. These results differ from the findings of Marsh et al,^[18] of a delayed wave I latency with a normal interpeak I-V latency in patients on hemodialysis. Based on the previous studies,^[19,20] which mapped the anatomical region represented by BAER waves, the present study as well as previous studies,^[6,19,20] may suggest that conduction along the pons and midbrain is more susceptible to the effect of uraemia. The study by Marsh et al may suggest that the acoustic nerve is more vulnerable to the effect of uraemia.

A comparison between controls and the CKD group showed that wave III,V absolute latency and III-V and I-V interpeak latencies were significantly prolonged. Pagani et al,^[9] (1993) noted the prolongation of ABR wave III and V latencies among patients with end stage CRF which is comparable to our study.

Effect of different blood biochemistry levels and duration of dialysis on hearing threshold and brainstem evoked response audiometry.

Electrolyte disturbances, particularly sodium, water imbalance, hypertension, Vitamin D deficiency, and elevated serum urea levels, are proposed mechanisms for hearing impairment in patients with CKD.^[3,9,13,14]

Defects in the cationic gradient of endolymphatic fluid can change hearing properties.^[7] There is a decreased conduction velocity in the sensory and motor units, with the sensory units being more compromised than the motor.

The hearing threshold were tested in CKD patients at different levels of blood urea nitrogen, serum creatinine, uric acid, sodium, potassium, chloride, calcium, phosphorus, Hb.

There is positive correlation of PTA, absolute latencies and interpeak latencies of right ear with blood urea nitrogen but none was statistically significantly associated. There is no correlation of PTA, absolute latencies and interpeak latencies with serum creatinine, uric acid, sodium, potassium, chloride, calcium, phosphorus and Hb which is in agreement with the results reported by Agarwal et al,^[16] and Reddy et al.^[11]

Samir et al. found no correlation between pure tone audiometry findings and otoacoustic emission (OAE) measures and serum electrolyte levels.^[17] Haider K. Saeed et al,^[10] also concluded no relation between the levels of blood urea, creatinine, serum potassium, serum calcium, and serum sodium and hearing loss.

There was a total of 29 cases who were on dialysis, and out of them 26 were on dialysis for <1 year and rest of the 3 patients were on dialysis for 1-2 years. On evaluating right ear of cases it was observed that PTA was significantly more in those who were on dialysis for 1-2 year (27.73 ± 16.33) in comparison to those on dialysis for <1 year (16.7 ± 6.57) with a p value of 0.027.

On left ear evaluation of cases, similar to right ear evaluation, PTA had statistically significant and positive association with duration of dialysis (pearson correlation 0.453; p value 0.014). The interpeak latency of wave 1-3, wave 3-5 and wave 1-5 were negatively associated with dialysis duration. A landmark study by Samir et al,^[17] provided evidence that haemodialysis per se might worsen hearing function.^[7] Subsequent studies by Nikolopoulos et al., Marsh et al., and Rossini et al. were unable, respectively, to arrive at a definite conclusion regarding the effect of haemodialysis on hearing in CKD.^[5,13,18] In contrast, Peyvandi et al,^[12] proposed in a recent study that prevalence and severity of hearing loss increases with duration of CKD and haemodialysis which is comparable with our study.

There is increased SRT in CKD patients in both ears but it is statistically significant in right ear only (p value <0.001) and there is decreased SDS in both ears in CKD group which is also statistically significant (p value < 0.001). Increased SRT in CKD patients may be due to uraemic effects which causes neural pathology. In contrast, Peyvandi et al,^[12] proposed in a recent study that SRT results, in almost all patients were equal to values that anticipated regarding PTA results. Most patients had good SDS in more than 78% of cases were more than 80.

CONCLUSION

CKD is now a common clinical problem. The disease is associated with much higher prevalence of hearing loss compared to general population.

There are multiple etiological factors responsible including dyselectrolytemia, diabetes, ototoxic drugs, hypertension and the effect of uraemia on nerve conduction dysfunction itself.

In this study, patients of CKD suffered from delayed neural conduction in the midbrain and pons as these patients have statistically significant prolongation of wave III and wave V. There is a positive correlation of blood urea nitrogen with PTA and BERA but it is statistically insignificant. There is increased hearing threshold with increased duration of dialysis, but it is statistically insignificant and it is within normal range. In CKD patients, there is increased speech reception threshold and decreased speech discrimination score which is statistically significant. Hearing impairment is associated with a significant impact on the physical, emotional and social well being of the patient. It is an invisible handicap that hampers the communication and interaction of the patients but many a times gets unrecognised by the health care practitioner.

This study strongly recommends a routine audiological evaluation for all patients of CKD. Apart from treating the hearing impairment in such patients and providing them rehabilitation, we also need to be cautious in the future and avoid the use of ototoxic drugs in these patients. These patients should avoid noise exposure as that may lead to worsening of auditory function.

REFERENCES

1. Bazzi C, Venturini CT, Pagani C, Arrigo G, D'Amico GD. Hearing loss in short and long term haemodialysed patients. *Nephrol Dial Transplant* 1995; 10: 1865-8. .
2. Johnson DW, Wathen RL, Mathog RH. Effects of hemodialysis on hearing threshold. *Otorhinolaryngol* 1976; 38: 129-39.
3. Gatland D, Tucker B, Chalstrey S, Keene M, Baker L. Hearing loss in chronic renal failure—hearing threshold changes following hemodialysis. *J R Soc Med* 1991; 84: 587-9.
4. Yassin A, Badry A, Fatthi A. The relationship between electrolyte balance and cochlear disturbances in cases of renal failure. *J Laryngol Otol* 1970; 84: 429-35.
5. Rossini M, Stefano D, Febbo A, Paolo D, Bascini M. Brainstem auditory responses (BAERs) in patients with chronic renal failure. *Electroencephalography Clin Neurophysiology*. 1984;57:507-14.
6. Hutchinson JC, Klodd DA. Electrophysiological analysis of auditory, vestibular and brainstem function in chronic renal failure. *Laryngoscope* 1982; 92: 833-43.
7. Adler D, Fiehn W, Ritz E. Inhibition of Na⁺,k⁺-stimulated ATPase in the cochlea of the guinea pig. A potential cause of disturbed inner ear function in terminal renal failure. *Acta Otolaryngol* 1980; 90: 55-60
8. Vilayur E, Gopinath B, Harris DC, Burlutsky G, McMahon CM, Mitchell P, et al. The association between reduced GFR and hearing loss: A cross-sectional population-based study. *Am J Kidney Dis* 2010;56:661-9
9. Pagani C, Bazzi C, Arrigo C, Venturini C, D'Amico G. Evoked potentials (VEPs and BAEs) in a large cohort of

- short- and long-term haemodialyzed patients. *Nephrol Dial Transplant*. 1993;8:1124–28.
10. Saeed HK, Al-Abbasi AM, Al-Maliki SK, Al-Asadi JN. Sensorineural hearing loss in patients with chronic renal failure on hemodialysis in Basrah, Iraq. *Tzu Chi Medical Journal* 2018; 30(4): 216–20
 11. Reddy EK, Surya Prakash DR, Rama Krishna MG et al. Proportion of hearing loss in chronic renal failure: Our experience. *Indian J Otol*. 2016;22:4–9.
 12. Peyvandi A, Roozbahany NA. Hearing loss in chronic renal failure patient undergoing hemodialysis. *Indian J Otolaryngol Head Neck Surg*. 2013; 65: 537–40.
 13. Nikolopoulos TP, Kandiloros DC, Segas JV, Nomicos PN, Ferekidis EA, Michelis KE et al. Auditory function in young patients with chronic renal failure. *Clin Otolaryngol Allied Sci*. 1997; 22(3): 222–5.
 14. Brookes GB. Vitamin D deficiency and deafness: 1984 update. *Am J Otol*. 1985; 6(1): 102–7.
 15. Quick CA, Fish A, Brown C. The relationship between cochlea and kidney. *Laryngoscope* 1973 ; 83(9): 1469–82 .
 16. Agarwal MK. A study of otorhinolaryngological manifestations in patients of chronic renal failure. *Indian J Otolaryngol Head Neck Surg*. 1997; 49(4): 316–20.
 17. Samir M, Riad H, Mahgoub M, Awad Z, Kamal N. Transient otoacoustic emissions in children with chronic renal failure. *Clin Otolaryngol Allied Sci*. 1998 ; 23(1): 87–90.
 18. Marsh JT, Brown WS, Wolcott D, Landsverk J, Nissenson AR. Electrophysiological indices of CNS function in hemodialysis and CAPD. *Kidney Int* 1986; 30(6): 957–63.
 19. Jewett DL: Volume conducted potentials in response to auditory stimuli as detected by averaging in the cat. *Electroencephalography clin Neurophysiol* 1970; 28: 609–18.
 20. Buchwald J Huang C-M. Far field acoustic response: Origins in the cat. *Science* 1975; 189: 382- 4
 21. Pirodda A, Cicero AF, Borghi C. Kidney disease and inner ear impairment: a simpler and closer pathogenic analogy? *Intern Emerg Med*. 2012; 7(Suppl 2): S93–5.
 22. Amali Adekwu, Basil C. Ezeanolue, Emmanuel I. Agaba. Prevalence of hearing loss in chronic kidney disease, stages 3-5 patients in a teaching hospital in Nigeria. *J Med Res Pract* 2012; 1(2): 74- 6
 23. Wigand, M.E., Meents, O., Kreusser, H. and Heidland, A. Cochleovestibular disturbance in renal failure and the influence of diuretics. *Audiology*, 1972, 11 (Suppl.): 31