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# The Prevalence of Hearing Loss in Dialysis Patients

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## Summary

**Background:** To define the prevalence of hearing loss in advanced stage chronic kidney disease (CKD) patients that undergo peritoneal dialysis or hemodialysis and to investigate the correlation of hearing loss to serum levels of urea and creatinine.

**Methods:** The prevalence of hearing loss was investigated in 79 CKD patients that undergo dialysis (hemo- or peritoneal) in comparison to 40 healthy controls. The participants were classified into three groups: Group 1 consisted of peritoneal dialysis patients, Group 2 was comprised of hemodialysis patients and Group 3 was the control group. Evaluation of hearing was performed using pure tone audiometry, while serum levels of urea and creatinine before and after the dialysis procedure were compared between the three groups and correlated to the degree of hearing loss.

**Results:** Number and percentage of cases affected by the bilateral, sensorineural hearing loss that is obvious at high frequencies were 9 (21.9%) in group 1; 16 (42.1%) in group 2; and 1 (2.5%) in group 3. Serum urea and creatinine levels were higher in Group 2 compared to Group 1 ( $p = 0.00$ ). Hearing levels in terms of air and bone conduction thresholds were significantly worse in Group 2 ( $p = 0.00$ ). Conclusion: Sensorineural hearing loss occurs more frequently in CKD patients that require dialysis than in normal controls, and hemodialysis patients are more likely to be affected than the peritoneal dialysis patients. Hearing levels must be closely monitored in dialysis patients.

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## 1. Introduction

Chronic kidney disease (CKD) is an increasing public health problem. Patients in advanced stages of CKD require renal transplantation or dialysis for survival. Dialysis has effects on the function of almost every system in the body. In the literature, sensorineural hearing loss (SNHL) which has more influence on high frequencies than low frequencies, has been diagnosed in CKD patients [1, 2].

Many anatomical, physiological, pharmacological and pathological similarities exist between the renal tubular cells and the stria vascularis of the cochlea at the ultra-structural level [1, 2, 3]. Both structures include epithelia in close communication with their vascular supply and the basement membranes. This is most evident at the capillary endothelium in the Bowman's capsule in the proximal renal tubule and the capillaries of the stria vascularis in the cochlea. Basement membrane-lined intercellular channels exist both in the renal tubular cells and the stria vascularis. Epithelial cells have microvilli containing numerous mitochondria and play an active role in the transport of fluid

and electrolytes with the help of Na<sup>+</sup>- K<sup>+</sup> ion pump and carbonic anhydrase enzyme [1].

In previous studies, prevalence of hearing loss (HL) was reported to be increased in renal failure and it was postulated that there was a correlation between HL and electrolyte abnormalities in dialysis patients which was speculated to be linked to a malfunction of the ionic membrane transport [2, 3, 4]. Risk factors for development of HL in dialysis patients were plasma viscosity, hypercalcaemia, haemoglobin level, serum levels of calcium, potassium, urea and creatinine [5, 6, 7]. Decreased hearing in patients undergoing hemodialysis (HD) was documented and attributed to osmotic changes and fluctuation of electrolytes [6, 8, 9].

The aim of the current study is to define the prevalence of hearing loss in CKD patients on hemodialysis (HD) and peritoneal dialysis (PD).

## 2. Materials and methods

This prospective, controlled, clinical study was performed on 119 subjects between March 2010 and January 2011 by Otorhinolaryngology and Nephrology departments of a tertiary care center after the approval was obtained from Institutional Review Board.

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Table I. Serum levels of urea and creatinine in the three groups.

	Group 1 (PD) (n=41)	Group 2 (HD) (n=38)	Group 3 (n=40)	z	P
Pre-dialysis serum urea (mg/dL)	69.4 ± 22.1	148.8 ± 33.8		-7.594	.000
Post-dialysis serum urea (mg/dL)	27.6 ± 8.2	49.9 ± 13.3		-7.158	.000
Control serum urea (mg/dL)			28.7 ± 9.1		
Pre-dialysis serum creatinine (mg/dL)	3.3 ± 0.8	9.3 ± 1.8		-7.662	.000
Post-dialysis serum creatinine (mg/dL)	1.4 ± 0.5	3.6 ± 0.5		-7.670	.000
Control serum creatinine (mg/dL)			0.8 ± 0.2		

Patients with a history of co-existing otological disease, ear or head trauma, ototoxic drug usage and acoustic trauma were excluded from the study. Lifelong exposure was questioned for drugs like aminoglycosides and cisplatin which have a permanent toxic effect on cochlea. Use of medications like erythromycin or nonsteroidal anti-inflammatory drugs that have a reversible, temporary toxic effect were considered to be significant in last 6 months. For history of acoustic trauma, history of engaging in noisy hobbies, and history of recreational firearm exposure were questioned. A history of occupational noise exposure was considered if the participants had a full-time job at which they spoke louder to be heard by another person two feet away; or having participated in any of the following activities like working as a pilot or crew member on an aircraft, working in an engine room aboard a ship or using grenades during military service [10].

The subjects were divided into 3 groups: Group 1- patients on continuous cyclic peritoneal dialysis (CCPD), Group 2- patients on hemodialysis (HD) and Group 3- healthy controls. An otological examination and tympanometry were carried out to exclude any external and middle ear pathology. Patients with normal findings on examination, tuning fork test, tympanogram and stapedial reflexes were included in the study. Pure tone audiometry was performed on each subject in a sound-proof room by a trained audiological technician using a recently calibrated diagnostic audiometer. In HD, audiological evaluations were performed at least 4 hours after the completion of treatment to allow for the equilibration of electrolytes. Also in PD, audiological evaluations were performed at least 4 hours after the completion of the first dialysis of day. Audiometric hearing loss was defined as an average loss of greater than 20 decibels (dB) for the frequencies 0.5, 1, 2, 4 and 8 kHz. Peripheral blood samples to assess the urea, creatinine and albumin were obtained for biochemical analysis just before and 4 hours after the dialysis in HD patients. And blood samples were taken just before and 4 hours after the first dialysis of day in PD patients

### 3. Statistical analysis

Statistical analysis was performed by using SPSS 17.0. Kruskal Wallis Test was used to evaluate the audiometric results. Independent t- test was performed to compare urea-creatinine levels. (two-sided,  $p = 0.000$ ). A value of  $P < 0.01$  was considered to be statistically significant.

### 4. Results

This study was performed on 119 subjects. Seventy-nine dialysis patients and 40 healthy controls were enrolled in to this study. Group 1 included 41 patients (24 males, 18 females; mean age:  $29.1 \pm 5.3$ ) on CCPD, Group 2 included 38 patients (18 males, 20 females; mean age:  $29.8 \pm 6.1$ ) on HD and Group 3 included 40 healthy controls (22 males, 18 females; mean age:  $28.8 \pm 5.4$ ). There was no difference between these groups in terms of age and gender. No comorbidities had been reported for cases involved in this study.

Patients on PD or HD show different degrees of SNHL. We have found that the hearing loss was bilateral and more obvious in high frequencies. The number and percentage of patients suffering from this type of hearing loss was 9 (21.9%), 16 (42.1%) and 1 (2.5%) in groups 1, 2 and 3 respectively. Levels of serum urea and creatinine were higher in Group 2 (HD) compared to Group 1 (PD) both before and after the dialysis ( $p = 0.00$ ) (Table I). Both air and bone conduction hearing levels were significantly higher (worse) in Group 2 than in Group 1 and Group 3 ( $p = 0.00$ ) (Table II). Serum albumin levels in PD and HD groups are  $3.2 \pm 0.45$  gr/dl and  $3.5 \pm 0.31$  gr/dl respectively. Kt/V was used to assess the adequacy of HD and PD. Kt/V values for PD and HD patients are  $1.29 \pm 0.19$  and  $1.31 \pm 0.17$  respectively.

### 5. Discussion

The cochlea and kidney function in a similar way for active transport of fluid and electrolytes accomplished in the stria vascularis and the renal tubular cells [11, 12, 13, 14]. This similarity may account for the nephrotoxic and ototoxic effects of some medications and immunological pathologies of the two organs (ear and kidney).

Use of ototoxic drugs, electrolyte disturbances, hypertension, vitamin D deficiency being on HD have been correlated to the HL in CKD [2, 4, 15, 16, 17, 18, 19]. Adler *et al.* reported not only a distinctive reduction of Na<sup>+</sup>- K<sup>+</sup> activated ATPase in the inner ear of uremic guinea pigs, but also an inverse relationship between serum creatinine levels and Na<sup>+</sup>- K<sup>+</sup> activated ATPase [20]. Since Na<sup>+</sup>- K<sup>+</sup> activated ATPase in the cochlea is responsible for sustaining cationic gradients; they claimed that inhibition of this enzyme may be involved in the pathogenesis of inner ear dysfunction among uremic patients [20].

Table II. Pure tone audiometry results in the three groups.

	Bone conduction hearing level(dB)		Air conduction hearing level(dB)	
	Right	Left	Right	Left
Group 1 (PD) (n=41)	15.9 ± 9.9	15.9 ± 9.9	16.3 ± 10.4	16.3 ± 10.4
Group 2 (HD) (n=38)	24.3 ± 10.1	24.3 ± 10.0	24.8 ± 10.3	24.5 ± 9.8
Group 3 (control) (n=40)	7.4 ± 3.9	7.4 ± 3.8	7.8 ± 4.3	7.8 ± 4.2
p	.000	.000	.000	.000

We found that bilateral, SNHL particularly involving high frequencies is more likely to occur in patients on HD and PD as compared to normal controls. This finding is consistent with Komsuoglu *et al.*'s findings derived from 36 CKD patients and 10 controls [21]. In PD patients, serum urea and creatinine levels were found to be lower than patients on HD in our study. These findings suggest neural involvement in the auditory dysfunction associated with renal failure and hemodialysis [21]. Similarly, Baldini *et al.* reported that there were significant alterations in the auditory brainstem responses (ABRs) of uremic group from patients on chronic dialysis in comparison to the control group [22]. This finding was explained by the combined degeneration of central and peripheral nervous pathways in chronic uremic patients. Samir *et al.* investigated 34 children with CKD (27 on HD, 7 on conservative treatment) with evoked otoacoustic emissions (TEOAEs) [23]. They found that four children had conductive type hearing loss (11.8%), while five children (14.7%) had bilateral moderate to severe SNHL. Pure tone audiometry seems to be a practical and effective method for the assessment of hearing loss in patients on PD [22]. Cochlear dysfunction was significant only at low frequency levels and no correlation could be established with serum levels of urea, creatinine, sodium (Na+), or potassium (K+) [6]. Gatland *et al.* [24] reported low and high frequency HL, with the middle speech frequencies spared in adults with CKD. Hyponatremia, in addition to uremia, was thought to be responsible for HL. Improvement of low frequency HL after the dialysis reminds the likelihood role of osmotic disequilibrium on endolymphatic hydrops in the pathological process [24]. Morton *et al.* reported that prevalence of ultra high frequency hearing loss (10–20 kHz) in adult CKD patients on continuous ambulatory PD (CAPD) was as high as 50% [25].

Mancini *et al.* investigated the prevalence of SNHL in three groups of children with CKD [26]. The prevalence of HL was 29% in children receiving conservative treatment, 28% in children on HD and 47% in children with renal transplants. Nikolopoulos *et al.* [27] measured the auditory function among 46 children with renal failure; 22 with pre-end stage renal disease, 15 on HD, and 9 on CAPD. The overall rate of HL was found to be 41.3%, It was consistent with conductive type of HL and ototoxicity in 11% of children and no etiology could be determined for the remaining 30.4%. Hearing loss in dialysis may be related to the changes in equilibrium of electrolytes. It is more apparent in high frequencies, however low and mid-

dle frequencies may also be involved in up to 30% of the ears [28, 29]. Forty-seven percent of children on HD had HL, as compared to 32% in the pre-end stage CKD group and 0% in the CAPD group. It is noteworthy that children on HD suffer HL more often than those receiving conservative treatment [22].

Risvi and Holmes *et al.* [1] reported that the deterioration of hearing in CKD patients occurred in parallel to the progression of the disease and necessity for dialysis. Anatomic alterations such as collapse of the endolymphatic system, edema and atrophy occurred in the labyrinth and these changes were attributed to the osmotic disequilibrium induced by HD or PD [1]. Kusakari *et al.* [30] found that inner ear dysfunction was not associated with hematocrit, serum levels of blood urea nitrogen (BUN) and creatinine or the duration of HD. Johnson *et al.* [6] reported that fluctuations of hearing were not correlated with levels of BUN, creatinine, K+, Na+, Ca++ and glucose. Jorgenson *et al.* [31] claimed that HL was not correlated to alterations in levels of creatinine, K+, Na+, Ca++, glucose, BUN, serum lipids, blood pressure and body weight.

Intraperitoneal aminoglycoside and vancomycin administration in CAPD patients may cause low- and high-frequency hearing loss, and this ototoxic effect may be another explanation for the development of HL in patients on PD [32].

Histopathological changes of inner ear of patients on HD vary from the mild loss of outer hair cells and spiral ganglion cells to the complete absence of the organ of Corti. But histopathological changes of inner ear of patients on PD has not been investigated in the literature. In addition, blue stained concretions in the stria vascularis and/or in the vestibular receptors of transplanted patients had been demonstrated [28, 33]. However, the pattern and significance of these histopathological changes needs to be further investigated to identify the impact of dialysis on the auditory pathways.

We think that the importance of HL in patients on HD may be attributed to the neurotoxicity of urea and creatinine, since patients on HD have higher levels of urea and creatinine both before and after the dialysis. Second, the osmotic disequilibrium may occur more abruptly and in a broader range in these patients, that may lead to hydrops and histopathological changes more apparently in patients on HD than patients on PD.

Enrollment of patients on CCPD makes our study unique, since the HL in patients on PD was mostly in-

vestigated in patients on CAPD in the literature [6, 30]. However, we had investigated serum levels of urea, creatinine and albumin; lack of analysis of hematocrit, sodium and potassium appears to be a limitation of our study.

In summary, patients that undergo HD and PD seem to have an increased prevalence of SNHL at high frequencies. HD patients are more vulnerable to HL than patients on PD, which may be due to the higher levels of serum urea and creatinine [27] or abrupt osmotic changes leading to deleterious histopathological changes in cochlea. Hearing levels must be closely monitored in dialysis patients and further studies are necessary both to understand the underlying pathophysiological mechanism and to elucidate the association between HL and types of dialysis.

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