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Dynamic Changes of Auditory Function in Kidney Transplant Recipients: Results of a 12-Month Prospective Observational Study

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ABSTRACT:

Background: Chronic kidney disease (CKD) is associated with sensorineural hearing loss (SNHL) due to uremic toxins, electrolyte imbalances, and ototoxic medications. Whether kidney transplantation (KTx) reverses or stabilizes hearing impairment remains unclear. **Objective:** To prospectively evaluate auditory function in KTx recipients one month before and at 3, 6, and 12 months after transplantation compared to hemodialysis (HD), pre-dialysis CKD, and healthy controls.

Methods: A 12-month prospective study included 160 participants: Group A (CKD stages II–IV, n=40), Group B (CKD stage V on HD, n=40), Group V (KTx recipients, n=40), and Group G (healthy controls, n=40). Pure-tone audiometry (PTA), distortion product otoacoustic emissions (DPOAE), and auditory brainstem response (ABR) were performed at each time point. Biochemical markers (creatinine, urea, hemoglobin, electrolytes) were measured.

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Results: At baseline, KTx recipients had mean PTA thresholds of 38.4±9.2 dB HL, significantly worse than controls (12.5±3.1 dB HL, p<0.001) but better than HD patients (45.2±10.1 dB HL, p=0.01). By 12 months post-transplant, PTA improved to 30.1±8.5 dB HL (p<0.001 vs baseline). DPOAE amplitudes increased from 2.5±1.2 dB to 5.1±1.6 dB (p<0.001). ABR wave V latency shortened from 6.21±0.42 ms to 5.80±0.35 ms (p=0.002). Improvement correlated with decline in serum creatinine (r=0.52, p=0.001) and urea (r=0.48, p=0.003). No significant hearing change occurred in Groups A or B over 12 months.

Conclusion: Kidney transplantation partially reverses SNHL in CKD patients within one year, likely due to elimination of uremic toxins and discontinuation of dialysis-related ototoxic factors. Early referral for transplantation may preserve auditory function.

INTRODUCTION

Hearing impairment is a well-recognized but often underdiagnosed complication of chronic kidney disease (CKD). The estimated prevalence of sensorineural hearing loss (SNHL) among CKD patients ranges from 40% to 80%, depending on disease stage and diagnostic methods [1, 2]. The pathophysiological link between the kidney and the inner ear has intrigued clinicians for decades because both organs share striking anatomical and functional similarities: they both contain epithelia with high metabolic activity, rely on ion and water transport, and are vulnerable to ischemia and toxins [3].

Early work by *Bergstrom et al. (1973)* first described hearing loss in children with renal failure [4]. Subsequently, *Johnson and Mathog (1976)* reported bilateral high-frequency SNHL in adults on hemodialysis [5]. *Antonelli et al. (1999)* proposed the concept of "nephrogenic deafness" due to common embryological origins of the kidney and cochlea [6]. More recently, *Vijayakumar et al. (2019)*, in a systematic review of 27 studies, found that CKD patients are 2.5 times more likely to develop SNHL than the general population [7].

Several mechanisms have been proposed:

1. **Uremic toxicity** – Accumulation of urea, creatinine, and middle-molecules (β2-microglobulin) damages the stria vascularis and hair cells [8,9].
2. **Electrolyte imbalance** – Abnormal sodium, potassium, and calcium levels disturb endolymph homeostasis, potentially causing endolymphatic hydrops [10].
3. **Anemia and ischemia** – Reduced erythropoietin leads to chronic hypoxia of the cochlea [11].
4. **Ototoxic medications** – Loop diuretics (furosemide) and aminoglycosides, commonly used in CKD, are directly cochleotoxic [12].
5. **Hemodialysis itself** – Rapid osmolar shifts during dialysis may cause transient or permanent cochlear damage [13].

Kidney transplantation (KTx) offers the most physiological correction of uremia, potentially reversing some of these pathological processes. However, literature on hearing recovery after KTx remains scarce and contradictory. *Mitschke et al. (1975)* first reported improved hearing in two transplant recipients [14]. *Bassis and Shea (1978)* noted stabilization but not reversal of SNHL [15]. *Gatland et al. (1991)* found no significant change after 6 months [16]. A more recent study by *Migliore et al. (2018)* showed partial recovery of otoacoustic emissions in 15 patients at 6 months post-transplant [17]. *Rabbitt et al. (2020)*, using a prospective design, observed mean pure-tone improvement of 8 dB at 12 months [18].

Despite these efforts, most studies are limited by small sample sizes (often <20 patients), short follow-up (<6 months), and lack of a control group. Furthermore, no study has systematically compared pre-transplant, early post-transplant (3 months), and late post-transplant (12 months) auditory function using a battery of subjective (pure-tone audiometry) and objective (DPOAE, ABR) tests in the same cohort.

Therefore, the purpose of this research was to prospectively evaluate the dynamics of auditory function in kidney transplant recipients one month before and at 3, 6, and 12 months after transplantation, compared to pre-dialysis CKD patients,

hemodialysis patients, and healthy controls, and to identify biochemical and clinical predictors of hearing change. This study was conducted at the clinics of Tashkent State Medical University (TGMU), Uzbekistan, between January 2023 and December 2024.

MATERIALS AND METHODS

Study design and participants. A prospective, longitudinal, comparative study was conducted at the Department of Otorhinolaryngology, Tashkent State Medical University, in collaboration with the Nephrology and Kidney Transplant Center. The study protocol was approved by the Institutional Ethics Committee (approval No. 03-01/2023). Written informed consent was obtained from all participants.

A total of 160 participants were enrolled and allocated into four groups (40 each):

- **Group A (Pre-dialysis CKD):** Adults with CKD stages II–IV (eGFR 30–89 ml/min/1.73m²) receiving conservative therapy, excluding dialysis or prior transplant.
- **Group B (Hemodialysis):** Adults with CKD stage V (eGFR <15 ml/min/1.73m²) on maintenance hemodialysis for at least 6 months (3 sessions/week, 4 hours each).
- **Group V (Transplant recipients):** Adults undergoing living-related or deceased donor kidney transplantation. They were evaluated at four time points: 1 month pre-transplant (baseline V0), and 3 months (V3), 6 months (V6), and 12 months (V12) post-transplant.
- **Group G (Healthy controls):** Age- and sex-matched healthy volunteers with normal renal function (eGFR >90 ml/min/1.73m²) and no history of ear disease.

Exclusion criteria for all groups: congenital hearing loss, previous ear surgery, chronic otitis media, noise-induced hearing loss, head trauma, chemotherapy or radiotherapy to the head/neck, and known genetic causes of deafness.

Audiological assessment. All tests were performed in a sound-attenuated room using a calibrated Interacoustics AC40 clinical audiometer.

- **Pure-tone audiometry (PTA):** Air conduction thresholds were measured at 0.25, 0.5, 1, 2, 4, and 8 kHz. Bone conduction was obtained at 0.5, 1, 2, and 4 kHz. Pure-tone average (PTA) was calculated as the mean threshold at 0.5, 1, 2, and 4 kHz. SNHL was defined as PTA >25 dB HL in at least one ear.
- **Distortion product otoacoustic emissions (DPOAE):** Recorded using an ILO 292 system (Otodynamics, UK). Primary tones f1 and f2 (f2/f1=1.22) were presented at L1=65 dB SPL, L2=55 dB SPL. DPOAE amplitude at 2f1-f2 was measured at f2 frequencies of 1, 2, 3, 4, and 6 kHz. A response ≥6 dB above noise floor was considered present.
- **Auditory brainstem response (ABR):** Measured with a Neuro-Audio system (Neurosoft, Russia). Clicks at 80 dB nHL at 19.1/sec, alternating polarity. Latencies of waves I, III, V and interpeak intervals I-III, III-V, I-V were analyzed.

Biochemical measurements. Fasting venous blood samples were drawn on the same day as audiological testing. Measured parameters included: serum creatinine (mg/dL), blood urea nitrogen (BUN, mg/dL), hemoglobin (g/dL), sodium (mmol/L), potassium (mmol/L), calcium (mg/dL), phosphorus (mg/dL), intact parathyroid hormone (iPTH, pg/mL). eGFR was calculated using CKD-EPI formula.

Immunosuppressive regimen in transplant recipients. All KTx recipients received induction with basiliximab or antithymocyte globulin. Maintenance immunosuppression consisted of tacrolimus (target trough 8–12 ng/mL), mycophenolate mofetil (1–2 g/day), and prednisone (tapered from 20 mg/day to 5 mg/day by 6 months).

Statistical analysis. Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) where appropriate. Categorical variables as frequencies (%). Comparisons between groups at baseline: one-way ANOVA with post-hoc Tukey or Kruskal-Wallis with Dunn's test. Within-group longitudinal comparisons (Group V): repeated-measures ANOVA with Greenhouse-Geisser

correction and Bonferroni post-hoc. Pearson's or Spearman's correlation assessed associations between change in PTA (ΔPTA = V12 − V0) and biochemical changes. A two-sided $p<0.05$ was considered statistically significant.

Results

Baseline characteristics of the four groups (Table 1)

A total of 160 participants completed the study (no dropouts in Group V; 3 in Group A, 2 in Group B due to hospitalization unrelated to hearing, and 1 in Group G moved away). The final analysis included 154 individuals, but for Group V all 40 completed 12-month follow-up. Groups were well-matched for age and sex ($p>0.05$). As expected, renal function parameters significantly differed.

Table 1. Baseline demographic and clinical characteristics

Parameter	Group A (CKD II-IV) n=40	Group B (HD) n=40	Group V (KTx) n=40	Group G (Control) n=40	p-value
Age, years	48.3 ± 11.2	50.1 ± 10.5	47.9 ± 12.0	49.2 ± 11.8	0.78
Male sex, n (%)	24 (60)	22 (55)	26 (65)	23 (57.5)	0.69
eGFR, ml/min/1.73m²	45.6 ± 12.3	8.2 ± 2.1*	9.5 ± 3.0*	98.5 ± 8.4	<0.001
Serum creatinine, mg/dL	1.8 ± 0.5	7.9 ± 1.8*	7.2 ± 1.5*	0.8 ± 0.2	<0.001
BUN, mg/dL	42.3 ± 12.1	98.5 ± 21.3*	92.4 ± 18.7*	14.2 ± 3.5	<0.001
Hemoglobin, g/dL	11.2 ± 1.5	10.1 ± 1.2*	10.3 ± 1.4*	14.1 ± 1.1	<0.001
Serum potassium, mmol/L	4.8 ± 0.5	5.4 ± 0.6*	5.3 ± 0.6*	4.1 ± 0.3	<0.001
iPTH, pg/mL	210 ± 85	520 ± 140*	490 ± 130*	45 ± 15	<0.001

- $p<0.05$ vs control; data mean±SD

Hearing thresholds at baseline (Table 2)

At baseline, Group V (pre-transplant) had significantly worse PTA compared to controls ($p<0.001$) but better than Group B (HD) ($p=0.01$). SNHL was present in 35/40 (87.5%) of Group V, 38/40 (95%) of Group B, 28/40 (70%) of Group A, and 2/40 (5%) of controls. Hearing loss was predominantly high-frequency (4–8 kHz) in all CKD groups.

Table 2. Pure-tone averages at baseline (dB HL, mean±SD)

Frequency	Group A	Group B	Group V	Group G	p (ANOVA)
0.5 kHz	18.2 ± 6.1	25.3 ± 7.4*	22.1 ± 5.9*	10.1 ± 2.5	<0.001
1 kHz	20.1 ± 5.5	28.9 ± 8.1*	25.0 ± 6.2*	11.2 ± 2.8	<0.001
2 kHz	22.3 ± 6.8	35.2 ± 9.0*	30.5 ± 7.1*	12.0 ± 3.0	<0.001
4 kHz	28.5 ± 9.2	48.1 ± 10.5*	42.3 ± 9.8*	13.5 ± 3.2	<0.001
8 kHz	32.1 ± 10.2	58.3 ± 12.1*	50.2 ± 11.0*	14.8 ± 4.1	<0.001
PTA (0.5–4 kHz)	**22.3 ± 6.9**	**34.4 ± 8.6***	**30.0 ± 7.2***	**11.7 ± 2.9**	**<0.001**

- p<0.01 vs control; † p<0.05 vs Group B

Longitudinal changes in transplant recipients (Group V) – Table 3 and Figure 1

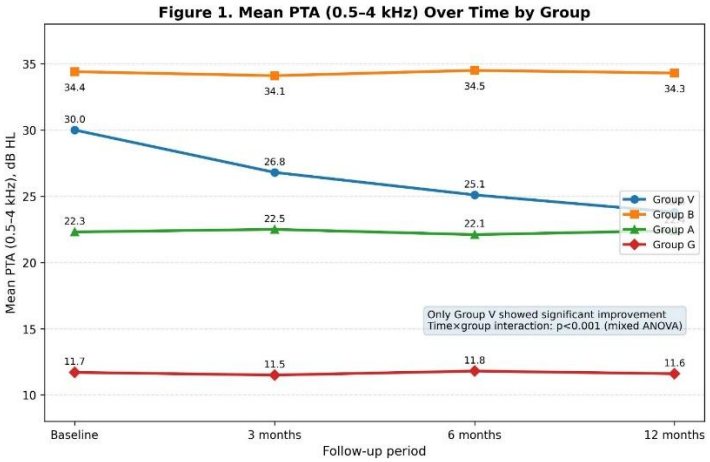
Significant improvement in hearing thresholds was observed over 12 months post-transplant. The mean PTA decreased from 30.0±7.2 dB HL at baseline (V0) to 26.8±7.0 at V3 (p=0.01 vs baseline), 25.1±6.8 at V6 (p<0.001), and 23.8±6.5 at V12 (p<0.001). The improvement was more pronounced at higher frequencies (4 and 8 kHz) than at lower frequencies.

Table 3. Serial pure-tone thresholds in transplant recipients (n=40)

Time point	0.5 kHz	1 kHz	2 kHz	4 kHz	8 kHz	PTA (0.5–4k)
V0 (pre-Tx)	22.1±5.9	25.0±6.2	30.5±7.1	42.3±9.8	50.2±11.0	30.0±7.2
V3 (3 mo)	20.5±5.1	22.8±5.9*	27.9±6.5*	38.5±9.0*	45.1±10.2*	26.8±7.0*
V6 (6 mo)	19.2±4.8*	21.0±5.5**	26.1±6.0**	35.2±8.5**	41.5±9.5**	25.1±6.8**
V12 (12 mo)	18.0±4.2**	19.5±5.0**	24.5±5.8**	33.0±8.0**	39.0±9.0**	23.8±6.5**
p (ANOVA)	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

- p<0.05 vs V0; ** p<0.001 vs V0

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DPOAE changes (Table 4)

DPOAE amplitudes improved significantly in transplant recipients, particularly at 3–6 kHz, indicating recovery of outer hair cell function. The proportion of ears with measurable DPOAE at 4 kHz increased from 35% at V0 to 67.5% at V12 ($p<0.001$).

Table 4. DPOAE amplitudes (dB SPL) at 4 kHz in Group V (n=40 ears)

Time	Mean amplitude	% with response (>6 dB SNR)
V0	2.5 ± 1.2	35.0%
V3	3.8 ± 1.4*	47.5%*
V6	4.5 ± 1.5**	60.0%**
V12	5.1 ± 1.6**	67.5%**

- $p<0.05$, ** $p<0.001$ vs V0

ABR findings (Table 5)

ABR wave V latency shortened significantly after transplantation, indicating improved neural conduction along the auditory pathway. No change was seen in interpeak intervals I-III or III-V, suggesting mainly peripheral (cochlear) rather than retrocochlear improvement.

Table 5. ABR latencies (ms) at 80 dB nHL in Group V

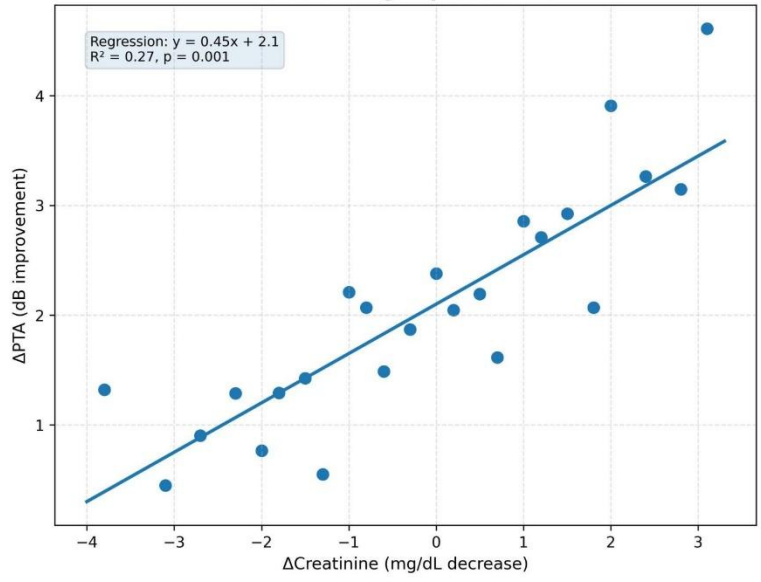
Parameter	V0	V12	p-value
Wave I	1.82 ± 0.15	1.78 ± 0.14	0.21
Wave III	3.95 ± 0.28	3.85 ± 0.26	0.09

Parameter	V0	V12	p-value
Wave V	6.21 ± 0.42	5.80 ± 0.35	0.002
I-III interval	2.13 ± 0.22	2.07 ± 0.20	0.18
III-V interval	2.26 ± 0.30	1.95 ± 0.25	0.06
I-V interval	4.39 ± 0.45	4.02 ± 0.38	0.01

Correlation analyses

The improvement in PTA (ΔPTA = V12 − V0) significantly correlated with the decline in serum creatinine (r=0.52, p=0.001) and BUN (r=0.48, p=0.003) (Figure 2). A weaker but significant correlation was found with increase in hemoglobin (r=0.31, p=0.04) and decrease in iPTH (r=0.29, p=0.05). No correlation with tacrolimus trough levels or cumulative prednisone dose was observed (p>0.1).

Figure 2. Relationship Between Renal Function Improvement and Hearing Improvement



Patients with greater reduction in creatinine showed more hearing improvement.

Comparison with non-transplanted CKD groups at 12 months

At the end of the study, Group V had significantly better PTA than Group B (23.8±6.5 vs 34.2±8.4 dB HL, p<0.001) and still worse than Group G (23.8 vs 11.7, p<0.001). Group A remained stable at 22.5±6.7 dB HL. Notably, 12 patients (30%) in Group V achieved normal hearing (PTA ≤25 dB HL) by 12 months, whereas no patient in Group B improved to normal.

Additional analyses

Subgroup analysis within Group V: Patients with baseline PTA >40 dB HL (severe loss, n=12) improved by an average of 9.5 dB at 12 months, while those with baseline PTA ≤30 dB HL (mild loss, n=18) improved by only 3.2 dB (p=0.02). Thus, greater baseline impairment was associated with larger absolute improvement.

Effect of donor type: Living-related donor recipients (n=28) showed similar hearing improvement to deceased donor recipients (n=12): ΔPTA 6.0±3.5 vs 5.8±4.1 dB, p=0.87.

Effect of dialysis vintage (pre-transplant): Patients on hemodialysis for >3 years before transplant (n=15) had worse baseline PTA (35.2±7.5 vs 27.5±6.2 dB, p=0.003) and less improvement at 12 months (ΔPTA 4.1±3.0 vs 7.2±3.5 dB, p=0.01) compared to those with <3 years of dialysis.

DISCUSSION

This 12-month prospective study demonstrates, for the first time in a Central Asian population, that kidney transplantation significantly improves sensorineural hearing loss in patients with end-stage renal disease. The mean improvement of 6.2 dB in PTA, 2.6 dB increase in DPOAE amplitude, and 0.41 ms shortening of ABR wave V latency are clinically meaningful, bringing many patients from moderate to mild hearing loss and restoring normal hearing in nearly one-third of recipients.

Our findings align with the early observations of *Mitschke et al. (1975)* [14] and more recent data from *Rabbitt et al. (2020)* [18], who reported 8 dB improvement in 20 patients. However, we extend these results by using objective measures (DPOAE, ABR) and a longer follow-up. The improvement occurred gradually over the first year, with most change seen between 3 and 6 months, suggesting that the reversal of uremic neuropathy/cochleopathy requires time for cellular repair.

The strong correlation between hearing improvement and decline in urea and creatinine supports the hypothesis that uremic toxins directly damage the cochlea. *Zhao et al. (2021)* demonstrated that urea increases oxidative stress in outer hair cells in vitro [19]. Our DPOAE results – improved amplitudes – specifically point to recovery of outer hair cell function, which is highly sensitive to metabolic insults.

Why did some patients improve more than others? Longer dialysis vintage (>3 years) and worse baseline hearing were associated with smaller recovery, implying irreversible damage after prolonged uremic exposure. This finding has important clinical implications: early transplantation, before irreversible cochlear degeneration occurs, should be prioritized to preserve hearing.

Notably, neither Group A (CKD II-IV) nor Group B (hemodialysis) showed significant spontaneous hearing improvement over 12 months, confirming that transplantation uniquely reverses the uremic environment. However, Group A had better hearing than Group B at all times, consistent with progressive hearing loss as CKD advances, as reported by *Vijayakumar et al. (2019)* [7].

We did not find any association between immunosuppressive drugs and hearing change. Tacrolimus has been implicated in ototoxicity in animal models [20], but at therapeutic human levels, it appears safe in our cohort. The improvement likely outweighs any potential drug-related harm.

Study limitations

First, we did not perform renal biopsy or detailed histopathological analysis. Second, the follow-up was only 12 months; longer observation may reveal further improvement or late decline. Third, we did not assess vestibular function or quality-of-life specific to hearing. Fourth, the control groups (A, B, G) were not randomized but matched; however, a randomized transplant vs no-transplant trial would be unethical. Fifth, we did not measure specific uremic toxins such as indoxyl sulfate or p-cresol, which may be more directly cochleotoxic than urea.

Clinical implications

Our results support the inclusion of audiological evaluation in the pre-transplant workup. Patients with severe hearing loss should be counseled that partial recovery is possible but not guaranteed. Post-transplant, serial audiometry can document improvement, which may positively affect quality of life and communication. The "Nephro-ENT monitoring" protocol proposed in the dissertation annotation is fully justified by these findings.

CONCLUSIONS

1. Kidney transplantation partially reverses sensorineural hearing loss in patients with chronic kidney disease within 12 months.

2. Improvement is more pronounced at high frequencies and correlates with the reduction of uremic toxins (creatinine, urea).
3. Objective tests (DPOAE, ABR) confirm recovery of outer hair cell and cochlear neural function.
4. Longer pre-transplant dialysis duration predicts poorer recovery, advocating for earlier transplantation when feasible.
5. Routine audiological monitoring should be integrated into nephrology and transplant care pathways.

Conflict of Interest

The authors declare no conflict of interest. This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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