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Original Research Article

Hearing Assessment in Patients with Chronic Renal Failure

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Abstract

Background: Chronic kidney disease has been associated with multisystemic complications including auditory dysfunction.

Objective: To evaluate hearing status in patients with chronic renal failure and assess its association with CKD in the absence of underlying co-morbidities.

Methods: Seventy patients with chronic renal failure underwent pure tone audiometry and biochemical evaluation.

Results: Hearing impairment was observed in 28.6% of participants, with unilateral involvement more common than bilateral. Mild to moderate sensorineural patterns predominated. Ototoxic drug exposure was limited, suggesting intrinsic renal contribution to cochlear dysfunction.

Conclusion: Chronic renal failure is associated with measurable hearing impairment independent of common systemic co-morbidities. Routine audiological screening may aid in early detection and intervention.

Keywords: Chronic Kidney Disease; Sensorineural Hearing Loss; Pure Tone Audiometry; Renal Dysfunction.

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Introduction

Chronic kidney disease (CKD) and chronic renal failure (CRF) represent a significant global health burden characterized by progressive loss of renal function and multisystemic complications. Among the lesser known but clinically important complications of CKD is auditory dysfunction. Hearing involves complex interactions between cochlear, neural, vascular, and metabolic processes, many of which can be affected by metabolic derangements seen in CKD [1].

The pathophysiology of CKD includes uremia, electrolyte imbalance, chronic inflammation, and vascular changes, all of which have been hypothesized to contribute to cochlear damage and sensorineural hearing loss (SNHL) [2]. In addition, prolonged exposure to uremic toxins and oxidative stress in CKD patients can accelerate aging of the auditory system and compromise both peripheral and central auditory pathways [3].

Several clinical studies have documented higher prevalence of hearing impairment in CKD populations compared to the general population, with sensorineural patterns being the most commonly reported form of hearing loss [4]. A cross-sectional study by Fufore et al. demonstrated that hearing loss was significantly more prevalent in patients with CKD, with the pattern predominantly showing deficits at higher frequencies, suggesting cochlear involvement [5].

Hearing impairment in this population can affect communication, quality of life, and social functioning; however, it often remains under-recognized due to overlapping symptoms with uremic and metabolic complaints. Early auditory evaluation is therefore critical to detect subclinical hearing deficits in CKD patients even in the absence of other co-morbidities [6].

Audiological assessments in CKD patients commonly include pure tone audiometry (PTA), which has been useful in identifying both low and high frequency hearing thresholds. Research has shown that PTA findings correlate with the severity and duration of renal dysfunction, implying a dose-response relationship between renal impairment and auditory decline [7]. Additionally, studies using otoacoustic emissions and auditory brainstem response testing have suggested cochlear and retrocochlear involvement in CKD, potentially

linked to metabolic imbalance and microvascular changes [8,9]. These objective measures can be particularly valuable in detecting early auditory dysfunction before it becomes clinically apparent.

While hearing loss in CKD has been documented in multiple studies, heterogeneous methodologies, varying inclusion criteria, and the presence of co-morbidities such as diabetes and hypertension have made it difficult to isolate the specific association between renal dysfunction and hearing impairment. There is a need for research that evaluates hearing status in CKD patients in the absence of underlying co-morbidities in order to clarify the direct impact of renal failure on auditory function. Understanding this association will not only improve patient care but also inform screening protocols aimed at early intervention and hearing preservation in this vulnerable population.

This study aims to evaluate hearing status in patients with chronic renal failure and investigate the association between hearing loss and CKD in the absence of other systemic co-morbidities.

Material and Methods

This hospital-based cross-sectional observational study was conducted in the Department of Otorhinolaryngology in collaboration with the Department of Nephrology at a tertiary care teaching hospital to evaluate hearing status in patients diagnosed with chronic renal failure (CRF). The study was carried out over a defined study period after obtaining prior approval from the Institutional Ethics Committee. The study protocol adhered to the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants after explaining the objectives, procedures, potential benefits, and possible risks involved in the study. Confidentiality of patient data was strictly maintained throughout the research process.

A total of 70 patients diagnosed with chronic kidney disease (CKD) stage 3 and above, based on estimated glomerular filtration rate (eGFR) criteria, were included in the study. Patients aged between 18 and 60 years were recruited from the Nephrology outpatient and inpatient departments. Only patients with chronic renal failure in the absence of underlying co-morbidities such as diabetes mellitus, hypertension, thyroid disorders, chronic otitis media, and history of ototoxic drug intake, noise exposure, previous ear surgery, or congenital hearing loss were included in order to isolate the direct association between CKD and hearing impairment. Patients with active middle ear pathology, history of acoustic trauma, or neurological disorders affecting hearing were excluded from the study.

A detailed clinical history was obtained from each participant, including duration of renal disease, treatment modality (conservative management or hemodialysis), and any subjective auditory complaints such as difficulty in hearing, tinnitus, or vertigo. General physical examination and systemic examination were performed to confirm eligibility. Otoscopic examination was conducted to rule out external or middle ear pathology.

All patients underwent comprehensive audiological evaluation in a sound-treated room using a calibrated pure tone audiometer. Pure tone audiometry (PTA) was performed to determine air conduction and bone conduction thresholds at frequencies ranging from 250 Hz to 8000 Hz. Hearing thresholds were recorded in decibels hearing level (dB HL). The average hearing threshold was calculated using the pure tone average at speech frequencies (500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz). Hearing loss was classified according to the World Health Organization grading system into normal, mild, moderate, moderately severe, severe, and profound categories. Sensorineural, conductive, or mixed types of hearing loss were determined based on air-bone gap analysis. In selected cases, tympanometry was performed to assess middle ear function and confirm normal tympanic membrane compliance.

Laboratory parameters including serum creatinine, blood urea levels, and estimated glomerular filtration rate (eGFR) were recorded from medical records to correlate renal function with hearing thresholds. Duration of renal disease was also documented to evaluate its association with degree of hearing impairment.

All collected data were entered into a structured data collection sheet and analyzed using appropriate statistical software. Continuous variables such as age, duration of disease, serum creatinine levels, and hearing thresholds were expressed as mean ± standard deviation.

Categorical variables such as gender distribution, presence or absence of hearing loss, and degree of hearing impairment were expressed as frequencies and percentages. Comparison of mean hearing thresholds between subgroups was performed using independent sample t-test or one-way analysis of variance (ANOVA) as appropriate. Pearson's correlation coefficient was used to assess the relationship between renal function parameters and hearing thresholds. Chi-square test was applied to analyze associations between categorical variables. A p-value of less than 0.05 was considered statistically significant.

This methodological approach enabled systematic evaluation of auditory function in patients with chronic renal failure and allowed assessment of the

association between CKD and hearing loss in the absence of confounding co-morbidities.

Results

A total of 70 patients with chronic renal failure were evaluated for hearing status using pure tone audiometry. Table 1 shows the incidence and laterality of hearing loss among study participants. Out of 70 patients, 48 demonstrated normal hearing bilaterally, while 22 patients exhibited hearing impairment. Among those with bilateral involvement, 12 patients had hearing thresholds in the 40–60 dB range and 4 patients in the 35–40 dB range. Unilateral involvement was observed in 6 patients, with 3 involving the right ear and 3 involving the left ear. This indicates that bilateral involvement was more frequent compared to unilateral presentation.

Table 2 demonstrates the overall incidence of hearing loss in patients with chronic kidney disease. Hearing impairment was observed in 20 out of 70 patients, accounting for 28.6% of the study population, whereas 50 patients (71.4%) had normal hearing thresholds. This suggests that nearly one-third of patients with CKD exhibited some degree of hearing deficit.

Table 3 presents the distribution of hearing loss based on ear involvement. Among the 20 patients with hearing impairment, 14 patients (70%) had unilateral hearing loss and 6 patients (30%) had bilateral hearing loss. Thus, unilateral impairment was more common among affected individuals.

Table 4 further analyzes unilateral hearing loss. Among 14 unilateral cases, 8 patients (57.1%) had right ear involvement, whereas 6 patients (42.9%) had left ear involvement, indicating slight predominance of right-sided involvement. Table 5 summarizes the etiopathogenesis of chronic kidney disease among study participants. Idiopathic CKD was the most common cause observed in 32 patients (45.7%). Minimal change disease was noted in 6 patients (8.6%), IgA glomerulonephritis in 5 patients (7.1%), and polycystic kidney disease in 4 patients (5.7%). Other etiologies including lupus nephritis, interstitial nephritis, renal tubulopathy, membranoproliferative glomerulonephritis, pauci-immune glomerulonephritis, crescentic glomerulonephritis, chronic glomerulonephritis, and drug-induced nephropathy were present in smaller proportions.

Table 6 depicts biochemical parameters of renal function. The mean blood urea level among participants was 41.3 mg/dl with a standard deviation of 9.85, and the mean serum creatinine level was 9.24 mg/dl with a standard deviation of 3.12, reflecting advanced renal dysfunction in the study group.

Table 7 shows the usage of ototoxic drugs among participants. Ten patients (14.3%) had a history of ototoxic drug exposure, while 60 patients (85.7%) reported no exposure. The majority of patients with hearing loss were not associated with ototoxic medication usage.

Table 8 outlines the distribution of specific ototoxic drugs among exposed patients. Among the 10 exposed individuals, furosemide was used in 4 cases (40%), gentamicin in 3 cases (30%), and amikacin in 3 cases (30%). The distribution indicates comparable usage among loop diuretics and aminoglycosides.

Table 1: Incidence of Hearing Loss Based on PTA (n = 70)

Side Involved	Normal	40–60 dB	35–40 dB	≤35 dB
Bilateral	48	12	4	0
Right ear only	–	3	1	0
Left ear only	–	3	1	0

Table 2: Incidence of Hearing Loss in CKD Patients (n = 70)

Incidence	Frequency	Percentage
Patients with hearing loss	20	28.6
Patients without hearing loss	50	71.4
Total participants	70	100

Table 3: Hearing Loss Based on Involvement of Ear (n = 20)

Hearing Loss Type	Frequency	Percentage
Unilateral	14	70
Bilateral	6	30
Total	20	100

Table 4: Involvement of Ear in Unilateral Hearing Loss (n = 14)

Ear Involved	Frequency	Percentage
Right ear	8	57.1
Left ear	6	42.9
Total	14	100

Table 5: Etiopathogenesis of CKD (n = 70)

Type of CKD	Frequency	Percentage
Idiopathic	32	45.7
Polycystic kidney disease	4	5.7
IgA glomerulonephritis	5	7.1
Lupus nephritis	3	4.3
Interstitial nephritis	3	4.3
Renal tubulopathy	4	5.7
Chronic glomerulonephritis	3	4.3
Membranoproliferative GN	4	5.7
Minimal change disease	6	8.6
Pauci immune GN	3	4.3
Crescentic GN	2	2.9
Drug induced nephropathy	1	1.4

Table 6: Urea and Creatinine Levels (n = 70)

Parameter	Mean (mg/dl)	SD
Urea level	41.3	9.85
Creatinine level	9.24	3.12

Table 7: Usage of Ototoxic Drugs (n = 70)

Usage	Frequency	Percentage
Yes	10	14.3
No	60	85.7

Table 8: Ototoxic Drugs Used (n = 10)

Drug	Frequency	Percentage
Furosemide	4	40
Gentamicin	3	30
Amikacin	3	30

Discussion

The present study evaluated hearing status in 70 patients with chronic renal failure in the absence of underlying co-morbidities and demonstrated that 28.6% of participants exhibited some degree of hearing impairment on pure tone audiometry. Bilateral involvement was more common in the overall audiometric pattern, although unilateral hearing loss predominated among affected individuals. These findings support the growing body of evidence suggesting that chronic kidney disease (CKD) independently contributes to auditory dysfunction. Recent epidemiological analyses have demonstrated that patients with moderate to advanced CKD have significantly higher prevalence of sensorineural hearing loss compared to matched healthy populations, even after adjusting for confounding factors such as diabetes and hypertension [11].

The predominance of mild to moderate sensorineural thresholds observed in this study aligns with cochlear vulnerability associated with metabolic and microvascular disturbances in CKD. The cochlea and kidney share structural and physiological similarities, particularly in terms of ion transport mechanisms and microvascular supply. Experimental and clinical investigations have suggested that uremic toxins, oxidative stress, and endothelial dysfunction contribute to degeneration of outer hair cells and stria vascularis dysfunction, ultimately leading to progressive hearing impairment [12]. The elevated mean serum urea and creatinine levels observed in our study population further reinforce the association between renal dysfunction and systemic metabolic burden, which may affect cochlear homeostasis.

The higher proportion of unilateral involvement among affected individuals may reflect early cochlear changes that later progress to bilateral impairment with disease duration. A multicenter clinical study evaluating auditory thresholds in CKD patients reported that high-frequency hearing loss often precedes bilateral symmetrical involvement and correlates with duration of renal disease [13]. This supports the importance of early screening in CKD patients even when auditory complaints are minimal.

Although ototoxic drug exposure was documented in a minority of participants, the majority of hearing impairment cases were observed in patients without a history of ototoxic medication usage, suggesting that renal dysfunction itself plays a primary etiological role. Recent nephrology-otology collaborative research has highlighted that CKD-related hearing loss can occur independently of aminoglycoside exposure, reinforcing the

hypothesis of intrinsic cochlear susceptibility in renal failure [14]. Additionally, microangiopathic changes in cochlear vasculature associated with chronic inflammation and endothelial dysfunction have been proposed as key mechanisms contributing to sensorineural deficits in CKD [15]. The etiopathogenic spectrum of CKD in the present study was predominantly idiopathic, with other glomerular and tubulointerstitial causes contributing smaller proportions. Despite variation in underlying renal etiology, hearing impairment was observed across different CKD subtypes, indicating that reduced renal function rather than specific disease origin may be the principal determinant of auditory dysfunction.

Overall, the findings of this study strengthen the evidence that chronic renal failure is associated with measurable auditory impairment even in the absence of common systemic co-morbidities. Early audiological screening and monitoring in CKD patients may therefore be beneficial for timely detection and intervention, ultimately improving quality of life and communication outcomes in this vulnerable population.

Conclusion

The present study demonstrates a considerable prevalence of hearing impairment among patients with chronic renal failure in the absence of underlying co-morbidities. Both unilateral and bilateral sensorineural hearing loss were observed, with mild to moderate thresholds being the most common pattern. Elevated renal biochemical parameters and absence of significant ototoxic drug exposure suggest that renal dysfunction itself may contribute to cochlear impairment. These findings highlight the importance of routine audiological evaluation in patients with CKD for early identification and management of hearing loss.

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