Evaluation of Cochlear Functions in Renal Failure by "Pure Tone Audiometry

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ABSTRACT

Introduction: Chronic kidney disease (CKD) has been associated with hearing loss. The kidney and stria vascularis of the cochlea share physiologic, ultra structural and antigenic similarities that could underlie the link between CKD and hearing loss. Aim: The present study is taken up to study magnitude of SNHL in chronic kidney disease patients.

Material and methods: 100 patients (200 ears) who had chronic kidney disease (CKD 5) and all patients underwent haemodialysis. All patients had Audiological evaluation with pure tone audiometry patients had sensorineural hearing loss.

Results: high frequencies are affected in 52%, middle frequencies in 9%, low frequencies in 2.5% of individuals. 2.5% had hearing loss in all frequencies. Hearing loss is observed only at 8000 Hz in 10% of individuals. hearing loss was found in 61 (61%) members bilaterally. Unilateral hearing loss is present in 3(1.5%) patients. Hearing loss present in 62.5% individuals (125 out of 200 ears). Sensorineural hearing loss present in 62.5% in patients with chronic renal failure and severity correlated with duration of disease. No correlation between other co variables.

Conclusions: It should encourage clinical nephrologists to include questions about hearing function in their preventive care protocols, to refer all patients reporting hearing loss to a hearing health professional for evaluation and/or rehabilitation (eg, hearing aids), and recommend that patients avoid further treatment with ototoxic medications to preserve their hearing ability.

Keywords: Chronic Kidney Disease, Sensorineural Hearing Impairment, Haemodialysis

INTRODUCTION

Sensorineural hearing impairment (SHI) has been reported in chronic renal failure (CRF) patients with a prevalence of 20-40%. The aetiopathogenetic mechanisms reported included osmotic alteration resulting in loss of hair cells, collapse of the endolymphatic space, oedema and atrophy of specialized auditory cells and in some cases due to complications of haemodialysis.

Chronic kidney disease (CKD) has been associated with hearing loss since 1927, when Alport first described a case in which hearing loss was associated. Familial kidney disease like HDR (hypoparathyroidism, deafness, and renal dysplasia) syndrome, brachio oto renal syndrome, Fabry disease, and MELAS (mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke) syndrome are some of the other rare conditions or syndromes in which hearing loss is closely linked to CKD.

Reports over many years also have shown association between various other chronic kidney conditions and hearing loss. However, all the current evidence is derived from small observational studies of patients with CKD or samples of patients on renal replacement therapy. Several small studies have indicated an increased prevalence of high-frequency hearing loss in patients with CKD or those with end-stage kidney disease who are on dialysis therapy. However, to date, no large population-based study has assessed the association between non syndromal CKD and hearing loss. Moreover, the exact cause and pathogenesis of hearing loss in the CKD population is unknown. An association between CKD and sensorineural hearing loss also is described in the pediatric age group, although in this population, effects were observed more commonly in transient evoked Otoacoustic emissions testing, rather than the pure-tone audiogram, suggesting that outer hair cell damage preceded neural damage.

Other smaller studies have examined associations between hearing loss and hemodialysis therapy and documented acute and long-term changes in hearing occurring in relation to hemodialysis. The kidney and stria vascularis of the cochlea share physiologic, ultra structural and antigenic similarities that could underlie the link between CKD and hearing loss. It has been suggested that common physiologic mechanisms involving fluid and electrolyte shifts in stria and kidney might explain the association between hearing loss and CKD. There also are certain anatomic similarities at an ultra-structural level and evidence for similar antigenicity of the cochlea and kidney. Multiple shared risk factors for CKD and hearing loss include age, diabetes, hypertension, and medications that are both ototoxic and nephrotoxic. Moreover, in patients with established CKD, multiple risk factors have been hypothesized to cause hearing loss, including use of ototoxic medications, hypertension, and

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diabetes, particularly in association with hypertension, electrolyte disturbances, and hemodialysis itself.

Hearing sensitivity is typically described in terms of how loud the sound must be in order to be detected. This is reported in decibels relative to the hearing of people without ear problems. Sensitivity is tested for a number of frequencies. Sound frequencies are reported in cycles per second or Hertz (Hz). Though human beings can typically hear up to 20,000 Hz, testing is usually done from 250 to 8,000 Hz.

Audiometry is a technique used by an audiologist or an otolaryngologist to measure hearing. There are two components of hearing loss: (a) conductive hearing loss, (b) sensorineural hearing loss.

Pure-tone audiometry (PTA) is a standard procedure used in clinics to measure the threshold of audibility for pure tones presented to a listener over headphones and via bone vibrators held on mastoids. Threshold measurements, made for an agreed set of frequencies, are expressed in decibels (dB) and plotted on a graph called a pure-tone audiogram. Hence the present study has been taken up to determine magnitude of hearing loss in chronic renal failure patients.

Stage	GFR, mL/min per 1.73 m^2
0	>90
1	90
2	60-89
3	30-59
4	15-29
5	<15

Table-1: Staging of Chronic renal failure

Degree of hearing loss	dB Hearing loss
Mild	20-40 26 to 40
Moderate	41-55 41 to 55
Moderately severe	56-70 56 to 70
Severe	71-91 71 to 91
Profound	>91 More than 91

Table-2: WHO Classification of hearing loss

Hearing loss	Bilateral (%)	Unilateral (%)	Total (%)
Speech frequency average	11	6	17
4000 Hz	48	5.5	53.5
8000 Hz	61	1.5	62.5

Table-3: Hearing loss at different frequencies

MATERIAL AND METHODS

A total of 100 patients were enrolled into the study between time period of December 2015 to August 2017 attending dialysis center in Rajiv Gandhi Institute of Medical Scinces, Kadapa.

Inclusion criteria: All cases diagnosed as chronic renal failure. They will be staged according to following criteria.

Exclusion criteria: Patients younger than fifteen years of age, who have undergone prior ear surgery, with tympanic membrane perforation, tympanosclerosis and otosclerosis.

Investigations done are Battery of audiological tests: Pure tone audiometry.

Nephrological investigations like serum creatinine, blood urea nitrogen, serum electrolytes.

Hearing-related questions included family history of hearing loss, past medical or surgical treatment of otologic conditions, diseases associated with hearing loss, and risk factors for ear disease. Other questions addressed exposure to noise at work.

Cases with past history of hearing loss, ear discharge, diabetes, and hypertension were not included in the study. Thereafter, all patients were subjected to basic tests of renal function Hemoglobin, Hematocrit, blood urea, serum creatinine, and blood urea nitrogen). On the basis of the findings of biochemical investigations, GFR was calculated. GFR calculated by Cockcroft-Gault formula.

Pure tone audiometry

pure-tone audiometry and was performed by qualified audiologists in sound-treated booths using Elkon EDA 3N3 MULTI audiometer.

All patients had baseline audiological evaluation with pure tone audiometry. Pure tone audiometry was performed for both air conduction and bone conduction for 250, 500, 1000, 2000, 4000, 8000Hz. Because bone conduction hearing testing is limited to 4000 Hz, measurements ≥ 4000 Hz were performed using air conduction testing alone. Sensorineural hearing at high frequencies (8000 Hz) tested by air conduction is unaffected by, and independent of, middle ear effusion.

Patients divided in to three age groups (15-30, 31-45, 46-60), duration of disease in to two groups (<5,>/= 5 years), Hemodialysis in to two groups (<3 years,>/=3years). All three variables compared with hearing loss. Hearing loss divided according to pure tone average, mild, moderate, moderately severe, severe and profound. Also divided according to frequencies involved in to low, middle and high frequencies (table-2).

WHO (1980) recommended above classification on basis of

Hearing loss	Duration			
	<5years (%)	>5years (%)	Total HL (%)	P value
8000 Hz	32.8	78.9	62.5	0.001
4000 Hz	30	45	53.5	0.001
PTA	10.5	24	17	0.001

Table-4: CKD duration and hearing loss

pure tone audiogram taking average of thresholds of hearing frequencies 500,1000,2000 Hz with reference to ISO: R. 389-1970.

Procedure of PTA

The patient is instructed about the procedure in detail. The examiner should be able to observe the subject, but care should be taken to provide no visual clues to the subject, as the examiner operates the audiometer. The headphones are placed over the subject's ears, so that the centre of each transducer is at the ear.

Tones should be presented for 1 to 3 seconds with the intervals of 1 to 3 seconds between each presentation. It is important to randomize the intervals and to avoid presenting the tones in a rhythmic fashion to facilitate the recognition of true response. The subject responds as soon as he hears the sound. A method of air conduction threshold assessment by conventional Hughson-Weslake technique slightly modified by Carharts and Jerger is described below.

Technique of air conduction test

The various frequencies are presented in the following order, 1000, 2000, 4000, 8000, repeated again followed by 500, 250, 125 Hz. For given frequency, the initial presentation should be an arbitrary presumed suprathreshold level, to allow easy recognition and identification. If patient hears then the tone is decreased by 10dB steps until patient stops hearing. Once this stage is reached, the tone is raised by 5dB. If the patient hears this tone, the sound is again decreased by 10dB. If he does not hear it, the tone is again raised by 5dB. In this way by several threshold crossings, the exact hearing threshold is obtained when one gets at least 3 out of responses correct. Though threshold is defined as 'the lowest intensity heard on 50 percent of occasions of repeated crossing', but in clinical practise, this is not usually possible on clinical audiometer where gradations are in 5dB. The second ear is tested in a similar manner. The faintest audible intensity as established above is recorded against the test frequency on a standard audiogram chart as the threshold intensity. By convention, the symbols 'o' and 'x' are used for air conduction thresholds for the right and left ears respectively. If the maximum intensity of the audiometer at a given frequency cannot be heard, this is indicated by a downward pointing arrow at the level of the maximum output on the appropriate frequency line.

Technique of bone conduction test

Bone conduction thresholds are obtained in an identical manner to those described for air conduction, but the sound stimulus is produced by a bone vibrator placed on the mastoid process and held firmly, by means of a head band. Care is taken to remove any intervening hair and contact with the cartilaginous external meatus or pinna is also avoided during the test as these structures may carry air conducted sounds. The vibrator should be placed on the mastoid process of the ear with the worse air conduction threshold averaged over the frequency range 250 to 5000Hz. Measurements are restricted to the frequency range 250 to 4000Hz and calibration standards do not generally give data for stimuli outside this range. The test is examined at 1000Hz followed by 2000, 4000, 500 and 250Hz. The subject is instructed to respond to sound regardless of the side on which the sound is actually heard. It must be emphasized that without the use of masking it is not possible to determine the ear that is responsible for the detection of the 'non masked, bone conduction threshold. Masking is mandatory for-

1. All bone conduction studies, whether the unmasked bone conduction is 10dB or more better than the worse air conduction.
2. Air conduction studies.
   a. When the difference in left and right unmasked air conduction threshold is 40dB or more, and
   b. Whether the unmasked bone conduction is 40dB or more better than the worse air conduction.

These requirements for masking may be readily understood considering certain facts regarding the transmission of air and the bone conduction sounds across the head. An air conducted sound is transmitted across the skull with an internal attenuation of the order of 50dB; while the attenuation for a bone conducted sound is negligible. Hence, in this later condition and apparent threshold level may be a

Relation of hearing loss with	P Value
Hemodialysis duration	0.08 (weak relation)*
GFR	0.2
Serum Creatinine	0.68
Haemoglobin	0.429
Serum Electrolytes	0.176
Serum. Calcium	0.11
Diastolic blood pressure	0.3

Table-5: Correlation of hearing loss with variables

Study	No: of subjects	Age	Auditory method	Hearing loss / auditory function
Henrich *et al*[10]	20	adults	PTA	yes
Charachon *et al*[7]	54	adults	PTA	yes
Jonhson and Mathog[13]	61	adults	PTA	yes
Kusakari *et al*.[15]	229	adults	PTA	yes
Bergstrom[4]	151	children	PTA	yes
Mancini *et al*.[9]	68	children	PTA	yes
Nikolopoulos *et al*.[19]	46	children	PTA	yes
Stavroulaki *et al*.[19]	9	children	PTA+DPOAE	yes
Zeigelboim *et al*.[8]	37	adults	PTA	yes
Our study	100	adults	PTA	yes

Table-6: Studies on effects of CRF on hearing

record of the sensitivity of the cochlea not under test.
The 'shadowing' technique of determining the true auditory threshold is the most commonly used masking technique.
Hoods plateau method of masking:

Air conduction

1. The unmasked threshold of the ear is ascertained. If it is thought that there is a possibility of cross hearing, then a masking sound is introduced into the non-test ear at 10 or 15dB above air conduction threshold level of the non-test ear.
2. The tone is presented to the test ear (at unmasked threshold). If the patient gets the tone, masking will not be required**.**
3. The test tone is raised by 5dB, if the patient hears the tone, then next (4^{th}) step is started.
4. The masking level is raised by 5dB in the non-test ear. Tone is again presented to the test ear to see whether the patient is getting the tone or not.
5. The test tone is raised by 5dB or in 5dB steps till it is heard. Once the test tone is heard the masking level is raised by 5dB and it is checked whether test tone is still being heard.

Bone conduction

This is similar to air conduction masking.

1. Unmasked bone conduction threshold of test ear is ascertained.
2. A masking sound is introduced into the non-test ear at a level of 15dB above air conduction threshold for the non-test ear
3. Tone is given by bone conduction to see whether it is being heard or not. If heard, it indicates that masking threshold is correct. If not heard, then tone and masking sound are increased in 5dB steps alternately till masking sound level can be increased by 2-3 steps of 5 dB each

RESULTS

Proportion of hearing loss in patients of chronic renal failure (100 patients) to study the quantification of magnitude of sensorineural hearing loss. This is a cross sectional study. The study population included 71 Men and 29 Women, Age range was 15-60 years.
Duration of illness 1-9 years. Duration of hemodialysis was 1-6 years. The diastolic BP was between 90-110 mmHg. Patients with retracted tympanic membrane, thin tympanic membrane, tympanosclerosis fluid in the middle ear also excluded from study after otoscopic examination
Hearing loss, defined as average pure-tone threshold >26 dB for measurements pure tone average at frequencies of 0.5, 1.0, 2.0 kHz**.**
Hearing loss at speech frequency (500-2000 Hz) is seen in 17% of individuals. But hearing loss present at 4000 Hz in 53.5% individuals (48% Bilateral, 5.5% unilateral) and 8000 Hz in 62.5% individuals (61% Bilateral, 1.5% unilateral).
Hearing loss also observed at 4000 Hz in 53.7% individuals. Mild degree hearing loss present in 46% of patients, Moderate hearing loss present in 7% of patients, moderately severe in 0.5% of patients. Severity of hearing loss in different frequencies is not the same.
Hearing loss also observed at 8000 Hz in 62.5% individuals. Mild degree hearing loss present in 50% of patients, Moderate hearing loss present in 12% of patients, moderately severe in 0.5% of patients. Only 8000 Hz dip hearing loss in 10% of patients.
61 (61%) members bilaterally. Unilateral hearing loss is present in 3 (1.5%) patients. Total hearing loss present in 62.5% individuals (125 out of 200 ears).
high frequencies are affected in 52%, middle frequencies in 9%, low frequencies in 2.5% of individuals. 2.5% had hearing loss in all frequencies. 10% of individuals have hearing loss only at 8000 Hz.
Patients were divided in to three groups based on age (15-30, 31-45, 46-55 years) and two groups based on duration of disease (<5,>/= 5 years), Hemodialysis in to two groups(<3 years,>/=3years). All three variables compared with hearing loss. Hearing loss divided according in to mild, moderate, moderately severe, severe and profound and based on frequency in to low, middle and high frequency hearing loss. All patients had sensorineural hearing loss.
There is significant correlation between duration of illness and hearing loss (P=0.01).

DISCUSSION

Hearing loss in the CKD population has been reported as being mainly sensorineural. Other causes of sensorineural hearing loss include age, diabetes, congenital hereditary otonephropathies, ototoxic drugs, such as furosemide or exposure to work- or industrial- related noise.
The cochlea and kidney have similar physiological mechanisms, namely the active transport of fluid and electrolytes accomplished by the stria vascularis and the glomerulus, respectively[2,3]. They may also have common antigenicity [1,2]. These may account for similar effects of medications (i.e. nephrotoxic and ototoxic effects of aminoglycosides) and immunological factors on the two organs. Inner ear and kidney development are both influenced by similar genetic factors in hereditary conditions such as Alport's syndrome and branchio-oto-renal syndrome. Several aetiological factors have been linked to hearing loss in renal failure [4] including use of ototoxic medications, electrolyte disturbances, hypertension [5] and haemodialysis treatment itself.[6]
The higher incidence of hearing loss among children with CRF has long been established and is constantly being verified by new studies Charachon et al.[7] reported that 75% of 54 patients with CRF had hearing loss. Zeigelboim et al.[8] measured thresholds between 9and 18 kHz in 37 patients with CRF undergoing conservative treatment and a control group with normal hearing function. Age ranges in both groups were 30–59 years. They found a more severe high-frequency hearing loss in the group with CRF. Hearing loss among patients with CRF seemed to deteriorate further a year after the first evaluation.
Our study age range was 15 to 60 years. Hearing loss assessed up to 8 KHz only.

Bergstrom et al.[4] compared a group of patients with hearing loss of unknown etiology, one with strial deposits and one with neither. They found no difference between the groups. Zeigelboim *et al*[8] reported more severe high-frequency hearing loss in the group with CRF. Hearing loss among patients with CRF seemed to deteriorate further a year after the first evaluation.

Mancini et al.[9] reported hearing loss in 47.5% of patients with congenital disease and in 21% of children with acquired renal disease. Henrich et al.[10] found that 75% of the patients showed no deterioration of hearing during the 4-year time of follow-up. They concluded that hearing loss is common in renal failure, but it does not worsen with duration of treatment. In our study there was significant correlation between duration of disease and the hearing loss.

Risvi and Holmes[11] reported a patient with progressive hearing loss parallel to progression of CRF, peritoneal dialysis and haemodialysis. They found anatomic changes in the labyrinth, which they attributed to osmotic disequilibrium caused by haemodialysis. Antonelli *et al*[12] reported that pure tone hearing loss as well as wave I latency of the CRF group was correlated with age and negatively correlated with serum albumin level. Wave I was additionally correlated with calcaemia. Johnson *et al.*[13] investigated auditory function in older adults using pure tone measurments in 71 adults he observed hearing loss in CRF patients. Samir et al.,[14] found no correlation between pure tone audiometry findings and OAE measures and serum electrolyte levels.

Kusakari et al.,[15] reported that inner ear dysfunction (including hearing loss and vestibular dysfunction or a combination) was not correlated with BUN and serum creatinine levels or with and serum urea nitrogen, creatinine, potassium, Sodium, Calcium and glucose levels.

In our study there was no correlation between age, sex, potassium, calcium, serum creatinine, diastolic blood pressure, albumin, globulin level (P valve> 0.05).

Johnson *et al.*[13] found no relationship between fluctuations of hearing and serum urea nitrogen, creatinine, K^{+}, Na^{+}, Ca^{++} and glucose.

In a similar report Mancini et al.,[9] suggested that neural conduction along the auditory pathway is delayed irrespective of haemodialysis onset, basically due to the disease. Baldini et al, found no correlation between ABR wave prolongation measures and plasma level of vitamin B12, folic acid, PTH and beta-2 microglobulin. Duration of disease and/or blood measures does not seem to have a significant impact on auditory function.

But in our study there is significant correlation of duration of disease and hearing loss. In our study there is no correlation between age, sex, potassium, calcium, serum creatinine, diastolic blood pressure, albumin, globulin level (P valve> 0.05).

Several studies have suggested that longer hemodialysis session lengths may be beneficial, although these studies are confounded by a variety of patient characteristics, including body size and nutritional status.

Bergstrom et al.[16] reported hearing loss in 40% of the CRF patients on haemodialysis. Bergstrom[16] and Thompson[10] reported that 47% of 151 pediatric end-stage renal patients had hearing loss. Hearing loss is a more common finding reported than vestibular dysfunction.

Kusakari et al.[17] reported on inner ear function of 229 patients on chronic haemodialysis. They found that 60% had hearing loss, 36% had vestibular dysfunction and 26% had a combination of both. Johnson and Mathog[13] noted high frequency hearing loss in 61 adults early in the course of haemodialysis.

In our study, we had high frequency hearing loss in 50 percent of the patients and 13 percent hearing loss in patients with Hemodialysis.

Mancini et al,[9] found sensorineural hearing loss in 29% of the children on conservative treatment, 28% of the children on haemodialysis and 47% of the children with renal transplants. There were no correlations between hearing loss, duration of nephropathy and haemodialysis treatment. In our study in adults haemodialysis patient have 62.5% individuals having hearing loss

Samir et al,[14] found a significantly higher incidence of cochlear dysfunction among children on haemodialysis compared with children on conservative treatment, in contrast to despite overall similar median duration of haemodialysis. However, renal function among patients on dialysis is worse than among patients on conservative treatment, which further complicates the distinction between the effects of a more severe renal impairment from effects of the treatment. Albeit novel and interesting, this finding should be interpreted with caution, in light of the small number of subjects in the conservative treatment group.

We did not have conservative treatment group to compare with haemodialysis.

Marsh et al,[18] concluded that the CAPD group showed function closer to normal than the chronic dialysis patients. Nikolopoulos et al.[19] Evaluated 41.3% had hearing loss. Conductive hearing loss and ototoxicity accounted for 11%, whereas 30.4% was of unknown aetiology, therefore, it could be attributed to CRF or haemodialysis. Hearing was mostly impaired in the high frequencies, with 30% of the ears affected to a lesser degree in the middle and low frequencies. Forty-seven percent of the children in the haemodialysis group had hearing loss, compared with 32% in the pre-end stage of renal insufficiency group and none in the CAPD group.

Rossini et al,[20] recorded ABRs from 17 CRF patients on conservative treatment and 11 on chronic dialysis. They found abnormal responses in 32.15% of the patients. Waveform morphology was normal in most of the patients; with latency prolongation of all waves following wave I. Altered ABRs were more frequent in the conservative treatment group. The above studies showed that method of treatment may influence the impact of the disease on hearing, a topic yet to be conclusively investigated.

Gartland et al,[5] documented a low frequency hearing loss, which improved significantly on one-third of the patients after dialysis. As low-frequency sensorineural hearing

loss is related to endolymphatic_hydrops, they postulated that changes in fluid balance during haemodialysis may be accountable for the low frequency hearing improvement. However, there was no correlation between weight and hearing changes after haemodialysis.

In our study we had low incidence of hearing loss in lower frequencies. But we didn't include 125 Hz as in above study. Ozturan and Lam[3] found a notch at 6 kHz among CRF patients not related to haemodialysis indices. Therefore, the frequency specificity of possible CRF/ haemodialysis effects remains inconclusive. In our study we had notch at 8k and 4k Hz. But statistically not significant with hearing loss when compared to other indices.

Serbetcioglu et al,[6] noted a permanent high frequency hearing loss, but no specific effects of haemodialysis. Similarly, Nikolopoulos et al.[19] found no effect of a single haemodialysis session on the hearing of nine haemodialysed children. Kligerman et al. evaluated the hearing of patients with CRF, following 12 of them for 1 year as they were going through haemodialysis. A second group of patients not on haemodialysis were re-evaluated at the end of the year; a third group having received haemodialysis for 1.5, 2, 3 and 6 years were included in the study.

In our study, we divided patients in to two groups <5 and >5 years. There is no statistical significance when compared to hearing loss.

Similarly, Bazzi et al [21] did not report a correlation between haemodialysis duration and severity of hearing loss. Therefore, duration on haemodialysis treatment did not appear to affect the degree of hearing loss in the CRF patient population.

Ozturan and Lam[3] examined the effects of a single session of haemodialysis on pure tone thresholds and DPOAEs. They tested 15 patients of 19–45 years of age prior to and following a session of haemodialysis in a similar study with Stavroulaki et al.[19] There were no significant changes in the pure tone thresholds or the DPOAE amplitude in either study. As ours cross sectional study, we did not have follow up study after haemodialysis

Pratt et al.[22] obtained ABRs from 38 patients before and after haemodialysis along with blood chemistry data. They found abnormal ABRs in 24% of the patients at slow stimulus presentation rate (10/s) and in 44% of the patients at the fast presentation rate (55/s). These abnormalities consisted of prolonged latencies and interpeak latency differences indicating both a cochlear and a retrocochlear involvement. The temporary effect of haemodialysis on peaks III and V at the slow rate and I and V at the fast rate were correlated with changes in calcium levels. These findings are consistent with Rossini et al. who found a decrease in I–V interpeak latency 26 h following haemodialysis. However, this finding was noted in only two patients, which precludes generalization.

Pagani et al.[21] found prolongation of wave and interwave latencies when compared with a group of non-CRF controls, but no difference between the groups with CRF. In other words, they found evidence of pathology along the auditory pathway in the CRF groups, with no indication that the length of dialysis treatment or the length of the disease. The high incidence of hearing loss among children and adults with CRF is well-documented in published reports.

Duration on haemodialysis treatment does not seem to have a significant impact, although the method of treatment may influence the impact of the disease on hearing. The literature concurs that the main site of lesion is cochlear with some retrocochlear findings in auditory brainstem audiometry. However, lack of correlation between hearing function and a blood measure precludes a detailed description of the mechanisms causing hearing loss in CRF. Changes in the dialysis treatment have eliminated the temporary effects of single session of dialysis on hearing function.

Bazzi et al [21] concluded there is a high incidence of hearing loss in hemodialysis patients and that the number of years of dialysis treatment did not in itself influence the prevalence of hearing loss. All patients in our study, all patients underwent haemodialysis. In our study, there is no significant correlation between hearing loss magnitude and duration of haemodialysis.

CONCLUSION

Evidence of a possible link between kidney function and hearing loss, as suggested by our study, potentially could modify the usual care of people with CKD. It should encourage clinical nephrologists to include questions about hearing function in their preventive care protocols, to refer all patients reporting hearing loss to a hearing health professional for evaluation and/or rehabilitation (eg, hearing aids), and recommend that patients avoid further treatment with ototoxic medications to preserve their hearing ability.

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