Sensorineural Hearing Loss in Chronic Kidney Disease – Outcome of Trial of Steroid Therapy

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ABSTRACT

The prevalence of sensorineural hearing loss (SHL) in chronic kidney disease (CKD) patient was between 10% - 67%. However, the outcome of treatment has not been well studied, especially in Nigeria. Our hypothesis is that steroid therapy may achieve improvement in hearing in these patients, hence we report the outcome following a trial of oral steroid. This was prospective study involving CKD patients with SHL. The subjects were commenced on oral prednisolone 60mg/day tailed over 4 weeks and this was repeated after 1 month and each patient had 3 courses. The pure tone average (PTA) was measured with a computer audiometer BA 20 Kamplex before and after treatment. The study population of 25 subjects involved 14males and 11 females between the ages of 19 and 65 years, (46 $\pm$ 21.2). The PTA was between 65 - 100dB, mean = 70dB compared with post treatment hearing acuity of 50 – 95dB, mean = 65dB. Comparing the pre- and post- treatment PTA, an improvement of 5dB was seen in 2/25(8%), no change in 4/25(16%) while the PTA was increased by 10 – 30dB in 19/25(76%). Pearson correlation to compare the mean of pre- and post– treatment PTA revealed no significant difference (P =0.08). In conclusion, SHL in CKD patients was not responsive to steroid therapy; this is probably due to various persistent aetiological factors. However, this is an indication for further search for the effective treatment in order to improve quality of life in these patients.

Keywords: Sensorineural hearing loss; chronic kidney disease; steroid therapy; pure tone average.

INTRODUCTION

Kidney – related ear dysfunction has been established, as the cochlea and retrocochlear region have been reported to be susceptible to the effect of uremia [7] but the pathogenesis is still being investigated. The reported prevalence of otologic symptoms in chronic kidney disease (CKD) was between 10% - 67% [1-3]. In addition, patients undergoing treatment with hemodialysis (HD) has also been found to manifest some degree of sensorineural hearing loss [4-6]. Several factors which are often present in these patients include ototoxicity, the advanced age and the possible accelerated presbycusis[8]. The multiplicity of aetiological factors make the treatment of the SHL difficult hence persistent. This probably accounted for the reason why the treatment of the hearing loss has not been emphasized in the literature. The purpose of this study is to report the hearing outcome in a trial of systemic steroid. Our hypothesis is that steroid therapy may achieve recovery of hearing function by suppressing the cochlear inflammatory response in these groups of patients.

MATERIALS AND METHOD

Following report of sudden hearing loss in 2 patients on hemodialysis [3], 33 CKD patients were recruited

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for Pure Tone Audiometry (PTA) at admission and after three sessions of hemodialysis. The pure tone audiometry was done with a computer audiometer BA 20 Kamplex in the sound – proof (acoustic) booth in the ENT clinic. The hearing acuity was measured in db at the frequencies 500 – 8000Hz. The average for the four frequencies 500Hz, 1000Hz, 2000Hz and 4000Hz were recorded. A similar number of age and sex matched control were selected among volunteers who were otherwise clinically healthy and had pure tone audiometry done and recorded.

All patients with hearing loss, defined as pure tone average greater than 30dB, were commenced on prednisolone 60mg/day tailed over 4 weeks.. This course was repeated after 1 month interval and each patient had 3 dose regimes. Among these, 25 were followed up with PTA. The first audiometry was done immediately at the end of the first course of steroid, then at 3 – 6 month interval.

Inclusion criteria: Patients with chronic kidney disease, already under management by nephrologist, with complaints of hearing loss, or detected to have hearing loss on routine audiometry. Exclusion criteria: History of otitis media, unconscious patients and severe illness precluding patients from responding at audiometry.

The data was processed using the Statistical Package for the Social Sciences (SPSS Inc, Chicago, Illinois, USA). The mean hearing acuity of pre- and post – steroid treatment were determined and paired student t – test was used to find the significance of the difference. Pearson correlation coefficient was used to find the correlation between PTA and other variables such as duration of illness, blood pressure, serum creatinine, dosage of diuretics and age.

RESULT

A total of 25 patients were treated and followed up for 1 – 3 years. They are made up of 14 males and 11 females between the ages of 19 – 65 years, mean 46(SD = 21.2). There was an improvement of 5dB between pre- and post- treatment PTA in 2/25(8%) patients, the values were the same in 4/25(16%) while the PTA was increased by 10 – 30dB in 19/25(76%). The pretreatment pure tone average (PTA) was between 65 - 100dB, mean = 70dB compared with post treatment hearing acuity of 50 – 95dB, mean = 65dB.

Pearson correlation to compare the mean of pre- and post– treatment hearing acuity revealed no significant difference (P =0.08).

DISCUSSION

The major finding from this study revealed that there was no significant difference in the mean pure tone average pre- and post – treatment of the hearing loss with systemic steroid. This is an indication that the hearing loss in chronic renal failure was not responsive to steroid therapy. Most reports are in support of persistence and/or deterioration of hearing loss during and after hemodialysis. Simone *et al* [9] also reported further deterioration of hearing function in CKD after 1 year of follow-up. They found high frequency hearing loss in 37 patients with chronic renal failure undergoing conservative medical treatment [9]. Gatland *et al* [10] reported increased hearing threshold after hemodialysis in 22/31 ears with pre-existing low frequency loss with little change in other frequencies and no correlation with weight changes. They suggested treatment induced changes in fluid and electrolyte composition of endolymph as possible mechanisms [10]. Mancini *et al* found SNHL in 29% of patients on conservative treatment, 28% of patients on hemodialysis, and 47% after renal transplantation. They also found significant correlation with the administration of ototoxic drugs thus hypothesized that SNHL may be reduced in patients with CKD or on renal replacement therapy by strictly monitoring ototoxic therapy [11]. The choice of steroid is based on the thinking that steroid could reduce the cochlear inflammatory response to uraemia and other toxic products of metabolism in renal failure, however the persistence of the hearing loss after steroid therapy appeared not to have substantiated this impression**.** It may also be due to the multiplicity of factors responsible for hearing loss in these patients. Continued use of diuretics and antihypertensives resulting in cochleotoxicity is one major factor in these patients. The other factors include ototoxins, axonal uremic neuropathy, anemia, and toxic degradation products from cellulose acetate dialyzer membranes [1-7]. In addition there are various inherited conditions in which renal diseases are associated with sensorineural deafness and the hearing loss in these conditions are persistent. These include alports, refsum disease, Charcot-Marie-Tooth, ataxia hyperuricaemia, ichthyosis, branchio- oto- renal (BOR) syndrome etc. There is a chance that this may be missed in these patients and the underlying genetic defect may be responsible for the persistence of the deafness [12]. In BOR and renal tubular acidosis, genetic mutation on the EYA1 gene on

chromosome 8q13.3 and mutations in ATP6V1B1 and ATP6V0A4 expression within the ***cochlea*** has been reported [13-15].

All these factors are conditions that can persist chronically in the uraemic state leading to irreversibility of the hearing loss once it develops.

We conclude that the observed sensorineural hearing loss in chronic renal failure patients was not responsive to steroid therapy; this is probably due to various persistent aetiological factors in these patients. However, this report further emphasizes the continued search for the treatment of sensorineural hearing loss in chronic kidney disease; otherwise it still appears an irreversible process.

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