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Hypothesizing the Role of Renal Secondary Hypertension in Ischemic Sudden Sensorineural Hearing Loss: A aase Report

Prasanna V — Bachelor of Audiology & Speech Language Pathology, Department of Speech & Hearing, Manipal College of Allied Health Sciences, Manipal University.

Kishan M M — Assistant Professor-Senior, Master of Audiology & Speech Language Pathology, Department of Speech & Hearing, Manipal College of Allied Health Sciences, Manipal University.

Ranjana Elizabeth James — Bachelor of Audiology & Speech Language Pathology, Department of Speech & Hearing, Manipal College of Allied Health Sciences, Manipal University

ABSTRACT

Understanding the pathophysiology of Ischemic sudden sensorineural hearing loss (ISSNHL) continues to be an enigma. With numerous probable etiologies cited and many hypotheses attempting to explain the pathophysiology, it is quite essential to understand the stand of various theories for the management of the condition. The widely believed etiology being vascular abnormalities in the microcirculatory cochlear structures throws a light for further understanding which is required to explain the recovery in cases of sudden sensorineural hearing loss. This case report is an evidence of sudden sensorineural hearing loss associated with chronic renal condition of Cystitis Glandularis, assumed to have had a vascular cause owing to hypertension secondary to chronic renal disease and recovered significantly with the right treatment.

INTRODUCTION

American academy of Otolaryngology (1996) explains Ischemic sudden sensorineural hearing loss (ISSNHL) as a condition in which the hearing acuity decreases by 30dB or more over three or more frequencies in 72 hours or less. ISSNHL is observed mostly unilaterally, however also is observed bilaterally, and may be the cause for any degree of loss. This condition is explained with various causes but vascular and viral infections have been cited as the most common factor (1) and the focus of this article will be on the vascular dimension of ISSNHL etiologies. Condition of vascular constriction leading to hypertension with the potential compromise of the microcirculation explains the vascular cause of ISSNHL.

Hypertension in cases of chronic kidney diseases (CKD) is often observed. It is termed secondary hypertension as it occurs in the presence of a medical condition. Cystitis Glandularis is a condition of metaplastic transformation of the mucosal cells lining the urinary bladder and is considered to be a rare CKD. Irrespective of acute or chronic, all kidney diseases affect the normal physiology of nephrons which can have a hand on the ionic concentrations in the body especially the Sodium and Potassium concentrations in the blood. Interestingly, the incidence of hearing loss in CKD is also observed in high percentages and has been noted as a factor which increases risk of having hearing loss The incidence of hearing loss was 41% in the low, 15% in the middle and 53% in the high frequency ranges respectively in CKD (2).

This triggers the need for understanding the pathophysiology of renal secondary hypertension and its possible effect on hearing can bring about a change in the outlook of audiologists towards ISSNHL and can greatly aid in treating the cause rather than the symptom of sudden hearing loss.

CASE REPORT

Reporting a 50 year old case that reportedly had difficulty in micturation, hematuria, dysuria, pain in the right loin during reporting developed sudden hearing loss bilaterally with loss more in the left ear. The patient reportedly sustained sudden onset of hearing loss after 2 years of developing the renal symptoms ,and hadn't recovered in the span of 2 months. The patient was examined for the renal and hearing symptoms. Person was subjected to ultrasonography, blood sample testing, and Pure tone Audiometry (PTA). PTA was carried out by a calibrated GSI-61 Clinical Audiometer with TDH-50P headphones and B-71 bone vibrator in a sound treated audiometric room with noise levels within the permissible limits (ANSI S3.1-1999) using modified Hughson Westlake procedure. The patient was followed up since the development of renal symptoms over the span of 2.5 years and over 6 weeks after developing sudden hearing loss. PTA was carried out three times in the 6 weeks span along with ENT medical management of the hearing loss, mainly to improve microcirculation using vasodilators group.

RESULTS

Ultrasonography showed papilliferous growth involving the right posterolateral wall of bladder base and right ureteric orifice involved in the lesion. Histopathological reports made the final diagnosis of Cystitis Glandularis. The patient was treated for the same with right stenting and anti inflammatory drugs.

Table 1 Ionic concentration in the blood

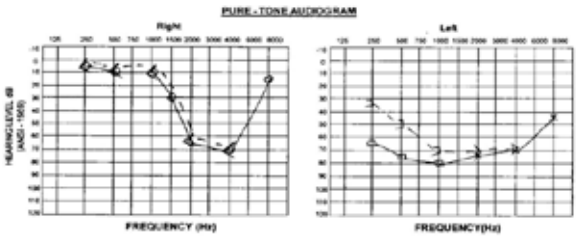
Blood test	Sodium levels	Potassium levels
First testing	139mEq	4.7mEq
Second testing	138mEq	3.6mEq

Impression: Increased sodium and potassium levels in the blood.

Table 2 Thresholds in the first PTA testing.

Ear	250 Hz		500Hz		1kHz		2kHz		4kHz		8kHz	
	AC dBHL	BC	AC dBHL	BC	AC dBHL	BC	AC dBHL	BC	AC dBHL	BC	AC dBHL	BC
Right	5	5	10	10	10	10	65	65	70	70	55	-
Left	65	35	75	50	80	70	75	70	70	70	45	

Figure 1 Pure tone audiogram of first evaluation.



Impression: Right ear- Moderately severe high frequency sensorineural hearing loss.

Left ear- Severe sensorineural hearing loss.

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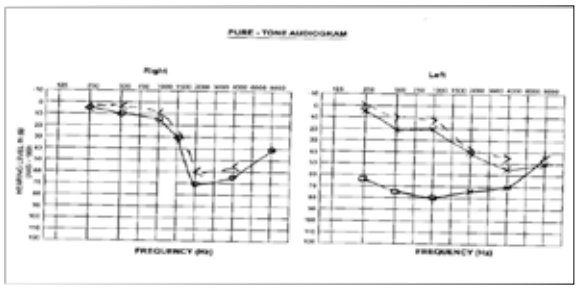
Table 3 Pure tone audiometry in the second evaluation (Follow up post 2 weeks of medical management).

Ear	250 Hz		500Hz		1kHz		2kHz		4kHz		8kHz	
	AC BC dBHL		AC BC dBHL		AC BC dBHL		AC BC dBHL		AC BC dBHL		AC BC dBHL	
Right	5(5)	5(5)	10(10)	5(10)	15(10)	10(10)	70(65)	60(65)	65(70)	55(70)	40(55)	-
Left	5(65)	5(35)	20(75)	10(50)	20(80)	15(70)	40(75)	40(70)	55(70)	45(70)	40(45)	-

***bracketed are the thresholds obtained in the previous evaluation.**

Figure 2 Pure tone audiogram of second evaluation.



Impression: Right- Moderate High frequency sensorineural hearing loss

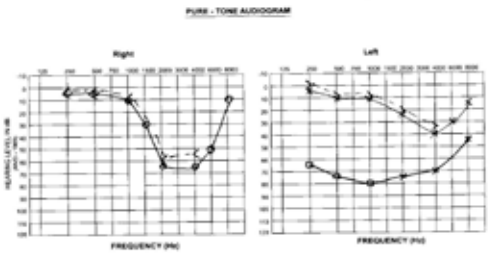
Left- Mild High frequency sensorineural hearing loss.

Table 4 Pure tone audiometry in the third evaluation (Follow up post 6 weeks of medical management).

Ear	250 Hz		500Hz		1kHz		2kHz		4kHz		8kHz	
	AC BC dBHL		AC BC dBHL		AC BC dBHL		AC BC dBHL		AC BC dBHL		AC BC dBHL	
Right	5(5)	5(5)	10(10)	5(5)	15(15)	10(10)	70(70)	60(60)	65(65)	55(55)	40(40)	-
Left	5(5)	5(5)	10(20)	10(10)	10(20)	10(15)	25(40)	20(40)	40(55)	35(45)	15(40)	-

***bracketed are the thresholds obtained in the previous evaluation.**

Figure 3 Pure tone audiogram of the third evaluation.



Impression: Right ear- Mild to moderate High frequency sensorineural hearing loss.

Left ear- Mild sloping High frequency sensorineural hearing loss.

DISCUSSION

Cause of SSNHL can be found only in 10 to 20% of cases (3) which leads to trial and error management strategy or a global management strategy. Thus, in the above mentioned case, the probable pathophysiology causing SSNHL was probed into.

Endocochlear potentials

Endocochlear potential which is considered prime for hearing is generated at the scala media of the cochlea . The ionic potentials are derived from vascular rich stria vascularis. The resting potential of +80mV which is caused by the potential difference created by the sodium potassium ATPase pump. When the blood rich in Sodium and Potassium ions reaches Stria vascularis, these ions diffuse into the cochlear fluids of perilymph (rich in sodium) and endolymph (rich in potassium) with the help of ATPse pump. This potential difference is vital for hearing without which action potential generation doesn't happen and thus no hearing (4).

Probable Pathophysiology.

CKD hinders the normal physiology of the renal systems namely Counter current multiplier, Counter current exchanger and Juxta glomerular apparatus physiology thereby majorly disturbing the ionic distribution in the body. When the overall sodium levels increase in the blood owing to the pathophysiology effect which isn't ideal, leads to an overall vasoconstriction, total peripheral constriction and increased cardiac output.

When the Juxta glomerular apparatus malfunctions, there is excess renin release which in turn might increase the conversion of angiotensin II. This ultimately acts on adrenal cortex to increase aldosterone which increases distal nephron reabsorptions thereby increasing salt retention and potassium excretion. It increases vasopressin levels, acts on the renal and intestinal systems to increase salt retention, acts on cardiac system to increase cardiac output and acts on circulatory system to cause peripheral vasoconstriction (5).

The overall increase in the sodium levels in the blood in turn causes secondary hypertension and may also cause blood vessel rupturing reducing the blood supply to the peripheral organs supplied through the microcirculatory systems, namely cochlea and thereby disrupting the endocochlear potential generation. This fact holds good with the reported case which can be observed with the increased sodium levels and reduced potassium levels in the blood as shown in table 1. Findings of people with increased sodium levels in blood having hypertension only strengthen our finding (6). If autoregulatory vasoconstriction which usually occurs when the blood volume increases contributed to the peripheral vasoconstriction is query, which had been a factor contributing would have only added on to the pathophysiological mechanisms.

When the client was subjected to medical management to improve microcirculation and vasodilator , the hearing recovered completely in the left ear, which to be precise had improved by 50 to 60dBHL from 250Hz to 1 kHz, 35dBHL at 2 kHz and 15dBHL at 4 kHz but hearing didn't recover in the right ear as shown in Figure 3 and table 4. This could be attributed to the delay in the initiation of the medical management (2 months after the onset) of ISSNHL as treatment initiated even a week post onset led to very less percentage of patients recovering (7) and also to the lack of availability of pre morbid hearing thresholds. The recovery of hearing in ISSNHL especially with a vascular cause is poorly explained in the literature and highly hypothesized. However, it can be believed that once the blood supply comes back to normal levels, the generation of endocochlear potentials takes place which otherwise is just enough for the anatomical sustenance of the cochlear structures (8).

CONCLUSION

ISSNHL is a complex condition which can be caused by various reasons; vascular cause is considered to be one among the major causes.

Current case had developed ISSNHL after 2 years of CKD and didn't recover in 2 months. When the CKD was probed into, the possible pathophysiology surfaced and when treated for the vascular cause, the client's left ear improved significantly within 6 weeks. However, hearing loss in right ear remained uncured. This directs to the strengthening of the already in-practice protocol of prolongation of Audiological management when medical management is being provided and hearing prostheses shall be an option only after 6 months of medical management shows no improvement. This case also provides basic evidence that the human network physiology is interlinked to one another and chronic renal problems could be a potential cause for ISSNHL. This condition should be studied under wide population for ascertaining the hypothesis.

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