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A Case of Alcohol Induced Peripheral Neuropathy with Bilateral Sensory-Neural Hearing Loss

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Abstract: *Our patient, a 32 years old male presented with chief complains of progressive bilateral hearing loss since last 8 months, burning and tingling sensation (paresthesia) in bilateral lower limbs and difficulty in walking gradually worsening since last 4 month. Clinical examination revealed loss of pain, temperature, fine touch, joint position and vibration sense in bilateral lower limbs below ankle. Deep tendon reflexes were absent, power and tone were maintained in all the limbs. Clinically, patient was diagnosed to have bilateral hearing loss with sensory neuropathy. Only positive history is of patient being a hypertensive since 4 years on tablet Telmisartan and a chronic alcoholism since 8 to 10 years in abstinence since 4 months. Nerve conduction study revealed predominantly sensory, lower limb, Axonal peripheral neuropathy affecting large fibers. Neuro-imaging (MRI brain - plain) revealed no abnormality. ENT examination and hearing assessment concluded bilateral sensory-neural hearing loss. After excluding all possible causes, toxic neuropathy wasconsidered. Sensory-neural hearing loss is uncommon in alcoholic peripheral neuropathy, and we suggest hearing assessment in all patients with peripheral neuropathy, especially of toxic etiology.*

Key words: *Alcoholism, Alcohol abuse, Peripheral Neuropathy, Alcoholic Neuropathy, Sensory-Neural Hearing loss.*

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I. Introduction

Alcohol is the most common abusedsubstance in the world. After ingestion of alcohol, it is distributedin all body tissues and also crosses the blood-brain barrier. Ethanol abuse thus contributes to damage in a variety of tissues including liver, the central and peripheral nervous systems and skeletal and cardiac muscles. Alcohol induced peripheral neuropathy is a potentially incapacitating complication of long-term excessive consumption of alcohol. It is characterized by pain and dysesthesias, primarily in lower extremities and is poorly relieved on available therapy[1-2]. Alcoholic neuropathy is associated with several risk factors such as malnutrition, thiamine deficiency, and direct toxicity of alcohol[3-4].

Scientific studies employing clinical and electro-diagnostic criteria have estimated that neuropathy is present in 25-66% of defined 'chronic alcoholics'. The factors most directly associated with the development of alcoholic neuropathy include the duration and amount of total lifetime alcohol consumption[4]. It occurs in those patients consuming 100 grams of alcohol daily for several years. Neuropathy is more prevalent in frequent, heavy and continuous drinkers compared to more episodic drinkers.

A review of international literature on the effects of chronic alcoholism on hearing revealed a paucity of studies. Alcoholism related sensory-neural hearing loss has been documented; the methods however raise concerns about whether variables such as age, duration of alcohol abuse, and past and present noise exposure might also have contributed to the observed hearing loss.

Case Report:

A 32 years old man was admitted with complains of gradually progressive hearing loss since last 8 months, bilateral severe burning and tingling sensation in lower limbs and upper limbs (lower limb more than upper limb, and in upper limb more on the right side)since last 4 months, and difficulty in walking since last 4 months. His symptoms have been gradually worsening over last 4 months. His medical history involves Hypertension since last 4 years for which he on regular medication of Tablet Telmisartan. Patient is a chronic alcoholic since last 8 – 10 years and is in abstinence since last 4 months. Patient was taking approximately 1 liter of alcohol per day and was a daily drinker.

Patient's birth was normal with normal milestone of development. His parents denied any history of hearing loss in the family, and also denied any trauma, seizures or infections to the patients in the childhood. Patient also denies any recent history of Trauma, Loss of consciousness, headaches, or vomiting episodes. Patient also denies history of fever or any infections before the onset of complains. He had normal bowel and bladder habits. Patient is a vendor of vegetables and is not exposed to unsafe levels of noise. Patient also does

not give history of acoustic trauma. Patient is a non-diabetic. Patient does not give history of foot drop or loss of any power in upper or lower limb.

On clinical examination, vital signs and body temperature were normal. All the sensation including pain, temperature, fine touch, joint position sense, and vibration were lost in bilateral lower limbs below ankle. There is no muscle wasting and tone and power of the upper and lower limbs is maintained. Bilateral deep tendon reflexes were absent and bilateral plantar reflex have flexor response. Patient has high stepping and wide based gait with sensory ataxia. There is no postural drop in blood pressure and no excessive sweating.Clinical evaluation of cardiovascular, respiratory system and abdomen were normal.

Hematology and biochemistry including serum electrolytes, lactate levels, liver function tests, and thyroid profile were within normal range. ANA was also negative. Serum protein electrophoresis of the patient was normal and showed no M band. Radio-diagnostics involved Ultrasonography which did not reveal hepatomegaly or liver parenchymal disease. MRI scan of Brain revealed a normal study with no cerebellar involvement (degeneration) or CP angle SOL. Nerve Conduction Velocity study gave low amplitude responses giving impression of generalized, symmetrical, predominantly sensory, lower limb > upper limb, axonal peripheral neuropathy affecting large fibers (Fig 1).

ENT evaluation concluded intact Tympanic Membrane bilaterally. Pure Tone Audiometry and Brainstem Evoked Response Audiometry revealed bilateral sensory-neural hearing loss quantifying right side as severe and left side as moderately severe (Fig 2). Transient Evoked Oto-Acoustic Emission (TEOAE) noted an absence of bilateral oto-acoustic emission (OAE) response (Fig 3).

Sensory NCS

Nerve / Sites	Rec. Site	Ampl. µV	Lat. ms	Dist. cm	Vel. m/s	Dur. ms
L SURAL - Lat Malleolus						
Mid Calf	Ankle	NR	NR	14	NR	NR
R SURAL - Lat Malleolus						
Mid Calf	Ankle	NR	NR	14	NR	NR
L SUP PERONEAL - Foot						
Lateral Leg	Foot	NR	NR	14	NR	NR
R SUP PERONEAL - Foot						
Lateral Leg	Foot	NR	NR	14	NR	NR

Sensory NCS

Nerve / Sites	Rec. Site	Latency ms	Peak Ampl µV	Distance cm	Velocity m/s
R MEDIAN - Index finger					
Wrist	II	2.34	9.1	14.5	61.9
R ULNAR - Little finger					
Wrist	V	2.40	7.5	12	50.1
R RADIAL					
Forearm	Thumb	2.45	9.2	14	57.2

Motor NCS

Nerve / Sites	Latency ms	Ampl mV	Distance cm	Velocity m/s
R MEDIAN - APB				
Wrist	3.13	8.7		
Elbow	8.59	6.6	26	47.5
R ULNAR - ADM				
Wrist	3.02	9.3		
Below .Elbow	7.60	8.5	24.5	53.5
R COMM PERONEAL - EDB				
Ankle	4.95	2.9		
Fib Head	12.14	2.7	30	41.7
L COMM PERONEAL - EDB				
Ankle	4.53	2.2		
Fib Head	11.30	1.9	31	45.8
R COMM PERONEAL - Tib Ant				
Fib Head	3.70	4.9		
L COMM PERONEAL - Tib Ant				
Fib Head	2.08	4.1		
L TIBIAL (KNEE) - AH				
Ankle	5.42	7.9		
Knee	14.74	6.2	40	42.9
R TIBIAL (KNEE) - AH				
Ankle	4.84	6.4		
Knee	13.65	4.5	40	45.4
L TIBIAL (KNEE) - Gastroc				
Ankle	5.00	13.6		
R TIBIAL (KNEE) - Gastroc				
Ankle	3.80	13.9		

FIGURE 1

Nerve Conduction Report: Generalized, symmetrical, predominantly sensory, Axonal peripheral neuropathy affecting large fiber. Lower limb is affected more than upper limb.

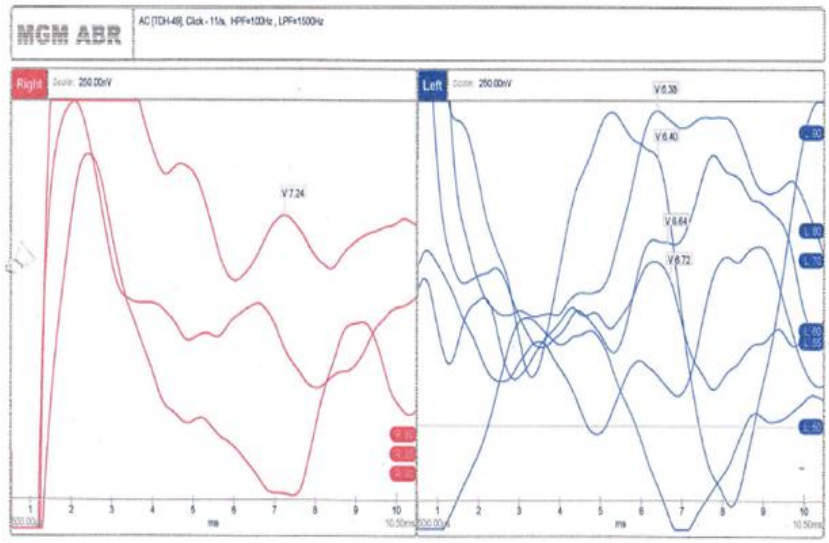

FIGURE 2

BERA: Done in awakening status, each ear tested separately by click stimulus. V peak found in both ears at highest intensity level, traced up to 60dBnHLin left ear and 90dBnHL in right ear

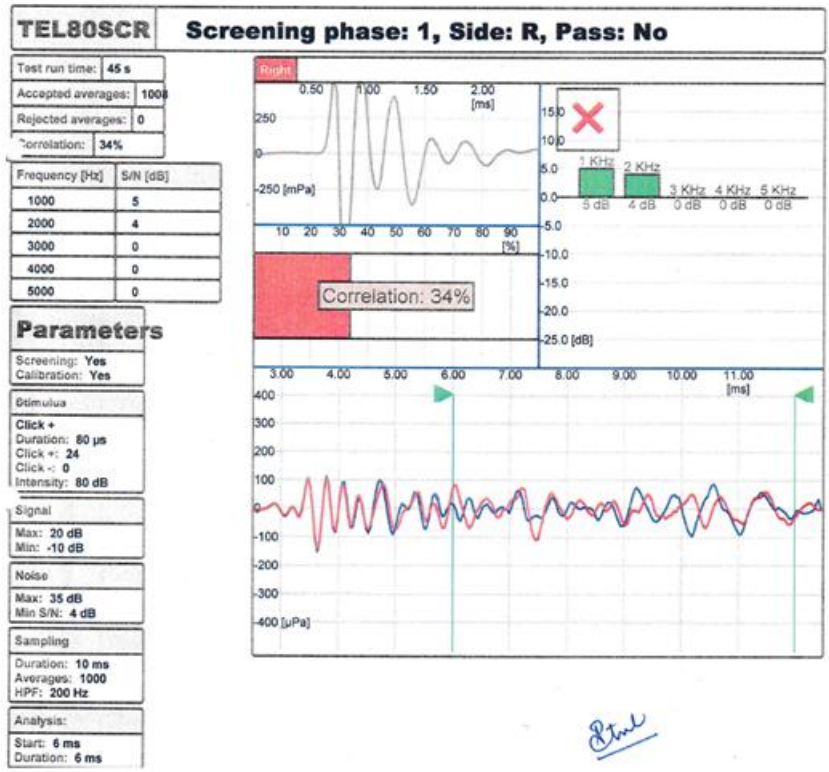

FIGURE 3

Transient Evoked Oto-Acoustic Emission (TEOAE) :- Absence of oto-acoustic emission (OAE) response

II. Discussion

Peripheral neuropathy is damage to or disease affecting nerves, which may impair sensation, movement, gland or organ function, or other aspects of health, depending on type of nerve affected. According to the pathology, it is classified in to Axonal or Demyelinating peripheral neuropathy. Axonal neuropathy is gradual on onset, sensory more than motor involvement, with preservation of most deep tendon reflexes (DTRs). Recovery takes years with some form or residual deformity. NCV usually shows decreased amplitude with near normal conduction velocity. Demyelinating peripheral neuropathy is usually acute onset with motor more than sensory involvement. DTRs are all absent and NCV shows decreased conduction velocity with increased latency. Recovery is rapid and complete.

In our patient of Axonal peripheral neuropathy with bilateral sensory-neural hearing loss, multiple differential diagnoses were considered before the final diagnosis of alcohol induced peripheral neuropathy with bilateral sensory neural hearing loss was made. This included genetic causes, diabetes, drug and toxin induced neuropathy, mitochondrial disorders, vasculitis, CP angle tumors, and CIDP.

Charcot Marie Tooth disease/Hereditary sensory motor neuropathies are most common inherited neuromuscular disorder characterized by inherited neuropathies without known metabolic derangements.CMT diseases have a significant family history (can be autosomal dominant or recessive). The age of presentation varies, depending on the type of CMT disease. Patient initially presents with distal and sometime proximal muscle weakness resulting in characteristic stork leg or inverted champagne bottle appearance. Though nerve deafness is a characteristic, other characteristics such as cataract, retinitis pigmentosa were not present in our patient on ophthalmic examination. Echocardiography was within normal limits and there was no evidence of cardiomyopathy. Patient also has improvement over the course of hospitalization with excludes CMT.

Diabetic Neuropathy is the most common complication of diabetes mellitus. Manifestations include symptoms of somatic and/or autonomic nervous system. Clinical features include pain and paresthesia, postural hypotension, diarrhea, abdominal bloating, erectile dysfunction, etc. Fasting and post prandial blood sugar levels, as well as glycosylated hemoglobin levels in our patient were in normal range and he had no family history of diabetes, ruling out diabetes as the cause of peripheral neuropathy and bilateral hearing loss.

Drug Induced / Toxic Neuropathydevelop as a complication of toxic effect of various drugs and other environmental exposures. Some of the frequently used drugs causing neuropathy include anti-cancer drugs, anti-epileptics, anti-tubercular drugs such as Isoniazid and ethambutol, chloroquine and hydroxychloquine, colchicine, dapsone, lithium, etc. Patient has no prior history of intake of above mentioned drugs. Patient also gives no occupational exposure to heavy metals like lead, mercury, arsenic.

Mitochondrial Diseases are often associated with peripheral neuropathies which are heterogeneous in their clinical, neurophysiological, and histopathological characteristics. Mitochondrial diseases often present during first or second decade of life. Signs and symptoms include loss of muscle coordination, muscle weakness, visual problems, hearing problems, seizure disorder, and weight loss.
With normal serum lactate level and absence of above mentioned symptoms, mitochondrial pathology seemed unlikely.
Cerebellar ataxia (degeneration), CP angle tumors were ruled out with a normal MRI brain study. Cyanocobalamin (B12) deficiency was ruled out with absence of anemia, normocytic RBCs, and absence of UMN signs. Hypothyroidism as a cause was also ruled out. Laboratory tests for HIV infection were nonreactive, and history or taking anti-retroviral drugs was also eliminated.

Mechanism of Hearing Loss is still unclear in alcoholic neuropathy. Studies in the literature that studied alcoholic ototoxicity have suggested that toxic effect of alcohol occurs in the basal portion of the cochlea, where maximum vibration of the basilar membrane due to high frequency sounds takes place (5). Oto-acoustic emissions are a function of external hair cells of organ of Corti, thus absent emissions point towards involvement of cochlea.

Management of alcoholic peripheral neuropathy includes symptom control as there is not definitive treatment.Gamma aminobutyric acid (GABA) analogue Gabapentin has proven helpful in cases of neuropathic pain. Use of amitriptyline for chronic and neuropathic pain is recommended.Nutritional therapy with parenteral multivitamins is beneficial to implement until the person can maintain adequate nutritional intake.Treatments also include vitamin supplementation (especially thiamine).In more severe cases of nutritional deficiency 320 mg/day of Benfotiamine for 4 weeks followed by 120 mg/day for 4 more weeks may be prescribed.*Comprehensive physical therapy* for patients with alcoholic neuropathy may include Gait and balance training, Range of motion (ROM) exercises and stretching, and Strength training of weakened muscles.

Prognosisof alcoholic peripheral neuropathy is good provided the patient stops drinking. Rehabilitation and abstinence results in marked clinical improvement within 6 to 9 months, which include good ambulation, and marked reduction in sensory symptoms and ataxia.

III. Conclusion

Our patient is a case of chronic alcoholism induced peripheral neuropathy with sensory-neural hearing loss. Hearing loss was also attributed to alcohol abuse as all the other causes of bilateral hearing loss were ruled out and the findings of battery of tests performed are in concurrence with findings in literatures of ototoxicity due to alcohol abuse. We suggest hearing assessment in all patients of peripheral neuropathy, especially those secondary to chronic alcoholism enabling early diagnosis and management.

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