A Case Study Of Dietary Deficiency On Peripheral Nerve Functions In Chronic Alcoholic Patient

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Abstract: Alcoholic neuropathy is most likely result of dietary deficiency rather than direct neurotoxic effect of alcohol. A male alcoholic patient, aged 34-years old with clear clinical sign of peripheral neuropathy was examined after his habit of six years chronic alcoholic drinking. Conduction velocities, latencies and nerve action potential amplitudes was measured from median, radial, common peroneal and sural nerves on respective upper and lower limb and the results showed that there was decrease in conduction velocity of common peroneal, and posterior tibial in lower limbs. However, sensory nerve conduction (SNCV) of sural nerve (right and left) was normal in lower limb. Based on the results observed in our study, we conclude that the combination of vitamin B_{12}, uridine, and cytidine can be safe and effective in the treatment of patients presenting alcoholic polyneuropathy. So the prognosis of alcoholic peripheral neuropathy is good and independent of age provided that intake of alcohol is withdrawn completely.

Keyword: Alcoholic, Neuropathy, Vitamin-B_{12}, Nutrient.

Introduction:

Alcohol is one of the most commonly used substances in the world. After excess consumption, alcohol distributes throughout body tissues and rapidly crosses the blood-brain barrier. It is not surprising that ethanol abuse significantly contributes to damage in a variety of tissues including liver, the central and peripheral nervous systems, and skeletal and cardiac muscle. The factors most directly associated with the development of alcoholic neuropathy include the duration and amount of total lifetime alcohol consumption. Alcoholic peripheral neuropathy is a potentially incapacitating complication of long-term excessive consumption of alcohol characterized by pain and dysesthesias, primarily in the lower extremities [1]. Alcohol related neuropathy is associated with several risk factors, such as malnutrition, thiamine deficiency, direct toxicity of alcohol and a family history of alcoholism [2]. Vitamin B12 deficiency has been associated with significant neurological pathology, including peripheral neuropathy [3].

Case history:

We reported one case of alcoholic neuropathy, which are most likely result of dietary deficiency rather than direct neurotoxic effect of alcohol. A patient of 34-years old had type-2 diabetes (DMT2) from past five years, somehow his diabetes was under control due to proper medication , but he had continuous habit of taking alcohol from past six-years. He was referred to hospital for GI infection treatment, where he got admitted. He gave the history of constipation, abdominal colic and occasional muscle and joint aches with fatigue. After few days later, he started noticed some burning and tingling sensation in his lower limb. His was felling abdominal tenderness; other physical examination was normal. His thyroid level and pulses was normal. He was not depressed. He denied chest pain or shortness of breath. He denied any other symptoms and had no fever or chills, cough, bloody stools, or hematuria. We observed clinically the bulk, tone and strength of muscle for both upper and lower limb. However, there was decrease in bulk of muscle, muscle strength, weakness with muscle atrophy in both upper and lower limb, and increase in muscletone in lower limb as compare to upper limb. On subsequent days the weakness was more progressed in lower extremities. There was diffuse weakness in lower extremities (Figure 1), distal was greater (grade 3**)** than proximal (grade 4). Gait was normal. His cranial nerve exam was intact. His sensation was intact on both upper and lower extremities. Evaluation of his blood glucose level revealed normal fasting and postprandial blood glucose levels. Laboratory studies revealed normal chemistries of His HbA1c 5.2% (normal 4.0–6.0%). The blood count, showed there was increased in ESR (26mm/hr) and WBC count (17000/mm^3), which may be due to presence of GI infection. Deficiency of vitamin B_{12} (11.20 nmol/L) along with macrocytic anemia was observed in peripheral blood smear while folic acid content was normal in blood. Serum calcium level was also normal. A complete lipid panel, liver screening, and a renal profile were all normal.

Neurological examination indicated: The patient was alert and oriented with intact speech and memory, pupils equally reactive to light. *Electrodiagnostic investigation*: Nerve conduction study (NCS) was carried out in a quiet room of neurophysiology laboratory at a temperature of 26^0 to 30^0C by using Neuroperfect-2000. Skin temperatures were recorded and maintained above 32°C for all recordings. Nerve conduction studies were performed using standard techniques of supramaxima1 percutaneous stimulation and surface recording. The nerves (Common peroneal, posterior tibial for motor and sural for sensory) in lower limb were stimulated sub-cutaneous along their course where they are relatively superficial. The skin resistance was reduced by rubbing with spirit swab; the active electrode was placed over muscle belly and reference electrode over tendon. Amplitudes of compound muscle action potentials (CMAPs) were measured from baseline to negative peak and were reported for stimulation at distal and proximal sites; conduction velocity was measured in the lower limb. Evidence of abnormal temporal dispersion was estimated by comparing proximal and distal CMAP amplitudes, F response latencies were measured as the minimal latency in a series of F responses following distal (wrist or ankle) motor nerve stimulation. Sensory nerve action potential (SNAP) amplitudes were measured peak to peak. Nerve conduction studies showed, abnormal neurological examination, sensory nerve conduction (SNCV) to be normal in median (right and left) and ulnar nerve (right and left) of upper limb (Table 1 & 2) Whereas there was decrease in motor nerve conduction (MNCV) in common peroneal (right and left), tibial nerve (right and left) of lower limb. However, sensory nerve conduction (SNCV) of sural nerve (right and left) was normal in lower limb

(Table 1 & 2). F response latency was also prolonged for common peroneal (right and left), and tibial nerve (right and left) of lower limb (Table 3).

Discussion:

We observed Vitamin B_{12} deficiency in patient. In the early stages of alcoholic neuropathy, patients complain of pain in the extremities, which may be severe and has been described as burning or 'like tearing. Vitamin B_{12} deficiency has been associated with peripheral neuropathy [4]. The human body requires vitamin B_{12} in order to maintain a variety of processes vital to health and maintenance among which are cell reproduction and growth, and nucleoprotein and myelin synthesis [5]. In the nervous system, vitamin B_{12} plays a role in nerve metabolism via the remethylation of homocysteine to methionine for de novo synthesis of sadenosylmethionine [6]. One of the mechanisms believed to be at play in vitamin B_{12} deficiency neuropathy is hypomethylation in the central nervous system. Inhibition of the B_{12}- dependent enzyme methionine synthase results in a fall in the ratio of S adenosylmethionine (SAM) to Sadenosylhomocysteine [7] and the resultant deficiency in SAM impairs methylation reactions in the myelin sheath. In deficiency states, neurological impairment is caused by interruption of myelin formation [8]. This may link to the absence of s-adenosylmethionine formation [9]. This impairs Myelin sheaths formation which has important role in regulation of nerve fiber conduction velocity [10].

Limitation of the Study:

Limitation of this finding can be overcome by testing serum metabolites such as methylmalonic acid and homocysteine can help in further diagnosis. Since chronic alcoholism can alter the intake, absorption and utilization of various nutrients (vitamin B2, vitamin B6, or vitamin E). These vitamin deficiency levels can be also checked. Needle electromyography (EMG) can also done, which measures the electrical signals in muscles and nerve biopsy to find out axonal neuropathy with reduced nerve fibre densities.

Conclusion:

Based on the results observed in our study, we conclude that the combination of vitamin B_{12}, uridine, and cytidine can be safe and effective in the treatment of patients presenting alcoholic polyneuropathy. Treatment with the intramuscular injection form followed by oral treatment reduced pain in relation to pretreatment values, increased vitamin B_{12} levels and improved motor coordination among affected patients. Several alcoholics showed normal or nearly normal scores in electroneurographical and clinical examination if they had all managed to stop drinking alcohol. So the prognosis of alcoholic peripheral neuropathy is good and independent of age provided that intake of alcohol is withdrawn completely.

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Conflict of interest

The authors declared no conflict of interest.

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TABLE 1: *MNCV (Upper and Lower limb)*

NERVE	Rec – Stim Site	Distance (mm)	Latency difference(ms)	NCV(m/s)
Rt. CPN	EDB- ANKLE EDB-FIB.HEAD	75 420	3.0 15.25	25.00 27.54
Lt. CPN	EDB- ANKLE EDB-FIB.HEAD	80 430	4.18 13.10	19.13 32.82
Rt. PTN	Abd. Halls- ANKLE Abd. Halls- POP. FOSSA	110 420	3.88 14.12	28.35 29.74
Lt. PTN	Abd. Halls- ANKLE Abd. Halls- POP. FOSSA	100 410	4.50 12.45	22.22 32.93
Rt. Median	APB-WRIST APB-ELBOW	90 240	2.20 4.15	40.90 57.83
Lt. Median	APB-WRIST APB-ELBOW	75 245	2.50 4.12	30.00 59.46
Rt. ULNAR	ADM-WRIST ADM-ELBOW	80 250	2.70 3.97	29.62 62.97
Lt. ULNAR	ADM-WRIST ADM-ELBOW	75 240	2.95 4.30	25.42 55.81

TABLE 2: *SNCV (Upper and Lower limb)*

NERVE	Rec – Stim Site	Distance (mm)	Latency difference (ms)	NCV (m/s)
Rt. SURAL	Laterals Malls-MID CALF	175	2.88	60.76
Lt. SURAL	Laterals Malls- MID CALF	170	2.65	64.15
Rt. Median	2nd Digit -WRIST	140	1.80	77.77
Lt. Median	2nd Digit- WRIST	135	1.70	79.41
Rt. ULNAR	5th Digit - WRIST	145	1.75	82.85
Lt. ULNAR	5th Digit - WRIST	125	1.65	75.75

TABLE 3: *F WAVE (Upper and Lower limb)*

NERVE	Distance (mm)	Latency difference (ms)	Velocity (m/s)
Rt. CPN	95	25.43	3.73
Lt. CPN	85	22.25	3.80
Rt. PTN	120	24.70	4.85
Lt. PTN	110	21.26	5.17
Rt. Median	100	10.50	9.52
Lt. Median	110	11.63	9.45
Rt. ULNAR	105	9.50	11.05
Lt. ULNAR	100	10.13	9.87

Figure 1: *An alcoholic patient with muscle atrophy.*