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Original Research Article

Nerve Conduction Study in Vitamin B 12 Deficiency Patients

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Abstract:
Vitamin B12 deficiency is clinically recognized to be associated with neurological disorders, such as dementia, cognitive impairment, and depression. B12 deficiency may cause demyelination of nerves in the peripheral and central nervous system and has been associated with peripheral neuropathy, loss of sensation in peripheral nerves, and weakness in lower extremities in older adults. 30 Vitamin B12 deficient subjects and 30 normal subjets as control were included in study. Nerve conduction study was done using the AD instrument–Nicolet in both groups. Comparison of sensory nerve parameters in Control Group and Vitamin B12 deficient subjects, there is highly significant slowing of sensory nerves median & ulnar ($p<0.001$) and significant decrease in amplitude of Median nerve and ulnar nerve ($p<0.01$) in Vitamin B12 deficient subjects compared to controls. Early diagnosis of neuropathy due to Vitamin B12 deficiency is important, because a myriad of adverse outcomes that can be progressive and irreversible neurological abnormalities can be averted with substitutive treatment.
Keywords: Vitamin B 12 deficiency, Nerve conduction.

Introduction

Vitamin B-12 deficiency is often considered synonymous with pernicious anemia, a rare condition manifesting as megaloblastic erythropoiesis, hyperhomocystinemia, methylmalonic acidemia, and neurological syndromes (subacute combined degeneration of cord, peripheral neuropathy, dementia and depression) [1]. The primary defect in this condition is a near-total block in the gastrointestinal absorption of vitamin B-12 which requires high-dose parenteral treatment. In developing countries such as India, inadequate dietary intake of B-12 due to socio-cultural factors leads to widely prevalent asymptomatic low B-12 status. In this scenario, lower doses of oral B-12 may be effective, safer and more affordable. In the last few decades it has become increasingly apparent that inadequate dietary intake of vitamin B-12 is a widely prevalent cause of low vitamin B-12 status in many populations [2]. This predominantly affects vegetarians who do not eat adequate amounts of animal origin foods (milk, eggs, fish, chicken and meat) and lower socioeconomic groups who cannot afford them and vegans. Small doses of oral vitamin B-12 may be adequate to improve vitamin status in these individuals [3,4]. There is sparse information on this issue. There are many different causes of B12 deficiency in older adults. More than half of older adults with B12 deficiency have food-cobalamin malabsorption, defined as impaired digestion and absorption of protein-bound B12 [5,6,7]. Other causes of low or deficient B12 are insufficient intake either from diet or supplements, pernicious anemia, gastric surgery, gastrointestinal disease, and certain medications (e.g., proton-pump inhibitors, metformin)

Vitamin B12 deficiency is clinically recognized to be associated with neurological disorders, such as dementia, cognitive impairment, and depression [8,9]. B12 deficiency may cause demyelination of nerves in the peripheral and central nervous system and has been associated with peripheral neuropathy, loss of sensation in peripheral nerves, and weakness in lower extremities in older adults [10,11,12]. In particular, vitamin B12 deficiency is associated with large fiber (type A) neuropathy; type A nerve fibers act as both sensory and motor fiber [13]. Thus, vitamin B12 may be associated with both sensory and motor peripheral nerve function. The objective of the present study was to evaluate the association between vitamin B12 status and peripheral nerve conduction parameters in middle aged adults.

Material and Methods

30 middle adults (both males and females) aged between 28 to 45 years were recruited after obtaining written informed consent. None of the 30 control subjects reported any symptoms suggestive of peripheral neuropathy. Subjects who underwent surgery or history of fracture or scars at the sites of

stimulation were excluded. 30 subjects with symptoms of vitamin B12 deficiency were included as patients. Following recruitment, each subject underwent anthropometry, nerve conduction assessment, vitamin B12 estimation and complete hemogram. Vitamin B12 was determined.

Nerve Conduction Study (NCS): Nerve conduction study was done using the AD instrument –Nicolet. For motor & sensory nerve study, output range was 20mA,max repeat rate 1Hz,pulse width 0.05ms stimulation with current ranging between 10-20mA was applied with increasing strength until desired response was obtained. Sensory nerves tested were Median, and Ulnar nerve.

Stimulation and Recording Sites of Sensory Nerves:

Sensory Nerve: Median.
Method of stimulation: Orthodromic.
Stimulation site: Index finger **Recording site:** Middle of the wrist.

Sensory Nerve: Ulnar.
Method of stimulation: Orthodromic.
Stimulation site: Little finger.
Recording site: Medial wrist.

Sensory nerve conduction study was done:
Parameters Assessed:

1. Sensory nerve action potential (SNAP) Amplitude.
2. Sensory nerve conduction velocity.

Statistical Analysis: Statistical analysis was done using SPSS version18 (software statistical package social science). Students unpaired t test was used to compare Nerve conduction parameters between the study and control groups . p value was calculated.
P Value <0.001 - Highly Significant.
P Value <0.01 – Significant.
P Value >0.01 - Not Significant.

Results

Table 1: Age and Vitamin B12 Levels

	Patients n=30. Mean± SD	Control n=30. Mean± SD	P value
Age in years	42.64±12.06	40.24±10.40	>0.001
Vitamin B12 levels pmol/L.	170.74±24.36	330.42±16.25	<0.001

Table 2: Nerve conduction values in vitamin B12 deficient subjects

Nerve conduction parameters	Patients n=30. Mean±SD	Control n=30. Mean± SD	P value
Median nerve			
SNCV (m/s)	40.18±4.18	62.14±3.28	<0.001
AMP (ms)	16.42±4.84	20.48±4.62	<0.01
Ulnar nerve			
SNCV (m/s)	44.24±4.68	60.28±3.98	<0.001
AMP (ms)	16.24±4.26	21.68±3.69	<0.01

AMP-amplitude, SNCV-sensory nerve conduction velocity: Table 1 shows low Vitamin B12 levels in patients which was 170.74±24.36 pmol/L(mean±Sd).

Table-2 shows Comparison of sensory nerve parameters in Control Group and Vitamin B12 deficient subjects, there is highly significant slowing of sensory nerves median & ulnar ($p<0.001$) and significant decrease in amplitude of Median nerve and ulnar nerve ($p<0.01$) in Vitamin B12 deficient subjects compared to controls.

Discussion

Vitamin B12 deficiency can occur as a result of malabsorption, gastrointestinal surgery, drugs, parasitic diseases, autoimmune diseases and genetic defects [14]. Low vitamin B-12 intake may lead to decreased bioavailability and functional deficiency of cobalamin. Although early noticeable symptoms of vitamin B-12 deficiency are nonspecific (unusual fatigue, digestion problems, frequent upper respiratory infections), the best-known clinical manifestations of cobalamin malabsorption are hematologic (pernicious anemia) and neurologic symptoms [15]. Though the pathophysiological mechanism of the neurological damage is not clear vitamin B12 deficiency leads to different pathological mechanisms in the central and peripheral nervous system [16]. Vitamin B12 deficiency should be considered in the differential diagnosis of all spinal cord, peripheral nerve, and neuropsychiatric disorders. Vitamin B12 repla cement should not be withheld from patients with borderline vitamin B12 levels, since the consequences of allowing myelopathy, neuropathy dementia, and mental disorders to worsen clearly outweigh any disadvantage of therapy [17]. The best-known neurologic manifestation of vitamin B12 deficiency is subacute combined degeneration. Isolated neuropathy or myelopathy may occur in dependently, but often appear concurrently. Peripheral neuropathy is frequently observed in symptomatic patients with vitamin B12 deficiency [18]. Lack of vitamin B12 was suggested to effect sensory nerves primarily. Nerve conduction studies of vitamin B12 deficient patients with clinically apparent neuropathy have shown decreased amplitudes of mostly sensory nerves in a majority of cases [19]. The other studies did not observe axonal

changes but observed primary demyelinating sensory neuropathy [20] whereas some studies have shown that motor nerves could also be effected due to vitamin B12 deficiency [21]. shows In our Study Comparison of sensory nerve parameters in Control Group and Vitamin B12 deficient subjects , there is highly significant slowing of sensory nerves median & ulnar ($p<0.001$) and significant decrease in amplitude of Median nerve and ulnar nerve ($p<0.01$) in Vitamin B12 deficient subjects compared to controls.

Conclusion

In conclusion, nerve conduction study may show pathological findings in neurologically symptomatic patients with vitamin B12 deficiency and therefore nerve conduction study is a method for detection of early peripheral neuropathy in vitamin B12 deficient patients. Early diagnosis is important, because a myriad of adverse outcomes that can be progressive and irreversible neurological abnormalities can be averted with substitutive treatment

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