Original Article

Peripheral Nerve Conduction and Vitamin B12 in Type 2 Diabetics with and without Neuropathy

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Abstract

Background: Diabetic peripheral neuropathy is the most common chronic complication of type 2 diabetes mellitus. B12 is needed for myelin maintenance, and its status may influence nerve function in type 2 diabetics. **Material and Methods:** This study recruited patients with type 2 diabetes at a tertiary care hospital in northern India. They were divided into two groups, patients with neuropathy, and patients without neuropathy. Body mass index, blood pressure, fasting and post prandial blood sugar, glycated haemoglobin, serum lipid profile and serum vitamin B12 were measured, and nerve conduction velocities were recorded. Groups were compared and linear regression of nerve conduction velocity on serum vitamin B12 was also performed. **Results:** Body mass index was higher in the neuropathy group, while age and blood pressure did not differ. This group had higher total cholesterol, triglyceride and low density lipoprotein cholesterol, and markedly lower high density lipoprotein cholesterol, whereas very low density lipoprotein cholesterol did not differ significantly. Glycated haemoglobin was higher and serum vitamin B12 lower in the neuropathy group. Among patients with neuropathy, serum vitamin B12 predicted conduction velocity in both motor and sensory nerves, with a stronger association for motor nerves. **Conclusion:** Serum vitamin B12 was lower in patients with diabetic peripheral neuropathy and showed a positive relationship with both motor and sensory conduction velocity, accounting for about one fifth of the variance in motor conduction.

Keywords: diabetic peripheral neuropathy; nerve conduction velocity; type 2 diabetes mellitus; vitamin B12.

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Introduction

Type 2 diabetes mellitus is a major and growing public health concern worldwide, with a particularly substantial burden in South Asia. The 11th edition of the International Diabetes Federation Diabetes Atlas estimates that 589 million adults aged 20–79 years are living with diabetes globally, including approximately 89.8 million in India.[1,2] The burden of diabetes in South Asia is further compounded by a large proportion of undiagnosed disease, highlighting the need for improved recognition and management of its chronic complications.[3]

Diabetic peripheral neuropathy (DPN) is one of the most common chronic complications of diabetes and represents an important cause of morbidity among affected individuals. It can involve progressive impairment of peripheral nerve function, leading to sensory loss, neuropathic pain and an increased risk of foot ulceration and other complications.[4,5] Early identification of factors associated with peripheral nerve dysfunction is therefore important, particularly because electrophysiological abnormalities may occur before clinically apparent neuropathic manifestations.[4,5]

Although chronic hyperglycaemia is a major contributor to diabetic nerve injury, the development of DPN is multifactorial and may involve several metabolic and vascular factors in addition to glycaemic abnormalities.[6,7] This has increased interest in potentially modifiable nutritional and metabolic factors that may contribute to peripheral nerve dysfunction.

Vitamin B12 is of particular interest because of its essential role in normal neurological function. Cobalamin acts as a cofactor in methionine and methylmalonyl-CoA metabolism, pathways that are important for neuronal function and myelin maintenance.[8] In patients with type 2 diabetes, long-term metformin therapy is also associated with reduced vitamin B12 levels, and vitamin B12 deficiency may contribute to neurological manifestations that can overlap with diabetic neuropathy.[9] Thus, assessment of vitamin B12 status may be relevant when evaluating peripheral nerve dysfunction in patients with type 2 diabetes.

Nerve conduction studies (NCS) provide an objective assessment of peripheral nerve function and can detect electrophysiological abnormalities in patients with diabetic neuropathy, including subclinical abnormalities that may precede clinically evident disease.[10]

This study aims to compare demographic, anthropometric, lipid,

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glycaemic and vitamin B12 profiles between patients with type 2 diabetes, with and without peripheral neuropathy, and to examine whether serum vitamin B12 predicts motor and sensory nerve conduction velocity in patients with neuropathy.

Materials and Methods

Study design and setting: This cross-sectional study was carried out in the Department of Physiology and the Center for Diabetes and Endocrinology of a tertiary care teaching hospital between 2024 and 2026.

Ethics: The study protocol was approved by the Institutional Ethics Committee, approval number IEC/JNMC/1405, dated 20 April 2024. Informed consent was obtained from the participants before enrolment, and participants were free to withdraw at any stage.

Selection and description of participants: The study participants were adults with previously diagnosed type 2 diabetes mellitus attending diabetes clinic at a tertiary care hospital in India. 160 patients were recruited.

Inclusion criteria: Patients were eligible for inclusion if they were aged between 18 and 60 years, had type 2 diabetes mellitus diagnosed according to American Diabetes Association criteria for a minimum of one year, and were willing to undergo nerve conduction testing. Patients with normal nerve conduction findings formed the group without neuropathy, and patients with abnormal nerve conduction findings formed the neuropathy group.

Exclusion criteria: Participants were excluded if they had type 1 diabetes, or any condition other than diabetes known to cause neuropathy, including chronic alcohol use, hypothyroidism, chronic kidney disease, hepatic failure, leprosy, human immunodeficiency virus infection, vasculitis, malignancy, exposure to neurotoxic drugs or chemotherapy, and entrapment or compressive neuropathy. Patients already receiving vitamin B12 supplementation, pregnant women, and individuals with limb deformity, amputation, or local skin lesions that would prevent nerve conduction testing were also excluded.

Anthropometry and blood pressure: Height and weight were measured, and body mass index was calculated as weight in kilograms divided by height in metres squared. Blood pressure was recorded in the sitting position after 15 minutes of rest, and the mean of three readings was used.

Biochemical measurements: Venous blood was drawn after an overnight fast of at least eight hours, and a post prandial sample was taken two hours after a standard meal. Fasting lipid profile, comprising triglycerides, total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL), was estimated via standardised homogeneous enzymatic and spectrophotometric assays. HbA1c was measured using high-performance liquid chromatography. Vitamin B12 was measured by the chemiluminescence immunoassay method using the Beckman Coulter system (UniCel dxi 800 Access Immunoassay).

Nerve conduction study: Nerve conduction studies were performed using a NeuroStim4 NCV system (Medicaid Systems, Chandigarh, India). Standard electrode placement and stimulation techniques were followed according to established electrophysiological guidelines.

Statistics: Statistical analysis was performed using GraphPad Prism 11.0 (GraphPad Software, Boston, Massachusetts, USA). Continuous variables are presented as mean ± standard deviation. Between group comparisons of continuous variables were made using Welch's t test to account for unequal variances.

The relationship between serum vitamin B12 and nerve conduction was examined by simple linear regression, with conduction velocity as the dependent variables in separate models and serum vitamin B12 in pg/mL as the independent variable. The Pearson correlation coefficient, adjusted coefficient of determination, regression coefficient with its 95 per cent confidence interval, and the F statistic with its P value are reported. A two-sided P value below 0.05 was taken as statistically significant.

Results

A total of 160 patients with type 2 diabetes mellitus were studied, comprising 120 patients with abnormal nerve conduction velocity (NCV) findings and 40 patients with normal NCV findings. Throughout the manuscript, these groups are referred to as "with neuropathy" and "without neuropathy" groups, respectively.

Demographic and anthropometric characteristics: The two groups were comparable in age, with a mean of 48.16 ± 7.34 years in the neuropathy group and 46.92 ± 6.84 years in the group without neuropathy (P = 0.3330, [Figure 1A]). Systolic and diastolic blood pressure did not differ significantly (P = 0.1650 and P = 0.3140, [Figure 1B]). Body mass index was higher in the neuropathy group at 25.53 ± 2.63 kg/m² compared with 24.04 ± 2.71 kg/m² in the group without neuropathy (P = 0.003, Figure 1C), a mean difference of about 1.5 kg/m². These findings are shown in [Table 1].

Lipid profile: The lipid measurements differed between the groups [Table 2, Figure 1D]. Total cholesterol was 171.99 ± 49.14 mg/dL in the neuropathy group against 157.21 ± 35.89 mg/dL in the group without neuropathy (P = 0.0443). Triglyceride was 195.48 ± 70.42 against 170.32 ± 40.18 mg/dL (P = 0.0062) and low density lipoprotein cholesterol was 109.82 ± 35.97 against 88.64 ± 24.36 mg/dL (P < 0.0001). Very low density lipoprotein cholesterol was also higher in the neuropathy group; however, the difference was not statistically significant (38.16 ± 13.32 against 34.05 ± 10.81 mg/dL, P = 0.0535). High density lipoprotein cholesterol was lower in the neuropathy group (24.64 ± 7.59 against 35.11 ± 6.97 mg/dL, P < 0.0001). The pattern in the neuropathy group was therefore one of raised low density lipoprotein cholesterol and triglyceride together with a substantial reduction in high density lipoprotein cholesterol, which at 10.47 mg/dL was the largest lipid difference observed.

Glycaemic control and serum vitamin B12: Glycated haemoglobin was 8.33 ± 1.51 per cent in the neuropathy group and 7.12 ± 1.42 per cent in the group without neuropathy (P < 0.001), a mean difference of 1.21 percentage points [Table 3, Figure 1E]. Fasting blood sugar was 127.10 ± 17.99 mg/dL in the neuropathy group against 114.83 ± 12.84 mg/dL in the group without neuropathy (P < 0.0001), and post prandial

blood sugar was 199.25 ± 20.72 against 186.34 ± 23.07 mg/dL (P = 0.0026, [Figure 1F]). Serum vitamin B12 was lower in the neuropathy group at 470.86 ± 233.41 pg/mL compared with 642.23 ± 249.35 pg/mL in the group without neuropathy (P = 0.0003, [Figure 1G]), a difference of about 171 pg/mL. Both group means lie within the conventional laboratory reference range.

Nerve conduction: Ulnar nerve conduction was markedly slower in the neuropathy group [Table 4, Figure 1H and 1I]. Motor conduction velocity was 48.25 ± 7.56 m/s in the neuropathy group against 61.09 ± 3.52 m/s in the group without neuropathy, a mean reduction of about 12.8 m/s (P < 0.0001). Sensory conduction velocity was 43.68 ± 5.81 m/s against 52.36 ± 2.88 m/s, a mean reduction of about 8.7 m/s (P < 0.0001). The standard deviation in the neuropathy group was roughly twice that of the group without neuropathy for both parameters, indicating a wider spread of values and a range of severity among patients with neuropathy.

Relationship between serum vitamin B12 and nerve conduction velocity: Among the 120 patients with neuropathy, serum vitamin B12 showed a positive linear relationship with both conduction parameters [Table 5]. For ulnar motor conduction velocity, the correlation coefficient was 0.46129 and the adjusted coefficient of determination was 0.20612, so the model accounted for about 21 per cent of the variance in motor conduction velocity. The regression coefficient was 0.01393 m/s for every 1 pg/mL rise in serum vitamin B12 (95 per cent confidence interval 0.00904 to 0.01882, F = 31.8834, P < 0.0001). Expressed on a more usable scale, every 100 pg/mL rise in serum vitamin B12 corresponded to a 1.39 m/s faster motor conduction velocity, with a confidence interval running from 0.90 to 1.88 m/s.

The relationship with ulnar sensory conduction velocity was in the same direction and was also significant, though weaker. The correlation coefficient was 0.31543 and the adjusted coefficient of determination was 0.09186, so about 9 per cent of the variance was accounted for. The regression coefficient was 0.00839 m/s per pg/mL (95 per cent confidence interval 0.00379 to 0.01299, F = 13.0377, P = 0.00045), equivalent to 0.84 m/s for every 100 pg/mL rise in serum vitamin B12, with a confidence interval from 0.38 to 1.30 m/s. Both confidence intervals lay clear of zero, and neither included a negative slope.

Table 1: Demographic and anthropometric characteristics of the study groups

Parameter	Without neuropathy (n = 40) Mean ± SD	With neuropathy (n = 120) Mean ± SD	P value
Age (years)	46.92 ± 6.84	48.16 ± 7.34	0.3330
Body mass index (kg/m²)	24.04 ± 2.71	25.53 ± 2.63	0.0030
Systolic blood pressure (mmHg)	124.38 ± 10.42	127.29 ± 13.89	0.1650
Diastolic blood pressure (mmHg)	81.24 ± 3.35	81.95 ± 5.04	0.3140

Table 2: Lipid profile of the study groups

Parameter	Without neuropathy (n = 40) Mean ± SD	With neuropathy (n = 120) Mean ± SD	P value
Total cholesterol (mg/dL)	157.21 ± 35.89	171.99 ± 49.14	0.0443
Triglyceride (mg/dL)	170.32 ± 40.18	195.48 ± 70.42	0.0062
Low density lipoprotein cholesterol (mg/dL)	88.64 ± 24.36	109.82 ± 35.97	< 0.0001
Very low density lipoprotein cholesterol (mg/dL)	34.05 ± 10.81	38.16 ± 13.32	0.0535
High density lipoprotein cholesterol (mg/dL)	35.11 ± 6.97	24.64 ± 7.59	< 0.0001

Table 3: Glycaemic parameters and serum vitamin B12 in the study groups

Parameter	Without neuropathy (n = 40) Mean ± SD	With neuropathy (n = 120) Mean ± SD	P value
Glycated haemoglobin (%)	7.12 ± 1.42	8.33 ± 1.51	< 0.0010
Fasting blood sugar (mg/dL)	114.83 ± 12.84	127.10 ± 17.99	< 0.0001
Post prandial blood sugar (mg/dL)	186.34 ± 23.07	199.25 ± 20.72	0.0026
Serum vitamin B12 (pg/mL)	642.23 ± 249.35	470.86 ± 233.41	0.0003

Table 4: Ulnar nerve conduction parameters in the study groups

Parameter	Without neuropathy (n = 40) Mean ± SD	With neuropathy (n = 120) Mean ± SD	P value
Ulnar motor nerve conduction velocity (m/s)	61.09 ± 3.52	48.25 ± 7.56	< 0.0001
Ulnar sensory nerve conduction velocity (m/s)	52.36 ± 2.88	43.68 ± 5.81	< 0.0001

SD: standard deviation. Values are mean ± standard deviation. Comparisons were made using Welch's t test, P value below 0.05 was taken as significant.

Table 5: Linear regression of ulnar nerve conduction velocity on serum vitamin B12 in patients with type 2 diabetes mellitus and peripheral neuropathy (n = 120)

Dependent variable	r	Adjusted R^2	β coefficient	95% CI for β	F value	P value
Ulnar motor nerve conduction velocity (m/s)	0.46129	0.20612	0.01393	0.00904 to 0.01882	31.8834	< 0.0001
Ulnar sensory nerve conduction velocity (m/s)	0.31543	0.09186	0.00839	0.00379 to 0.01299	13.0377	0.00045

CI: confidence interval. P value below 0.05 was taken as significant.

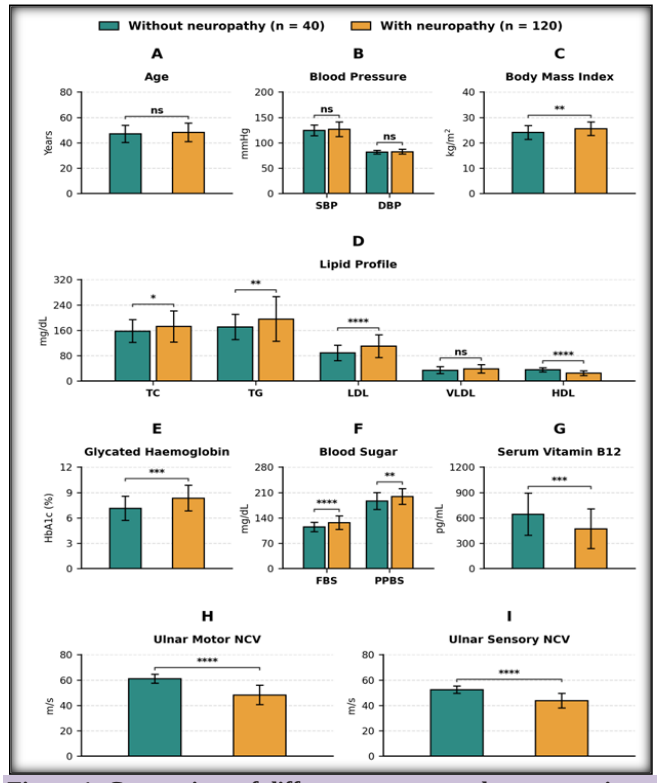

Figure 1: Comparison of different parameters between patients with type 2 diabetes mellitus with and without peripheral neuropathy. Bars represent group means and error bars represent standard deviation. (A) Age. (B) Systolic and diastolic blood pressure. (C) Body mass index. (D) Lipid profile. (E) Glycated haemoglobin. (F) Fasting and post prandial blood sugar. (G) Serum vitamin B12. (H) Ulnar motor nerve conduction velocity. (I) Ulnar sensory nerve conduction velocity. Groups were compared using Welch's t test. * P < 0.05, ** P < 0.01, *** P < 0.001, **** P < 0.0001, ns = not significant.

Discussion

The principal finding of this study was a positive linear association between serum vitamin B12 level and ulnar nerve conduction velocity among patients with type 2 diabetes mellitus with peripheral neuropathy. The association was demonstrable in both fibre types, although the motor model accounted for a substantially greater proportion of the variance in conduction velocity (adjusted $R^2 = 0.206$) than the sensory model (adjusted $R^2 = 0.092$), and the regression coefficient corresponded to a gain of 1.39 m/s in motor conduction velocity for every 100 pg/mL increase in serum vitamin B12. Secondary comparisons between the groups showed that patients with neuropathy had significantly higher body mass index, total cholesterol, triglyceride and low-density lipoprotein cholesterol, a markedly lower high-density lipoprotein cholesterol, and significantly higher fasting blood glucose, postprandial blood glucose and glycated haemoglobin than patients without neuropathy. Age, systolic blood pressure, diastolic blood pressure and very low-density lipoprotein cholesterol did not differ significantly between the groups. Taken together, these observations are consistent with the hypothesis that metabolic factors beyond glycaemia may contribute to the severity of large-fibre dysfunction in type 2 diabetes mellitus.

The observed association is biologically plausible given the role of cobalamin in two key metabolic pathways. Cobalamin-dependent methionine synthase catalyses the remethylation of homocysteine to methionine, thereby supporting the generation of S-adenosylmethionine and cellular methylation reactions.[11] In parallel, cobalamin-dependent methylmalonyl-CoA mutase converts methylmalonyl-CoA to succinyl-CoA; impaired activity results in accumulation of methylmalonic acid and has been implicated in disturbances of myelin metabolism.[12] Together, these metabolic abnormalities may contribute to impaired peripheral nerve function and reduced nerve conduction velocity. In our study, the association was stronger for motor than for sensory nerves, possibly because conduction velocity in large myelinated axons is strongly influenced by myelin integrity.

Vitamin B12 accounted for approximately one fifth of the variance in motor conduction velocity and less than one tenth of that in sensory conduction velocity, Thus, vitamin B12 should be regarded as one determinant among several, rather than a dominant determinant. Moreover, the mean concentration in the neuropathy group lay within the conventional laboratory reference interval, implying that B12 may influence nerve function even when the B12 concentration is not below the laboratory-defined normal range.

The between-group differences in adiposity and lipid fractions match with a broader literature implicating these factors in the pathogenesis of diabetic peripheral neuropathy. Although modest, the difference of 1.49 kg/m² in body mass index is consistent with a meta-analysis that identified obesity as a risk factor for painful diabetic neuropathy with an odds ratio of approximately 2.0, and with cohort data identifying weight, waist circumference and body mass index as predictors of incident neuropathy independent of age and sex.[13,14] Some researchers have argued that visceral adiposity rather than body mass index carries the greater part of this risk.[15] Low-density lipoprotein cholesterol, triglyceride and total cholesterol were also significantly raised, whereas very low-density lipoprotein cholesterol differed in the same direction without reaching conventional significance. In two cohorts of patients with type 2 diabetes, elevated triglyceride emerged as a strong predictor of incident polyneuropathy,[16] and analyses in other populations have described an association between raised low-density lipoprotein cholesterol and neuropathy that is partly mediated through glycated haemoglobin.[17] Proposed mechanisms include lipotoxic injury to Schwann cells and dorsal root ganglion neurons, signalling through receptors for oxidised low-density lipoprotein, impaired function of high-density lipoprotein particles, and microvascular compromise of the vasa nervorum.[7,18]

The present findings are consistent in direction with those reported in the existing literature. Lower vitamin B12 levels have been associated with slower nerve conduction velocity and impaired monofilament sensation in previous studies.[19]

Conclusion

In this study of patients with type 2 diabetes mellitus, those with peripheral neuropathy had higher body mass index, an unfavourable lipid profile, poorer glycaemic control and lower

serum vitamin B12 than those without neuropathy, together with markedly reduced ulnar motor and sensory conduction velocity. Within the neuropathy group, serum vitamin B12 showed a positive linear relationship with both motor and sensory conduction velocity, accounting for about one fifth of the variance in motor conduction. Moreover, higher serum vitamin B12 was associated with faster motor and sensory nerve conduction, with the association being more pronounced for motor conduction.

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Conflicts of interest
There are no conflicts of interest.

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