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Prevalence of Vitamin B12 Deficiency Among Patients Presenting With Neuropathic Symptoms: A Retrospective Cross-Sectional Analysis

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Abstract:

Background:
Vitamin B12 deficiency is a well-recognized and potentially reversible cause of peripheral neuropathy. Patients presenting with neuropathic symptoms are frequently evaluated for metabolic and nutritional causes; however, the burden of vitamin B12 deficiency in this population remains under-reported in routine clinical practice.

Objectives:
To determine the prevalence of vitamin B12 deficiency and describe its association with clinical and laboratory characteristics in patients presenting with neuropathic symptoms.

Methods:
This retrospective cross-sectional study was conducted at a tertiary care hospital and included adult patients who presented with neuropathic symptoms between January 2022 and December 2023. Based on an assumed prevalence of vitamin B12 deficiency of 30% among patients with neuropathic symptoms, a confidence level of 95%, and an absolute precision of 6%, the calculated minimum sample size was 224 patients. Demographic details, clinical presentation, comorbidities, and laboratory parameters including serum vitamin B12 levels were extracted from medical records. Vitamin B12 deficiency was defined as serum levels <200 pg/mL. Data were analyzed using descriptive statistics and comparative analysis where appropriate.

Results:
A total of 240 patients were included in the final analysis. The mean age was 49.6 ± 13.2 years, with a male predominance (58.3%). Vitamin B12 deficiency was identified in 82 patients (34.2%). Patients with vitamin B12 deficiency more commonly presented with distal symmetric sensory neuropathy compared to non-deficient patients (61.0% vs. 38.5%). The deficient group also showed a higher prevalence of anemia (42.7% vs. 21.4%) and elevated mean corpuscular volume. Diabetes mellitus was present in 46.3% of the overall cohort but did not show a statistically significant association with vitamin B12 deficiency.

Conclusion:
Vitamin B12 deficiency was observed in approximately one-third of patients presenting with neuropathic symptoms. Given its high prevalence and reversible nature, routine screening for vitamin B12 deficiency should be considered in patients presenting with neuropathic manifestations to facilitate early diagnosis and timely treatment.

INTRODUCTION

Peripheral neuropathy is a common neurological condition characterized by sensory, motor, and autonomic dysfunction resulting from damage to peripheral nerves. Patients frequently present with symptoms such as numbness, tingling, burning sensations, paresthesia, and, in advanced cases, weakness or gait disturbances. The etiology of peripheral neuropathy is diverse, with metabolic, infectious, autoimmune, toxic, and nutritional causes contributing significantly to its burden. Identifying reversible causes remains a key priority in clinical practice, as early diagnosis and appropriate treatment can halt progression and improve patient outcomes.

Vitamin B12 (cobalamin) is an essential water-soluble vitamin that plays a critical role in DNA synthesis, erythropoiesis, and neurological function. It is particularly important for myelin formation and maintenance through its involvement in methylation pathways and fatty acid metabolism. Deficiency of vitamin B12 leads to impaired myelin synthesis and accumulation of neurotoxic metabolites, resulting in demyelination and axonal degeneration within the peripheral and central nervous systems. Neurological manifestations may occur even in the absence of hematological abnormalities, making the diagnosis challenging if clinical suspicion is low.

Neuropathic symptoms are among the most common and earliest clinical manifestations of vitamin B12 deficiency. Patients may develop distal symmetric sensory neuropathy, classically involving the lower limbs, with progression to motor involvement in severe or prolonged deficiency. Despite the well-established pathophysiological link between vitamin B12 deficiency and neuropathy, the condition often remains underdiagnosed, particularly in resource-limited settings and in patients with overlapping risk factors such as diabetes mellitus, chronic alcohol use, vegetarian diets, malabsorption syndromes, and long-term use of medications such as metformin and proton pump inhibitors.

Several studies have reported variable prevalence rates of vitamin B12 deficiency among patients presenting with neuropathic symptoms, reflecting differences in population characteristics, dietary habits, and diagnostic thresholds used. In routine clinical practice, neuropathy is frequently attributed to more common etiologies such as diabetes mellitus, leading to delayed recognition of nutritional deficiencies that are potentially reversible. This diagnostic oversight may result in persistent symptoms and irreversible neurological damage if treatment is not initiated in a timely manner.

In developing countries, vitamin B12 deficiency represents a significant public health concern due to dietary patterns, socioeconomic factors, and limited access to preventive healthcare. Retrospective evaluation of routinely collected clinical data provides valuable insight into real-world disease burden and assists in identifying gaps in diagnostic and management strategies. Cross-sectional studies are particularly useful for estimating prevalence and describing clinical associations in hospital-based populations.

The present study was undertaken to evaluate the prevalence of vitamin B12 deficiency in patients presenting with neuropathic symptoms and to analyze associated clinical and laboratory characteristics using a retrospective cross-sectional design. By highlighting the burden of vitamin B12 deficiency in this patient population, this study aims to emphasize the importance of routine screening and early intervention in patients presenting with neuropathic manifestations.

MATERIALS AND METHODS

Study Design and Setting

This retrospective cross-sectional study was conducted at a tertiary care teaching hospital. Medical records of patients presenting with neuropathic symptoms were reviewed over a two-year period from January 2025 to December 2025. The study was designed to evaluate the prevalence of vitamin B12 deficiency and its association with clinical and laboratory parameters in this patient population.

Study Population

Adult patients (≥18 years) who presented with symptoms suggestive of peripheral neuropathy during the study period were considered eligible. Neuropathic symptoms included numbness, tingling, burning sensation, paresthesia, reduced sensation, or neuropathic pain involving the upper or lower limbs.

Inclusion Criteria

- Adult patients aged 18 years and above
- Presentation with clinical features suggestive of peripheral neuropathy
- Availability of documented serum vitamin B12 levels at the time of presentation
- Complete medical records with relevant clinical and laboratory data.

Exclusion Criteria

- Patients with documented causes of neuropathy unrelated to nutritional deficiency, such as acute traumatic nerve injury
- Known cases of hereditary neuropathy
- Patients with chronic renal failure on dialysis
- History of chemotherapy-induced neuropathy
- Incomplete or missing laboratory data.

Sample Size Calculation

The sample size was calculated using the formula for estimating prevalence in a cross-sectional study:
$n = Z^2 \times p \times q / d^2$
Where $Z = 1.96$ for a 95% confidence interval, p = assumed prevalence of vitamin B12 deficiency (30%), $q = 1 - p$, and d = absolute precision of 6%. The minimum calculated sample size was 224 patients. To account for incomplete records, a total of 240 patients were included in the final analysis.

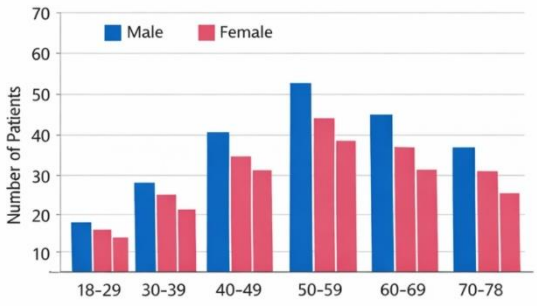
Data Collection

Data were collected retrospectively from hospital medical records using a structured data extraction form. The following variables were recorded:
Demographic details: age and sex
Clinical characteristics: type and duration of neuropathic symptoms, comorbidities such as diabetes mellitus and hypertension
Hematological parameters: hemoglobin, mean corpuscular volume
Biochemical parameters: serum vitamin B12 levels
Serum vitamin B12 levels were measured using standard chemiluminescent immunoassay techniques as part of routine clinical evaluation.

RESULTS

Study Population

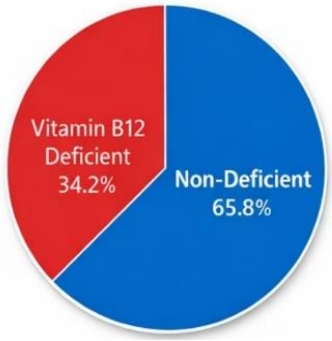
Figure 1: Age and Sex Distribution of Patients Presenting with Neuropathic Symptoms.



A total of 240 patients presenting with neuropathic symptoms were included in the final analysis. The mean age of the study population was 49.6 ± 13.2 years, with ages ranging from 19 to 78 years. Males constituted 58.3% of the cohort, while 41.7% were females.
The most commonly reported symptoms were numbness and tingling sensations, followed by burning pain and paresthesia. Distal symmetric sensory neuropathy was the predominant clinical pattern observed, accounting for more than half of the cases.
The demographic distribution of the study population according to age group and sex is illustrated in Figure 1.

Prevalence of Vitamin B12 Deficiency

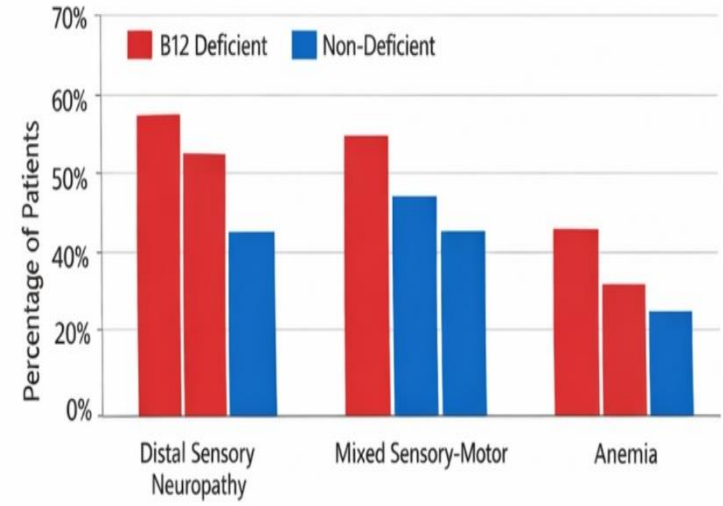
Figure 2: Prevalence of Vitamin B12 Deficiency among Patients with Neuropathic Symptoms.



Vitamin B12 deficiency, defined as serum vitamin B12 levels below 200 pg/mL, was identified in 82 patients, resulting in a prevalence of 34.2% among patients presenting with neuropathic symptoms. The remaining 65.8% had serum vitamin B12 levels within the normal range.
The proportion of vitamin B12 deficient and non-deficient patients is depicted in Figure 2.

Clinical Characteristics According to Vitamin B12 Status

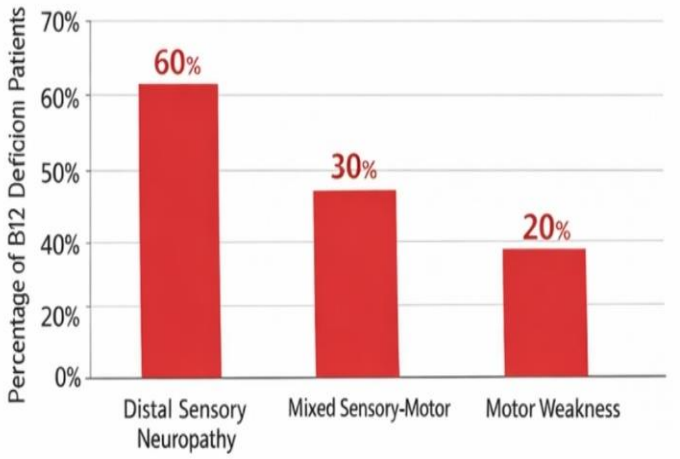
Figure 3: Neuropathy patterns in vitamin B12 Deficient and non-deficient patients.



Patients with vitamin B12 deficiency more frequently presented with distal symmetric sensory neuropathy compared to patients with normal vitamin B12 levels. Mixed sensory-motor neuropathy was more commonly observed in the non-deficient group.
Anemia was significantly more prevalent among vitamin B12 deficient patients, and this group also demonstrated higher mean corpuscular volume values, suggestive of macrocytosis. Although diabetes mellitus was common in the overall cohort, its distribution was similar in both vitamin B12 deficient and non-deficient groups, indicating no significant association.
The comparison of clinical patterns of neuropathy between vitamin B12 deficient and non-deficient patients is shown in Figure 3.

Pattern of Neuropathy in Vitamin B12 Deficient Patients

Figure 4: Distribution of Neuropathy Patterns among Vitamin B12 Deficient



Among patients with vitamin B12 deficiency, distal symmetric sensory neuropathy was the most frequent presentation, followed by mixed sensory-motor involvement. Motor weakness was observed mainly in patients with longer duration of symptoms, suggesting a progressive course in untreated deficiency.
The distribution of neuropathy patterns exclusively among vitamin B12 deficient patients is illustrated in Figure 4.

DISCUSSION

Peripheral neuropathy is a frequent clinical presentation with a wide range of etiologies, among which vitamin B12 deficiency represents an important and potentially reversible cause. In this retrospective cross-sectional study, vitamin B12 deficiency was identified in approximately one-third of patients presenting with neuropathic symptoms, highlighting its substantial burden in routine clinical practice. These findings emphasize the need for heightened clinical suspicion and routine screening for vitamin B12 deficiency in patients with neuropathic manifestations.
The prevalence of vitamin B12 deficiency observed in this study (34.2%) is consistent with previously reported rates in hospital-based populations presenting with neurological complaints. Variations in prevalence across studies may be attributed to differences in dietary habits, socioeconomic factors, underlying comorbidities, and diagnostic thresholds used to define deficiency. In developing countries, nutritional deficiencies remain common, and vitamin B12 deficiency is often underdiagnosed due to overlapping symptoms with more prevalent causes of neuropathy such as diabetes mellitus.
Distal symmetric sensory neuropathy was the most frequently observed clinical pattern, particularly among patients with vitamin B12 deficiency. This presentation is characteristic of cobalamin deficiency and is believed to result from impaired myelin synthesis and axonal degeneration affecting long peripheral nerves. The predominance of sensory symptoms, especially numbness and paresthesia, aligns with the known pathophysiology of vitamin B12 deficiency–related neuropathy, where sensory fibers are affected earlier than motor fibers.
Anemia and macrocytosis were significantly more common in the vitamin B12 deficient group; however, a notable proportion of deficient patients presented with neurological symptoms in the absence of marked hematological abnormalities. This finding underscores an important clinical consideration—neurological manifestations of vitamin B12 deficiency may precede or occur independently of anemia. Reliance solely on hematological parameters may therefore delay diagnosis and treatment, potentially leading to irreversible neurological damage.

Diabetes mellitus was a common comorbidity in the study population but did not show a significant association with vitamin B12 deficiency. This suggests that neuropathic symptoms in diabetic patients should not be automatically attributed to diabetic neuropathy without evaluating for coexisting nutritional deficiencies. Additionally, long-term use of medications such as metformin, commonly prescribed in diabetes, may further contribute to vitamin B12 deficiency, reinforcing the need for routine screening in this subgroup.

The retrospective cross-sectional design of this study provides valuable insight into real-world clinical practice; however, it also carries certain limitations. The reliance on medical records may result in incomplete data capture, and the temporal relationship between vitamin B12 deficiency and neuropathic symptoms cannot be definitively established. Furthermore, electrophysiological studies were not uniformly available, and functional outcomes following vitamin B12 replacement therapy could not be assessed.

Despite these limitations, the study highlights an important and modifiable contributor to neuropathic symptoms. Early identification and treatment of vitamin B12 deficiency can lead to symptom improvement and prevent progression of neurological damage. Incorporating routine vitamin B12 assessment into the diagnostic workup of patients presenting with neuropathic symptoms may therefore have significant clinical benefits.

CONCLUSION

Vitamin B12 deficiency was found to be common among patients presenting with neuropathic symptoms, affecting approximately one-third of the study population. Distal symmetric sensory neuropathy emerged as the predominant clinical presentation, and vitamin B12 deficient patients were more likely to exhibit anemia and macrocytosis, although neurological manifestations frequently occurred in the absence of significant hematological abnormalities. These findings highlight the importance of considering vitamin B12 deficiency in the differential diagnosis of neuropathy, regardless of the presence of anemia or coexisting comorbidities such as diabetes mellitus. Routine screening for vitamin B12 deficiency in patients presenting with neuropathic symptoms may facilitate early diagnosis and timely intervention, potentially preventing irreversible neurological damage and improving patient outcomes.

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