Relationship Between Body Mass Index and Vitamin B1 Levels with the Degree of HIV Neuropathy

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Abstract

Peripheral neuropathy is a common neurological complication of human immunodeficiency virus (HIV) infection. Nutritional factors, particularly vitamin B1 (thiamine), may contribute to peripheral nerve dysfunction because of their essential roles in cellular energy metabolism and neuronal function. This study aimed to investigate the associations of body mass index (BMI), dietary vitamin B1 intake, and serum vitamin B1 levels with peripheral neuropathy severity among people living with HIV. This analytical cross-sectional study included 90 patients with HIV-associated peripheral neuropathy who met the eligibility criteria. Data were obtained through medical history, physical examination, BMI assessment, dietary vitamin B1 intake assessment using a food frequency questionnaire, and serum vitamin B1 measurement. Participants were classified as having mild or moderate neuropathy. Categorical variables were analysed using the chi-square test, whereas continuous non-normally distributed variables were compared using the Mann Whitney U test. The mean age of the participants was 39.88 ± 10.46 years. Of the 90 participants, 70 (77.8%) were male. Normal BMI was observed in 40.0% of participants, while 25.6% were overweight, 20% were obese, and 14.4% were underweight. Forty-two participants (46.7%) had mild neuropathy and 48 (53.3%) had moderate neuropathy. Dietary vitamin B1 intake did not differ significantly between the mild and moderate neuropathy groups [median: 2.61 (0.44–5.99) vs. 2.46 (0.44–7.01) mg/day, respectively; p = 0.683]. Serum vitamin B1 levels were significantly higher in participants with mild neuropathy than in those with moderate neuropathy [median: 1,427.25 (417.39–3,083.42) vs. 748.60 (29.88–10,585.42) ng/mL, respectively; p < 0.001]. BMI was not significantly associated with neuropathy severity (p = 0.261). Serum vitamin B1 levels were significantly lower in participants with moderate neuropathy than in those with mild neuropathy, whereas BMI and dietary vitamin B1 intake were not significantly associated with neuropathy severity. Given the wide range of serum vitamin B1 concentrations observed, these findings do not support routine thiamine supplementation in all patients. Instead, thiamine status should be monitored and confirmed deficiencies should be appropriately treated. Prospective studies controlling for antiretroviral therapy, immune status, and other potential confounders, while incorporating measurements of thiamine diphosphate and thiamine transporters, are warranted.

Keywords: HIV, peripheral neuropathy, vitamin B1, thiamine, body mass index, nutritional status

INTRODUCTION

Human Immunodeficiency Virus (HIV) infection remains a global health challenge despite substantial advances in antiretroviral (ARV) therapy have significantly improved patient survival. As the life expectancy of people living with HIV increases, chronic non-infectious complications are becoming increasingly prevalent, particularly neurological disorders affecting both the central and peripheral nervous systems (Bekker et al., 2023). One of the most common neurological complications is HIV-associated peripheral neuropathy,

which is characterized by sensory impairment, neuropathic pain, paresthesias, and reduced motor function. This condition can substantially impair patient's quality of life (Motwani et al., 2022; Xu & Yu, 2024).

The pathogenesis of HIV-associated peripheral neuropathy is complex and involves interactions among virological, immunological, metabolic, and nutritional factors. Human Immunodeficiency Virus (HIV) does not directly infect neurons; rather, it activates macrophages and microglia, which subsequently produce a variety of pro-inflammatory mediators (Liu & Tang, 2023; Jazebi et al., 2021). These mediators induce oxidative stress, mitochondrial dysfunction, impaired axonal transport, and myelin damage, ultimately leading to peripheral nerve degeneration. Furthermore, persistent chronic inflammation may accelerate neuronal injury through increased free radical production and disruption of cellular energy metabolism (Gagliardi et al., 2025; Borrajo et al., 2021).

One factor that may influence this process is nutritional status. Body Mass Index (BMI) is a widely used indicator of nutritional status and body composition. Among people living with HIV, alterations in BMI frequently occur as a result of increased energy requirements, chronic systemic inflammation, impaired nutrient absorption, and the adverse effects of antiretroviral therapy (Blaauw, 2025; Obeagu, 2024; Ramirez et al., 2024). A low BMI may reflect malnutrition and loss of muscle mass, both of which are associated with increased metabolic stress and greater susceptibility to nerve damage. Conversely, elevated BMI and excess adipose tissue accumulation have also been linked to increased production of proinflammatory cytokines, which may exacerbate neuroinflammatory processes (Boleti et al., 2023; Schmitt & Gaspar, 2023; Woo et al., 2022).

In addition to overall nutritional status, micronutrients play a critical role in maintaining peripheral nerve function, particularly vitamin B1 (thiamine). Vitamin B1 is a water-soluble vitamin that serves as an essential coenzyme in cellular energy metabolism. In its active form, thiamine pyrophosphate, it functions as a cofactor for several key enzymes, including pyruvate dehydrogenase, α-ketoglutarate dehydrogenase, and transketolase, which are involved in the Krebs cycle and the pentose phosphate pathway. These metabolic pathways are essential for the production of adenosine triphosphate (ATP) and nicotinamide adenine dinucleotide phosphate (NADPH), both of which are required to maintain neuronal function and structural integrity (Mrowicka et al., 2023; Alturfan, 2023). Assessing BMI and vitamin B1 status simultaneously provides complementary information regarding nutritional and metabolic factors relevant to peripheral nerve health. Whereas BMI reflects overall nutritional status and

body composition, serum vitamin B1 levels provide a more specific measure of thiamine availability for neuronal energy metabolism.

Vitamin B1 deficiency disrupts mitochondrial energy production, resulting in lactate accumulation, increased oxidative stress, and impaired neuronal metabolism. These disturbances can lead to impaired nerve impulse transmission, axonal degeneration, and Schwann cell injury, all of which contribute to the development of peripheral neuropathy. Among people living with HIV, vitamin B1 deficiency may arise from inadequate dietary intake, impaired gastrointestinal absorption, increased metabolic demands, or chronic inflammation that interferes with micronutrient utilization.

Experimental and clinical studies have demonstrated that impaired thiamine metabolism is closely associated with various neurodegenerative disorders and peripheral neuropathies. However, the relationship between vitamin B1 status and neuropathy severity among people living with HIV remains incompletely understood. Some studies have reported an association between low thiamine levels and an increased risk of neuropathy, whereas others have yielded inconsistent findings. These discrepancies suggest that HIV-associated peripheral neuropathy is influenced by multiple interacting factors, including nutritional status, systemic inflammation, mitochondrial dysfunction, and vitamin metabolism (Stein et al., 2021; Alvarez et al., 2026).

From a biomolecular perspective, BMI and vitamin B1 status may represent two important determinants in the pathogenesis of HIV-associated peripheral neuropathy: systemic metabolic status and cellular cofactor availability for energy metabolism (Tyberg, 2022). An imbalance in either of these factors may exacerbate nerve injury through mechanisms involving neuroinflammation, oxidative stress, and mitochondrial dysfunction. Therefore, evaluating the relationship among BMI, vitamin B1 status, and neuropathy severity may help identify potentially modifiable nutritional and metabolic factors relevant to HIV-associated peripheral neuropathy.

However, evidence regarding the association between vitamin B1 status and the severity of peripheral neuropathy among people living with HIV remains limited and inconsistent, particularly in Indonesia. Among patients receiving HIV care at Dr. M. Djamil Hospital, Padang, data on vitamin B1 status and its relationship with peripheral neuropathy severity are also scarce. Therefore, investigating the associations of BMI and vitamin B1 status with neuropathy severity in this population is important for generating locally relevant evidence on nutritional factors associated with HIV-related peripheral neuropathy.

Based on the aforementioned considerations, this study aimed to examine the associations of Body Mass Index (BMI) and serum vitamin B1 levels with neuropathy severity among people living with HIV . The findings are expected to provide a scientific basis for understanding the roles of nutritional status and thiamine metabolism in the pathogenesis of HIV-associated peripheral neuropathy and to support the development of nutrition-based preventive and adjunctive therapeutic strategies for this population. A priori, we hypothesized that lower serum vitamin B1 levels would be associated with greater neuropathy severity and that BMI would be significantly associated with neuropathy severity among people living with HIV.

METHODS

This analytical cross-sectional study involved HIV patients with peripheral neuropathy who attended the VCT Outpatient Clinic at Dr. M. Djamil General Hospital, Padang, Indonesia. Data were collected from July 2025 to February 2026. A total of 90 participants were recruited using purposive sampling based on predefined inclusion and exclusion criteria. The inclusion criteria were HIV patients aged 18–59 years with peripheral neuropathy who were willing to participate and provided written informed consent. The exclusion criteria included chronic kidney disease, impaired liver function, autoimmune disorders, pregnancy, malignancy, type 2 diabetes mellitus, and current use of steroid or immunosuppressive therapy. Peripheral neuropathy was assessed using the Indonesian version of the Toronto Clinical Scoring System (TCSS). Based on the total TCSS score, neuropathy severity was classified as mild (6–8), moderate (9–11), or severe (12–19). Of the 90 participants who met the eligibility criteria, all were included in the final analysis. The sample size of 90 participants was determined by the number of eligible patients available during the study period rather than by a formal sample size calculation.

Venous blood samples were collected at the laboratory of Dr. M. Djamil General Hospital. Vitamin B1 levels were analyzed at the Infectious Diseases Diagnostic and Research Center, Faculty of Medicine, Andalas University. Vitamin B1 concentrations were measured using an enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's instructions, and absorbance was determined using an ELISA reader. Dietary vitamin B1 intake was assessed using a Semi-Quantitative Food Frequency Questionnaire (SQ-FFQ). The questionnaire was administered through direct interviews to assess the frequency and portion size of food and beverage consumption. Household measures (*Ukuran Rumah Tangga*[URT]) were used to record portion sizes and were subsequently converted into grams. Reported

consumption frequencies and portion sizes were converted into average daily intake, and the vitamin B1 content of the reported food items was used to estimate daily vitamin B1 intake, expressed in mg/day. The SQ-FFQ has demonstrated good to very good reliability for assessing energy and nutrient intake, with previous validation studies reporting Cronbach's alpha coefficients of 0.947 and 0.962 for the first and second administrations, respectively, and Intraclass Correlation Coefficient (ICC) values ranging from 0.75 to >0.90 for energy and most nutrients. These findings support the reliability of the instrument for assessing dietary intake in adult populations, including people living with HIV/AIDS.

Body weight was measured using a calibrated weighing scale, while height was measured using a stadiometer. Body mass index (BMI) was calculated by dividing body weight in kilograms by height in meters squared (kg/m²). BMI was classified into four categories: : underweight (≤18.49 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (>25–27 kg/m²), and obesity (>27 kg/m²). Demographic and clinical information, including age, duration of HIV infection, and antiretroviral regimen, was collected using a structured questionnaire.

Statistical analyses were performed using IBM SPSS Statistics version 25. The distribution of continuous variables was assessed using the Shapiro–Wilk test. Non-normally distributed continuous variables were summarized as medians and interquartile ranges and compared between the mild and moderate neuropathy groups using the Mann–Whitney U test. Normally distributed continuous variables were summarized as means and standard deviations. Spearman's rank correlation analysis was used to assess the associations of dietary vitamin B1 intake and serum vitamin B1 levels with neuropathy severity. Correlation coefficients (r_s) and two-sided p-values were reported, with statistical significance defined as $p < 0.05$

Potential confounding factors, including the duration of HIV infection, antiretroviral regimen, viral load, and CD4 count, were identified a priori because of their potential relevance to HIV-associated peripheral neuropathy. However, multivariable regression analysis was not performed because the study included only 90 participants, limiting the number of covariates that could be incorporated into a stable multivariable model. In addition, the limited number of participants within each neuropathy severity category reduced the feasibility of reliably estimating adjusted effects. Therefore, the primary analyses were restricted to unadjusted comparisons, and the observed associations were interpreted as exploratory rather than as independent or causal effects. The potential influence of these confounding factors was considered when interpreting the findings.

Sampling was conducted based on medical history taking and physical examination at the VCT clinic. Blood samples were collected from the study participants at the laboratory of Dr. M. Djamil General Hospital. Vitamin B1 levels were analyzed at the Infectious Diseases Diagnostic and Research Center, Faculty of Medicine, Andalas University. This study received ethical approval from the Health Research Ethics Committee of Dr. M. Djamil General Hospital, Padang, under approval/reference number DP.04.03/D.XVI.10.1/160/2025, effective from April 1, 2025. All participants provided written informed consent before enrollment in the study.

RESULTS

Before further analysis, the respondents' characteristics are presented to provide a general overview of the study participants. These characteristics involved gender, age, body mass index, drug dosage, duration of HIV, neuropathy degree, and vitamin B1 intake and biomarker levels. Presenting these data is important as a basis for understanding the respondents' baseline conditions and supporting a more comprehensive interpretation of the study findings. The detailed characteristics of the respondents are shown in Table 1.

Table 1 Respondent Characteristics Results (n=90)

Variable	f (%)
Gender	
Male	70 (77,8)
Female	20 (22,2)
BMI (kg/m2)	
Underweight	13(14,4)
Normal	36(40)
Overweight	23(25,6)
Obese	18(20)
Duration of HIV (years)	
<1 year	12(13,3)
1-5 years	38(42,2)
>5 years	40(44,4)
Neuropathy Degree	
Mild	42(46,7)
Moderate	48(53.3)
Age	
18-25	6(6.7)
26-45	61(67.8)
>45	23(25.6)

Table 2 Distribution of Dietary Vitamin B1 Intake and Serum Vitamin B1 Levels among Study Participants

Variable	Mild Neuropathy (42) Median (min-max)	Moderate Neuropathy (48) Median (min-max)	p
Dietary vitamin B1 intake assessed using an FFQ (mg/day)	2,61(0,44-5.99)	2.46 (0.44-7.01)	0.683
Serum vitamin B1 level (ng/mL)	1427.25(417.39-3083.42)	748.60 (29.88-10585.42)	<0.001

A total of 90 participants with HIV-associated peripheral neuropathy were included in the study. Most participants were male (70; 77.8%), while 20 (22.2%) were female. Regarding nutritional status, 36 participants (40.0%) had a normal body mass index (BMI), 23 (25.6%) were overweight, 18 (20.0%) were obese, and 13 (14.4%) were underweight.

Most participants had been living with HIV for more than five years (40; 44.4%), followed by those with an HIV duration of 1 5 years (38; 42.2%). The remaining 12 participants (13.3%) had an HIV duration of less than one year. Based on neuropathy severity, 48 participants (53.3%) had moderate neuropathy, whereas 42 (46.7%) had mild neuropathy.
Table 2 shows that the median dietary vitamin B1 intake was 2.61 mg/day (range: 0.44 5.99) in participants with mild neuropathy and 2.46 mg/day (range: 0.44 7.01) in those with moderate neuropathy. There was no statistically significant difference in dietary vitamin B1 intake between the two groups ($p = 0.683$).

In contrast, the median serum vitamin B1 level was significantly higher in participants with mild neuropathy than in those with moderate neuropathy: 1,427.25 (range: 417.39 3,083.42) versus 748.60 (range: 29.88 10,585.42), respectively ($p < 0.001$). These results demonstrate a significant difference in serum vitamin B1 levels according to neuropathy severity, whereas reported dietary vitamin B1 intake did not differ significantly between the groups. Spearman's rank correlation analysis showed no significant correlation between dietary vitamin B1 intake and neuropathy severity ($r_s = -0.043$, $p = 0.685$). In contrast, serum vitamin B1 levels showed a statistically significant negative correlation with neuropathy severity ($r_s = -0.490$, $p < 0.001$).

DISCUSSIONS

This study aimed to evaluate the associations of Body Mass Index (BMI) and vitamin B1 levels with the severity of peripheral neuropathy among people living with HIV. The findings demonstrated a significant association between vitamin B1 levels and neuropathy severity ($p<0.001$), whereas no significant association was observed between BMI and neuropathy severity ($p=0.261$). These results suggest that vitamin B1 status may be associated

with variations in neuropathy severity among people living with HIV , whereas BMI alone may not adequately reflect the nutritional or metabolic factors related to peripheral nerve dysfunction. Because of the cross-sectional design of this study, these associations should not be interpreted as evidence of causality.

Most respondents had been living with HIV for more than five years. A longer duration of HIV infection may be associated with a greater burden of chronic inflammatory and metabolic processes that can affect peripheral nerve function. Persistent immune activation associated with HIV may contribute to neuroinflammation, oxidative stress, and mitochondrial dysfunction, all of which have been implicated in peripheral nerve injury. In addition, long-term exposure to antiretroviral therapy may represent another factor associated with the development or progression of peripheral neuropathy. However, because the duration of HIV infection and antiretroviral exposure were not evaluated using multivariable analysis in this study, their independent contributions to neuropathy severity could not be determined. Therefore, the findings should be interpreted as supporting the multifactorial nature of HIV-associated peripheral neuropathy rather than suggesting that a longer duration of HIV directly causes neuropathy (Wondmagegn et al., 2024).

No significant association was observed between BMI and neuropathy severity ($p=0.261$). Most participants had BMI values within the normal range, although some participants were classified as underweight or overweight. The relatively limited variability in BMI may have reduced the ability to detect an association between BMI and neuropathy severity. In addition, BMI is a general anthropometric measure and does not distinguish between fat mass, lean mass, and body fat distribution. In people living with HIV, antiretroviral therapy may also influence body weight and body composition, potentially weakening the relationship between BMI and metabolic or nutritional factors relevant to neuropathy. Other metabolic and clinical factors, including glucose metabolism, systemic inflammation, duration of HIV infection, and antiretroviral exposure, may also contribute to peripheral nerve dysfunction. Therefore, the absence of a significant association in this study does not necessarily indicate that nutritional status is unrelated to neuropathy; rather, it suggests that BMI alone may not adequately capture the nutritional and metabolic characteristics most relevant to neuropathy in this population (Wondmagegn et al., 2024; Tsalamandris et al., 2023).

These findings are broadly consistent with those of Vermaak et al., (2015), who reported no significant associations between components of metabolic syndrome and distal sensory polyneuropathy (DSP). Similarly, a study conducted in Makassar, Indonesia, found no significant correlation between BMI and DSP severity. However, that study identified a

significant correlation between HIV disease stage and DSP severity ($p = 0.032$), suggesting that disease-related characteristics may be more closely associated with neuropathy severity than general anthropometric measures. Although these studies support the absence of a clear association between BMI and neuropathy severity, their findings should be interpreted with caution because they evaluated different clinical variables and may have involved populations with differing demographic and clinical profiles.

Vitamin B1 intake among respondents showed considerable variability. Some patients had low vitamin B1 intakes, which may increase the risk of neuropathy through impaired energy metabolism in nerve cells. However, neuropathy was also observed in some patients with adequate or high vitamin B1 intake, suggesting that factors such as dietary intake, disease status, gastrointestinal absorption, and individual metabolic requirements may influence thiamine status and neuropathy risk. Thiamine deficiency may act as an additional factor that exacerbates nerve damage related to HIV infection, chronic inflammation, and antiretroviral drug toxicity.

The significant association between vitamin B1 levels and neuropathy severity in this study is biologically plausible because thiamine is an essential cofactor in neuronal energy metabolism. Its active form, thiamine pyrophosphate, is required for several enzymes involved in glucose oxidation and cellular energy production. Consequently, inadequate thiamine availability may impair neuronal energy metabolism and increase the susceptibility of peripheral nerves to metabolic and oxidative stress (Mrowicka et al., 2023).

Ndakala et al., (2017) highlighted the need for further research into vitamin-related factors associated with peripheral neuropathy among people living with HIV, particularly those receiving combination antiretroviral therapy. The authors also discussed the potential contribution of genetic variability to differences in vitamin status and emphasized the need for studies in resource-limited settings. Their findings provide a rationale for investigating vitamin status in HIV-associated neuropathy; however, they do not offer a directly comparable effect estimate for the relationship between serum vitamin B1 levels and neuropathy severity. Therefore, the significant association observed in the present study contributes to the growing body of evidence in this field, while also requiring confirmation through further clinical research.

Previous studies have reported mixed findings regarding the roles of BMI and metabolic factors in HIV-associated peripheral neuropathy. The absence of a significant association between BMI and neuropathy severity in the present study is consistent with findings reported by Vermaak et al. (2015) and the Makassar study, whereas Ellis et al. (2020)

identified an association between BMI and incident neuropathic pain, but not with incident distal sensory polyneuropathy (DSP). These differences underscore the importance of distinguishing between neuropathy occurrence, neuropathic pain, and neuropathy severity when comparing findings across studies. Evidence directly examining the relationship between serum vitamin B1 levels and neuropathy severity remains limited among the studies discussed. Accordingly, the present findings provide additional evidence from an Indonesian clinical population; however, further studies using comparable outcome measures and appropriate adjustment for potential confounding factors are needed to confirm these associations.

Several limitations should be considered when interpreting these findings. First, the cross-sectional design limits the ability to establish temporal or causal relationships between vitamin B1 status, BMI, and neuropathy severity. Second, the relatively small sample size of 90 participants may have reduced the statistical power to detect modest associations.

Third, participants were recruited from a single tertiary referral hospital, which may limit the generalizability of the findings to people living with HIV receiving care in primary healthcare facilities or other clinical settings. In addition, the predominance of male participants may also limit the applicability of the findings to women living with HIV. Fourth, although the Toronto Clinical Scoring System (TCSS) is a standardized clinical assessment tool, it includes subjective symptom components and may therefore be susceptible to reporting and observer bias. Finally, no multivariable analysis was performed; consequently, potential confounding factors, including age, sex, duration of HIV infection, antiretroviral regimen, and other metabolic or nutritional variables, could not be controlled. Therefore, the direction and magnitude of any residual confounding cannot be determined from the present data.

The objective of analysing the relationships of BMI and vitamin B1 with neuropathy severity was achieved. Serum vitamin B1 levels were significantly lower in the moderate neuropathy group than in the mild neuropathy group [median 748.60 (29.88 10,585.42) vs. 1,427.25 (417.39 3,083.42) ng/ml; $p<0.001$], whereas BMI did not differ significantly according to neuropathy severity ($p=0.261$). Dietary vitamin B1 intake also did not differ between the groups ($p=0.683$). Thus, only the serum vitamin B1 measure showed a significant association with neuropathy severity. Because of the cross-sectional design, this finding indicates an association and does not establish that lower serum vitamin B1 caused more severe neuropathy.

The wide distribution of serum vitamin B1 concentrations, including a very high maximum value in the moderate neuropathy group, does not support routine vitamin B1 supplementation based on the present findings. In clinical practice, thiamine status may be

evaluated when deficiency is clinically suspected, and confirmed deficiency should be corrected in accordance with appropriate clinical guidance. However, the unexpectedly high concentrations observed in some participants require verification and further investigation of possible supplementation, assay-related variation, altered thiamine distribution, and abnormalities of thiamine metabolism or cellular transport. Neuropathy should be assessed using a standardised instrument and interpreted alongside relevant clinical factors rather than serum vitamin B1 alone.

Future studies should use prospective, preferably multicentre, designs with larger samples and repeated measurements to clarify temporal relationships. Analyses should control for antiretroviral regimen and duration, CD4 cell count, viral load, HIV duration, age, sex, metabolic comorbidities, renal and hepatic function, and vitamin supplementation. Measurement of the biologically active form of thiamine, thiamine diphosphate (TDP), together with thiamine transporters such as THTR-1 and THTR-2, would provide a more complete assessment of thiamine availability and cellular handling. These approaches are needed to determine whether altered thiamine status contributes to neuropathy progression or instead reflects broader metabolic changes associated with HIV and its treatment.

CONCLUSIONS

This study demonstrates that serum vitamin B1 status was associated with peripheral neuropathy severity in people living with HIV, whereas BMI and dietary vitamin B1 intake were not significantly associated with neuropathy severity. The findings suggest that serum vitamin B1 may provide clinically relevant information regarding neuropathy severity, although the wide range of serum concentrations and the cross-sectional design do not establish causality or support routine vitamin B1 supplementation. Clinicians may consider assessing thiamine status when clinically appropriate and correcting confirmed deficiency as part of nutritional management. Future prospective studies should account for antiretroviral exposure and immune status and incorporate measurements of thiamine diphosphate (TDP) and thiamine transporters to clarify the underlying mechanisms and clinical implications of the observed association.

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