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Evaluating the Prevalence and Clinical Implications of Toxic Vitamin B6 Levels in Neuropathies: A Retrospective Observational Study at Burjeel Hospital, Dubai

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Abstract

Background: Vitamin B6 toxicity is an underrecognized cause of neuropathy, often overlooked in routine clinical practice. With the widespread availability of over-the-counter supplements, excessive intake has become increasingly common, potentially leading to sensory and motor complications.

Objective: This study aims to evaluate the prevalence of toxic vitamin B6 levels and its clinical implications in patients presenting with neuropathic symptoms at Burjeel Hospital, Dubai.

Methods: We conducted a retrospective observational study on adult patients who had their serum vitamin B6 levels tested during neurology clinic visits. Patients with previously diagnosed causes for neuropathy (diabetic neuropathy, etc.) were excluded. Clinical data, including sensory, motor, and cognitive symptoms, were analyzed for associations with B6 levels. Statistical analyses were performed using R, with significance set at $p < 0.05$.

Results: Among 68 patients, 30.9% showed toxic vitamin B6 levels (>37.7 µg/L). While no significant associations were found between B6 levels and demographic factors or most laboratory parameters, a significant positive correlation was noted between B6 and B12 levels ($p = 0.023$, $r = 0.48$). Sensory dysfunction was prevalent in 95.2% of patients presenting with toxic B6 levels. Additionally, motor symptoms were associated with elevated B6 levels ($p = 0.025$).

Conclusion: Vitamin B6 toxicity is a frequent but underdiagnosed contributor to neuropathy. Sensory dysfunction remains the hallmark of toxicity, whereas the association with motor symptoms suggests a broader neurological impact. Thus, increased clinical awareness and routine B6 screening in patients with unexplained neuropathy may improve diagnostic accuracy and patient outcomes.

Keywords: Clinical Implications, Neuropathies, motor complications, cognitive symptoms, vitamin B6, Sensory dysfunction, pyridoxine.

I. Introduction

Although vitamins and minerals are vital components of daily nutrition, their usage has increasingly been shaped by global marketing strategies rather than by the pursuit of better health. Believing high-dose supplements will enhance health, patients may inadvertently reach toxic levels, then present with neurological symptoms that can confuse both patient and physician, ultimately risking misdiagnosis. From sweet and tangy beverages to gummies, vitamin B6 has become a hidden culprit in various neurological manifestations.

Vitamin B6 (B6) is a collective term for all 3-hydroxy-2-methylpyridine derivatives that possess biological activity; although sometimes called pyridoxine, the former is the preferred term. The vitamin B6 family consists of three structurally related compounds: pyridoxine (PN), pyridoxal (PL), and pyridoxamine (PM), as well as their respective phosphorylated forms (1). Of these, pyridoxal phosphate (PLP) is the most biologically active and plays a central role as a coenzyme in more than 160 enzymatic processes throughout the body (2).

Vitamin B6 is predominantly ingested in its phosphorylated

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forms, pyridoxal phosphate (PLP) and pyridoxine phosphate (PNP), which are naturally present in various foods such as fortified cereals, fish, organ meats like liver, and an assortment of fruits and vegetables. (3). In adults, the typical daily intake of B6 from diet alone, without the use of supplements, is estimated to range from 1.4 to 3.1 mg, and may reach up to 5 mg (4,5).

Fortified foods and over-the-counter (OTC) supplements have become increasingly common strategies for preventing nutrient deficiencies and promoting better health. Among fortified foods, breakfast cereals are the primary sources of vitamin B6, typically containing between 0.06 and 3.2 mg per serving, with a median value of 0.36 mg. Some products, such as meal replacement nutritional beverages, contain significantly higher amounts (14–17 mg per serving), whilst some hot beverages marketed for relieving premenstrual symptoms were found to contain up to 65 mg per serving (5). Notably, many fortified food items do not clearly list the B6 content on the nutrition label.

Multivitamins, B-complex formulations, and individual vitamin B6 products are all common sources of supplemental B6. These supplements typically use pyridoxine hydrochloride as the active form, although some may include PLP. Capsules often contain 100 mg of B6, making them widely accessible in high doses. An analysis of 100 multivitamin products revealed that 72% exceeded the National Institutes of Health (NIH) recommended daily amount of 1.7 to 2 mg per capsule (6).

Vitamin B6 is often used on its own to help manage a range of medical conditions. One of its established uses is in alleviating nausea and vomiting during pregnancy, especially when other non-pharmacological measures fail to provide relief (7,8). It is also prescribed in cases of confirmed B6 deficiency, which can result from conditions such as impaired kidney function, autoimmune disorders, chronic alcohol consumption, or the use of specific medications like isoniazid, cycloserine, valproic acid, phenytoin, carbamazepine, primidone, hydralazine, and theophylline (9–16). Additionally, certain rare genetic disorders, known as vitamin B6-dependent metabolic conditions, require treatment with unusually high doses of the vitamin (9,17). Vitamin B6 is utilized in toxicology, especially in situations involving overdoses or poisoning from substances such as isoniazid, ethylene glycol, and hydrazine (18). Although evidence remains inconclusive, B6 has also been explored for off-label use in managing conditions like gestational diabetes, carpal tunnel syndrome, essential hypertension, and premenstrual syndrome (19).

It is well documented that the clinical picture of B6 toxicity presents as a sensory neuropathy, uncommonly complicated by motor neuropathy, skin lesions and cognitive problems. This condition was first identified in humans by Schaumburg et al. in 1983, where 7 adult individuals who ingested massive doses of B6 experienced gradually progressive sensory ataxia and severe distal limb impairment. All patients showed improvement after stopping their B6 supplementation (20). Electrophysiological assessments revealed that sensory nerve action potentials were either absent or significantly diminished, indicating that damage was localized to sensory axons and suggesting that the toxicity was affecting the dorsal root ganglia (21). Several other studies have repeatedly shown the same correlation (21–23). A clinical trial conducted in 1992, in which 5 healthy patients were given different doses of pyridoxine, established that B6 toxicity follows a dose-dependent pattern (24). Symptoms of sensory neuropathy in this triad included unstable gait, numbness in the feet, clumsiness in the hands, and significant impairment of position and vibration sensations in the limbs. While sensory neuropathy is almost always the exclusive manifestation, motor symptoms such as unsteadiness without incoordination or muscle cramps may present. A particular case of this involved a man who consumed an extremely high dose (9.6g) of B6 over an extended period, leading to muscle weakness and motor findings on electrophysiological tests alongside typical sensory symptoms. (25). Another case series also showed that some patients taking B6 supplements presented with motor symptoms, albeit uncommon when compared to sensory ones (26). Cutaneous manifestations, though infrequent, may include dermatitis, photosensitivity, and skin lesions (16). In addition, A clinical trial testing the effect of administering B6 for 10 days to healthy students demonstrated a decline in their memory abilities in a dose-related manner (27). Notably, symptoms subside after supplementation is stopped; however recovery can be incomplete with signs of irreversible damage in some cases.

Our study addresses a critical yet underrecognized issue in the field of neurology, one that contributes to potential misdiagnoses. There is no established prevalence figure for toxic B6 levels, despite its clinical relevance; and as a vitamin, it is not routinely assessed in standard diagnostic panels, leading to overlooked cases of toxicity-induced neuropathies by both neurologists and internists. By

highlighting this gap, our study emphasizes the need for increased awareness and routine screening of vitamin B6 levels to improve diagnostic accuracy and patient outcomes.

II. Materials & Methods

We conducted a retrospective observational study by looking through the electronic medical records of patients who visited the neurology clinic at Burjeel Hospital in Dubai between July 1, 2024, and February 15, 2025. Patients who were 18 years or older and whose serum B6 levels were measured as part of their clinical evaluation were included in the study. Patients with a documented neurological diagnosis that explained their symptoms, such as multiple sclerosis (MS), Guillain-Barré syndrome (GBS), or any other ailment, or who were younger than 18 years old, were not allowed to participate in the study.

We gathered information on gender, diabetes status, hemoglobin levels, serum vitamin B12 (B12) and thyroid-stimulating hormone (TSH) levels, and whether or not sensory, motor, and cognitive symptoms were present for each patient who met the inclusion criteria. To make the analysis simpler, participants were classified into three groups based on presenting symptoms: sensory, motor, and cognitive (general).

Regarding examination and analysis of nerve conduction studies (NCS), the number of cases were not statistically significant to be included due to private sector and financial limitations. Therefore, this study is retrospective not prospective. In addition, this study is oriented towards the doctors who practice in medicine, aiming to encourage them to list vitamin B6 toxicity as a main differential diagnosis in the absence of any known neurological disorder.

Since the attending neurologist determined the necessary tests on a case-by-case basis, the diagnostic tests varied from patient to patient. Vitamin B6 levels were determined using HPLC, and the lab advised that the reference level should be between 3 and 37.7 µg/L.

To look into potential relationships between serum B6 levels and the acquired clinical and laboratory data, we conducted statistical analyses. Due to the scarcity of information on the prevalence of B6 toxicity, both locally and internationally, no preliminary sample size determination was carried out; instead, the sample size was established based on the data gathered throughout the study period. The R programming language (version 4.4.2) was used to analyze the data, and $p<0.05$ was considered statistically significant.

III. Results

Four patients were eliminated from the study based on the previously indicated criteria, leaving 68 patients in total, 40 of whom were female (58.8%) and 28 of them being male (41.2%). Our study population's median age was 44. Diabetes was present in 19.1% of the population (n = 13). With a median of 20.6 (95% CI: 14.95 – 30.45) and a range of 3.7 – 153 µg/L, the serum B6 concentration was within the recognized reference range of 3–37.7 µg/L. 30.9% (n = 21) of the entire group had elevated B6 levels (>37.7µg/L). The Kolmogorov-Smirnov test, which was used to determine if serum B6 levels were within normal range, revealed abnormal findings ($p = 0.007982$). Thus, Mann-Whitney U tests were performed for all of the categorical independent variables, such as gender, diabetes status, and symptoms. Age, hemoglobin, TSH, and B12 levels are examples of continuous independent variables. The assumptions of simple regression were examined, and if they were satisfied, simple regression was performed using Pearson's correlation test. If not, Spearman's correlation was completed. Age and circulating B6 levels did not significantly correlate ($p = 0.07068$, Spearman's rho: 0.22). The association was modest even though the p-value was near the significant level. Despite having higher B6 levels than males (median = 18.8 µg/L, 95% CI: 12.85 – 35.0), females (median = 26.6 µg/L, 95% CI: 13.2 – 33.45) did not differ significantly ($p = 0.8608$). Similarly, B6 levels did not approach statistical significance ($p = 0.2873$) even though they seemed to be greater in diabetic patients (median = 30.6 µg/L, 95% CI: 17.0 – 65.05) than in non-diabetic people (median = 17.9, 95% CI: 13.0 – 28.35).

Similarly, relationships between B6 levels and laboratory tests (hemoglobin, B12, and TSH) were examined. For instance, there is no discernible correlation in hemoglobin levels and B6 levels ($p = 0.8164$, Spearman's rho: 0.05).

After confirming assumptions of linear regression, no statistically significant correlation was observed between B6 levels and TSH ($p = 0.09411$, Pearson's correlation: 0.432716). While the near-significant trend suggests a possible relationship, further studies with larger sample sizes may be necessary to clarify any underlying connections between vitamin B6 metabolism and thyroid function.

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Figure 1: The Correlation between Age and Serum B6 levels.

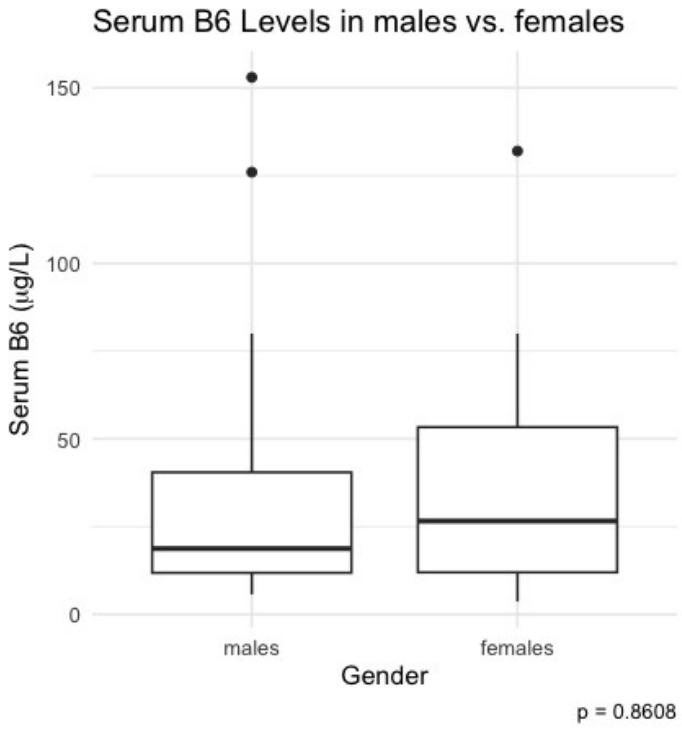

Figure 2: Serum B6 levels in males vs. females

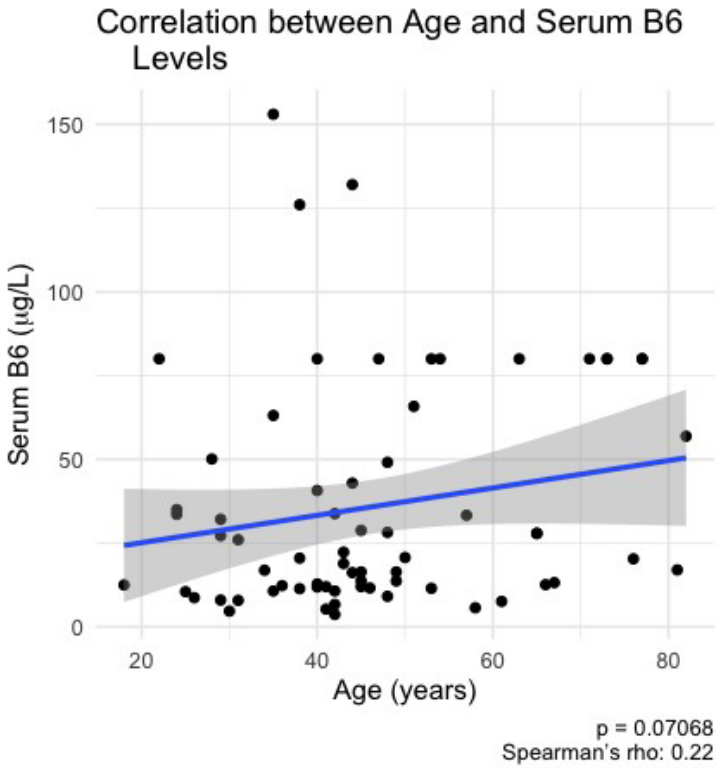

Figure 3: Serum B6 Levels in Diabetics vs. Non-diabetics

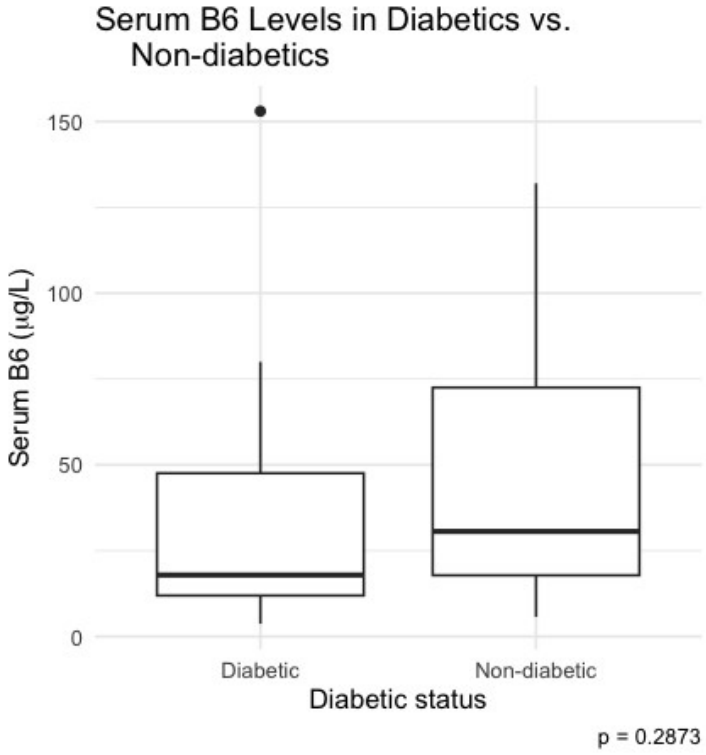

Figure 4: Correlation between Hemoglobin and Serum B6 levels.

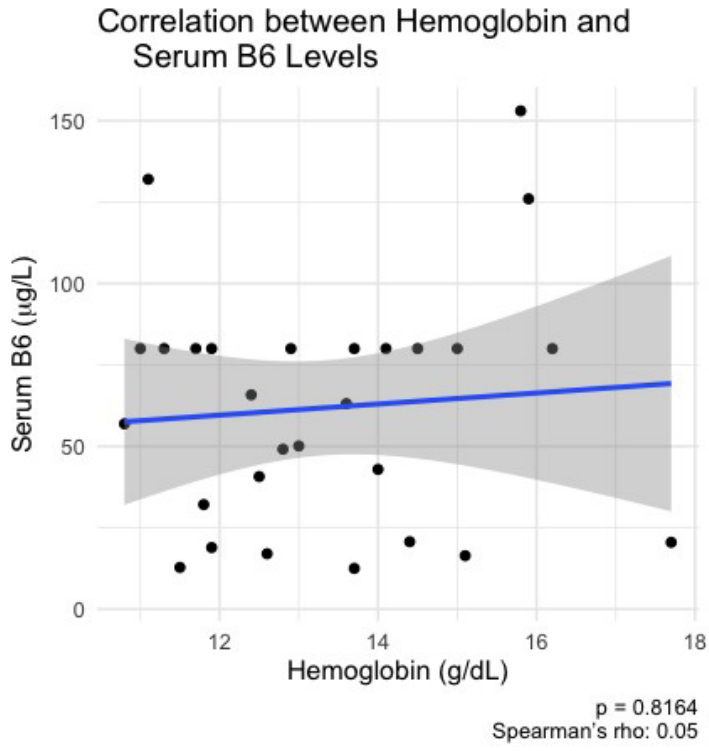

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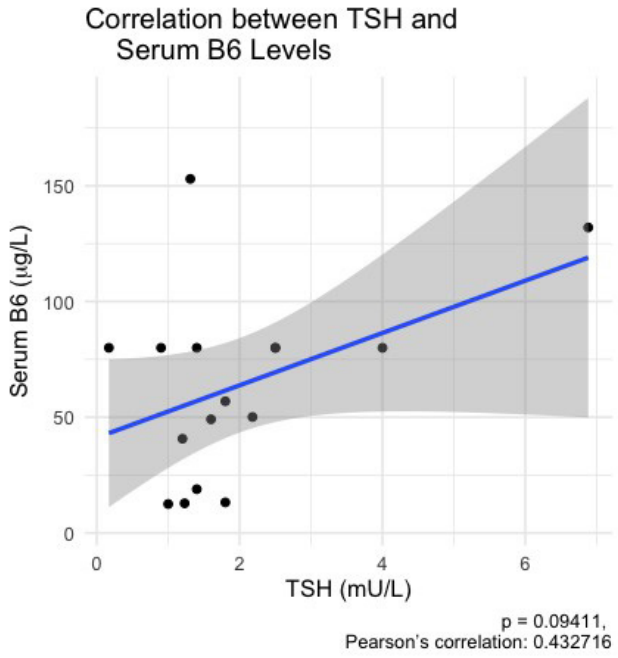
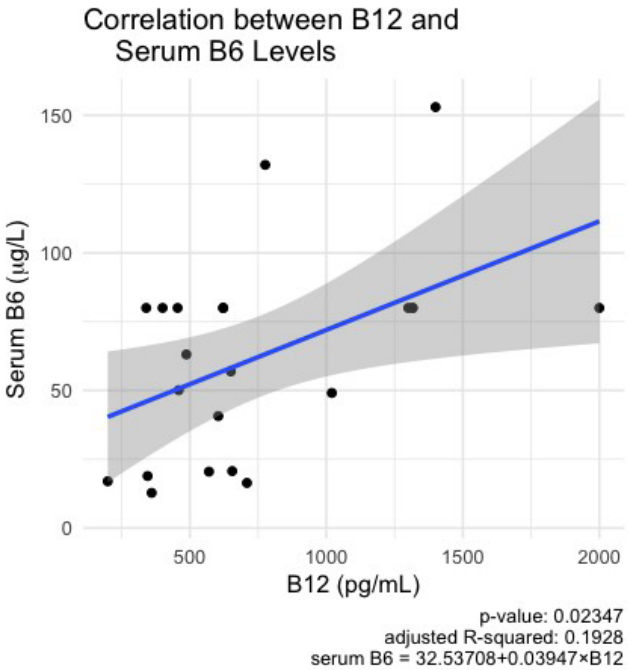
Figure 5: Correlation between TSH and Serum B6 Levels



The linear regression assumptions for vitamin B12 were validated. On the other hand, a statistically significant positive correlation between vitamins B6 and B12 levels was found (p = 0.02347, adjusted R2 = 0.1928), indicating that 19% of the variation in B6 levels could be explained by B12 levels. According to the regression equation that was derived, serum B6 = 32.53708 + 0.03947 × B12, there was a 0.039 increase in B6 for every 1 pg/mL increase in B12. Our conclusion was further supported by a Pearson correlation study, which revealed a moderately favorable association between serum B6 and B12 levels (r=0.48, 95% CI: 0.07 – 0.75).

Figure 6: Correlation between B12 and Serum B6 Levels



Regarding neurological manifestations, there were no discernible variations in B6 levels between patients who had sensory symptoms (p = 0.7958) or cognitive symptoms (p = 0.4407), indicating that B6 levels by themselves might not be a reliable indicator of the occurrence of these symptoms. Remarkably, there was a statistically significant correlation (p = 0.02517) between increased B6 levels and motor symptoms. Vitamin B6 levels were greater in patients with motor symptoms (median = 41.4, 95% CI: 27.9 – 80.0) than in those without them (median = 16.6, 95% CI: 12.5 – 29.65).

Figure 7: Serum B6 Levels in Patients with vs. without Sensory Symptoms

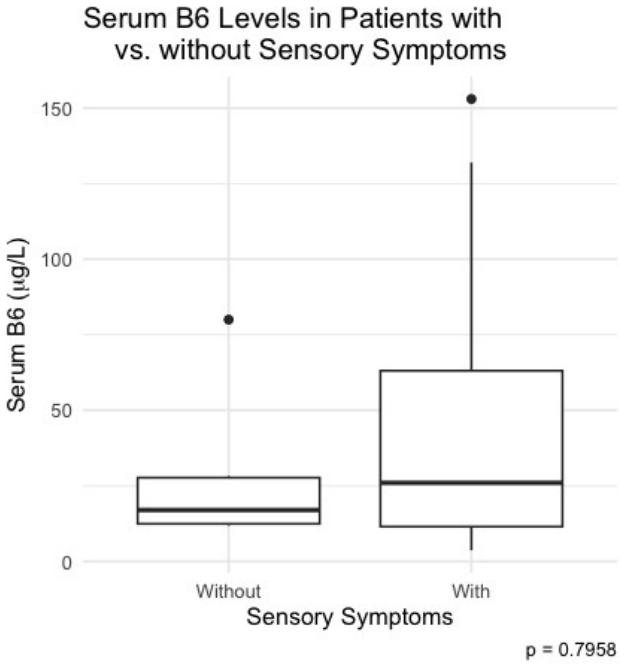
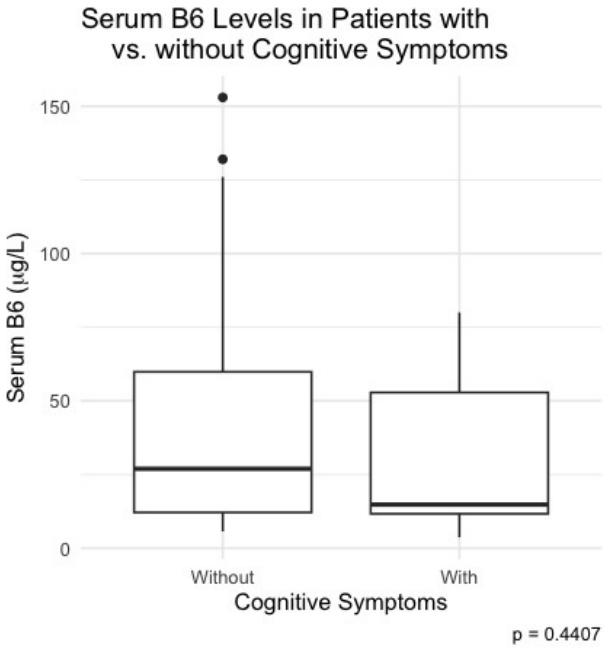


Figure 8: Serum B6 Levels in Patients with vs. without Cognitive Symptoms



International Journal of Medical Discoveries
Volume 1, Issue 2
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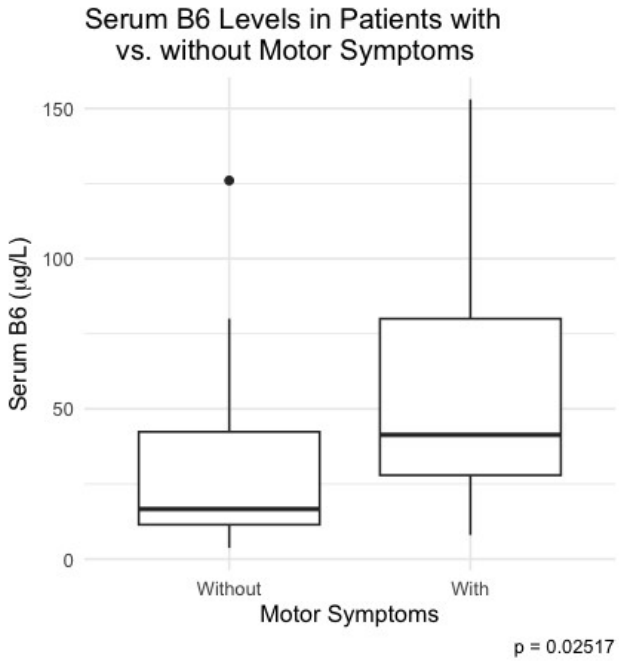
Figure 9: Serum B6 Levels in Patients with vs. without Motor symptoms



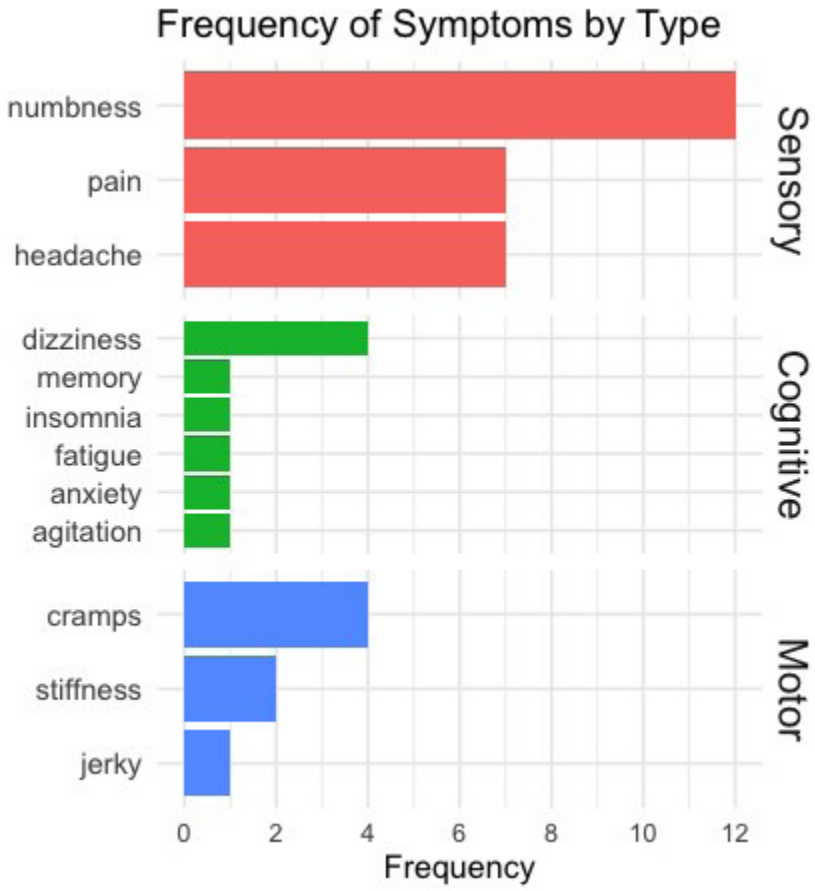
Vitamin B6 toxicity and sensory dysfunction are strongly associated, as seen by the 95.2% of patients with toxic B6 levels (n=21) who also had sensory symptoms. When symptom patterns were further stratified, 42.9% of patients had only sensory symptoms, whereas 57.1% had mixed neurological presentations, which included sensory symptoms along with cognitive or motor problems. Numbness (n = 12), dizziness (n = 4), and muscle cramps (n = 4) were the most prevalent sensory, cognitive, and motor complaints, respectively. All patients experienced lower limb numbness, while two patients experienced additional numbness in their upper limbs. Other limb-related symptoms, such as soreness and cramping in the muscles, were limited to the lower limbs. In every case, there was no particular pattern of distribution to the sensory symptoms.

Figure 10: The Distribution of Toxicity Effects

Distribution of Toxicity Effects

Sensory	Cognitive	Motor	Count	Percentage (%)
Yes	No	No	9	42.9
Yes	No	Yes	6	28.6
Yes	Yes	No	5	23.8
No	Yes	Yes	1	4.8

Figure 11: The Frequency of Symptoms by Type (Sensory, Cognitive and Motor)



IV. Discussion

With an emphasis on the correlation between serum vitamin B6 levels and neurological symptoms, laboratory measures, and demographic traits, our study sheds light on the epidemiological and clinical aspects related to B6 levels. The positive link between serum B6 and B12 levels was a noteworthy exception to our findings, which did not show significant relationships between B6 levels and a number of independent variables. Furthermore, we found a robust correlation between B6 toxicity and sensory impairment, as well as a significant relationship between increased B6 concentrations and motor symptoms.

Our cohort's median serum B6 concentration was 20.6 µg/L, which is within the recognized reference range of 3–37.7 µg/L. However, B6 toxicity, which is defined as values greater than 37.7 µg/L, was present in a significant percentage of subjects (30.9%). Because excessive B6 levels demonstrate a well-established role in neurotoxicity, the incidence of B6 toxicity in our group raises questions regarding possible subclinical or overt neurological repercussions. A trend toward higher B6 concentrations in females and diabetics was seen, despite the fact that we could not identify a statistically significant difference in B6 levels based on gender or diabetes status. To definitively determine if these tendencies are statistically and clinically significant, larger sample sizes may be required. The modest correlation (Spearman's rho: 0.22) indicates that age alone is unlikely to be a powerful predictor of B6 concentration, even if there is a near-significant trend between B6 levels and age ($p = 0.07068$). Likewise, no noteworthy correlations were discovered between hemoglobin or TSH levels and B6 levels. To find out if thyroid function affects B6 metabolism, more research with larger sample sizes and controlled study designs is necessary, even if the near-significant trend between B6 and TSH levels ($p = 0.09411$) may indicate a potential physiological association. The positive correlation between serum B6 and B12 levels ($p = 0.02347$, $r = 0.48$) was one of our study's most important conclusions. About 19% of the variation in B6 levels was explained by B12 levels, according to our regression model.

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This correlation is consistent with earlier studies that indicate B6 and B12 have related functions in metabolic pathways, specifically in the metabolism of amino acids and the control of homocysteine (28). However, despite the former explanation of their correlation, we found that their coexistence in widely consumed multivitamin supplements and injections is another valid factor to their concurrent increase. The causality and directionality of this link is still unknown, though. Future research should investigate if this correlation is caused by common dietary sources, metabolic interactions, or underlying pathophysiological mechanisms. Our findings support the known connection between B6 toxicity and sensory impairment in terms of neurological symptoms. The most common sensory complaint reported by nearly all individuals (95.2%) with B6 toxicity was numbness. This result is consistent with previous research showing that sensory neuropathy is a defining feature of B6 toxicity (5,20,21,23,25). Remarkably, we also found a strong correlation ($p = 0.02517$) between increased B6 levels and motor symptoms. The median B6 levels of patients with motor symptoms were significantly greater (41.4 µg/L) than those without motor symptoms (16.6 µg/L). This new finding raises the possibility that B6 toxicity plays a role in motor dysfunction, which calls for more research into the pathophysiological mechanisms underlying it. In particular, there was no obvious correlation between B6 levels and cognitive symptoms in our investigation. This could imply that cognitive impairment is more influenced by other factors than only B6 levels, such as comorbidities, underlying neurological diseases, or genetic predispositions. To clarify any modest effects of B6 dysregulation on cognitive function, further studies involving neuroimaging and cognitive evaluations may be required, given the wide range in cognitive symptom presentation and possible multifactorial implications.

The mechanisms behind vitamin B6-related toxicity and peripheral neuropathy are currently being studied. There are four primary theories about the pathophysiology of vitamin B6-induced peripheral neuropathy and neurotoxicity. According to the first theory, the aldehyde group present in both pyridoxal (PL) and pyridoxal-5'-phosphate (PLP) causes toxicity. However, PLP is typically linked to proteins, reducing its potential toxicity, while PL predominantly resides at physiological pH in a chemically inert hemiacetal form. Research indicates that PL or PLP's aldehyde groups may not be the exclusive cause of the neurotoxic effects (29). The secondary theory proposes that pyridoxine irradiation produces quinone methide-type intermediates, which could be a factor in toxicity. Since the toxicity of quinones is more widespread and not limited to neurons, this was also seen as an improbable contributing factor (29). The third theory suggests that high levels of plasma pyridoxine (PN) may prevent PLP from binding to its target enzymes, which could result in low serum B6 levels and consequent neuropathy that resembles B6 insufficiency (30). The fourth theory, which is currently considered the most reliable as it best explains the pathophysiology leading to toxic serum B6 levels, emphasizes that increased excitability in dorsal root ganglia and subsequent neuronal degeneration may result from mutations of the pyridoxal kinase gene, which deplete PLP and hinder the manufacture of neurotransmitters, including GABA. A similar clinical picture may result from pyridoxal kinase inhibition brought on by high PN levels (29). A noteworthy illustration of the need for additional research is the clinical presentation of vitamin B6-related neuropathy, which partially overlaps with that of vitamin B6 insufficiency. The acceptable daily limit of B6 intake is up for debate; as a result, according to various criteria, the upper limit (UL) varies from 10 mg/day to 100 mg/day. As of 2024, the adult UL in the United States (US) has remained at 100mg/day since its establishment in 1998 (16). A scientific opinion on the acceptable upper intake limits of B6 was published in 2023 by the European Food Safety Authority's (EFSA) Panel on Nutrition, Novel Foods, and Food Allergens. The panel established an upper limit of 12 mg/day of B6 for all individuals, including those who are pregnant or nursing, based on systematic reviews that looked at relationships between vitamin B6 and peripheral neuropathy (5). According to Dubai Municipality recommendations, the UL in the United Arab Emirates is established at 100 mg/day, which is comparable to the US recommendations.

As mentioned previously, peripheral neuropathy was the first known clinical manifestation of B6 toxicity in 1983 (20). Since then, numerous articles have discovered a similar pattern of toxicity. Such toxicity requires a daily dose that is higher than the established UL. On the other hand, food typically supplies up to 5 mg per day and typically 1.4 to 3.1 mg per day of B6 (4,5). These values fall below the upper limits specified in any of the previously listed standards. However, US consumers intake at least five times as much vitamin B6 from pills as from food (31). The effects of elevated B6 vitamers on human-derived cells were assessed in vitro in a previous study. The results showed that although the other vitamins were non-toxic,

pyridoxine caused cell death in a dose-dependent manner (30). This further supports that pyridoxine that is present in supplements is the most likely cause of the toxicity. There were no official dietary assessments in our study, but based on our clinical experience, every patient with toxic B6 levels mentioned getting their B6 from non-dietary sources such as supplements or fortified goods. Furthermore, our experience indicates that most people with B6 toxicity recover in around two months after stopping excessive intake, although we have not performed a systematic follow-up on them.

Common non-specific symptoms of vitamin B6 toxicity include burning, paresthesia, or numbness in the extremities, caused by peripheral neuropathy. Underdiagnosis or misinterpretation results from these symptoms' frequent comorbidity with other illnesses, such as diabetes and alcohol-related neuropathies (32). This diagnostic problem is greatly exacerbated by inadequate patient history taking and a lack of understanding among healthcare practitioners. To diagnose B6 toxicity, a comprehensive medical history is essential. In addition to thorough medical, occupational, and environmental exposure histories - physicians, nurses and even pharmacists should investigate the usage of dietary supplements, including over-the-counter vitamins. This can be done by checking medication lists, speaking with patients, or highlighting possible causes of high-dose B6 intake. Early detection and diagnosis can be improved by training medical practitioners on vitamin B6 toxicity, and will help avert cases of misdiagnosis. Collectively, these points emphasize how crucial it is to manage this uncommon yet potentially crippling illness collaboratively and as a team.

Our study adds to the body of knowledge on B6 metabolism and toxicity by offering insightful information on the epidemiological and clinical aspects linked to serum B6 levels. The inclusion of a well-characterized cohort, which enables a thorough examination of demographic, laboratory, and neurological variables, is a significant strength. Furthermore, in order to minimize confounding variables and note possible relationships, we used strong statistical techniques, such as regression models and correlation. Our results are more reliable and comparable to those of earlier research when a standardized reference range for B6 toxicity is used. Furthermore, our research reveals a new link between high B6 levels and motor symptoms, a topic that has not received much attention in previous studies. Future research on the molecular underpinnings of B6-related neurotoxicity beyond sensory impairment may be conducted based on our findings. Additionally, our study highlights the possible metabolic linkages between B6 and B12, indicating the need for additional research into their common physiological pathways. We further proposed that this possible connection results from their frequent presence in supplements, which is why we support more stringent regulation of these ingredients. Notwithstanding these advantages, our study contains a number of drawbacks. First, the near-significant trends for age and TSH levels suggest that our sample size may have hindered our ability to detect weaker relationships, hence why more conclusive results could be obtained from a bigger population. Another drawback is the absence of information on dietary intake, which would have made it easier to distinguish between toxicity from supplements and endogenous fluctuations in B6 levels. Additionally, the use of self-reported neurological symptoms raises the risk of recall bias, which may compromise the precision of estimations of symptom prevalence.

To conclude things, there was no patient follow-up in the trial, which would have shown the length of recovery and long-term results. To further validate our findings, future research should use objective neurological measures such as nerve conduction investigations and encompass a variety of demographics.

V. Conclusion

This study emphasizes the important yet often overlooked effects of toxic vitamin B6 levels on brain and nerve intactness. Given that a significant percentage of patients had B6 toxicity, our results highlight the urgent need for healthcare professionals to be more cognizant while evaluating neuropathic symptoms. While finding a link with development of motor symptoms points to the need for more investigation into B6 toxicity's wider neurological effects, the high correlation between B6 toxicity and sensory impairment is consistent with previous research.

In the future, we aim to conduct prospective studies that follow the American Academy of Neurology (AAN) guidelines to diagnose Mega B6 syndrome, which has taken into account the mandatory testing for NCS, for instance.

Because vitamin B6 is commonly found in dietary supplements, individuals with unexplained neuropathies should be screened on a regular basis to avoid misdiagnosis and treatment delays. Minimizing the hazards associated with excessive supplementing requires

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Volume 1, Issue 2
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increasing awareness amongst the general population and professionals. In parallel, prospective cohorts should be the main focus of future research in order to provide more precise clinical recommendations and guarantee safer vitamin B6 intake in the general population.

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