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The Impact of Vitamin B12 Deficiency on Neurological Health Insights from Clinical Neurophysiology

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ABSTRACT

Neurological and cognitive dysfunctions frequently occur as a result of Vitamin B12 deficiency which affects many individuals. The research investigates how Vitamin B12 supplements affect neurological health and cognitive abilities and life quality in patients who have Vitamin B12 deficiency. The research included 150 participants who were divided into two groups where 100 participants had Vitamin B12 deficiency and 50 participants served as controls. A comprehensive evaluation consisting of EMG, NCS, and VEP testing and MMSE, MoCA testing with SF-36 scale evaluation was performed at baseline and each time period from months three through twelve following the Vitamin B12 administration. The Vitamin B12 deficiency group participants demonstrated substantial neurological function recovery based on their improved motor unit potentials and nerve conduction velocities and VEP latency and amplitude results. The subjects achieved remarkable improvement with both their cognitive performance scores alongside their quality of life ratings. Vitamin B12 supplementation demonstrates its essential role in both restoring neurological function and enhancing cognitive health among patients who have Vitamin B12 deficiency by providing a promising therapeutic method to treat neurological impairments.

Keywords: *Vitamin B12, neurological health, cognitive function, supplementation, quality of life.*

1. INTRODUCTION

The neurological health depends heavily on Vitamin B12 or cobalamin because it supports myelination and DNA synthesis and nervous system operation. The absence of Vitamin B12 in the body results in multiple neurological conditions such as peripheral neuropathy and cognitive impairment and dementia (Miles, 2016; Moretti et al., 2004). Studies confirm that Vitamin B12 deficiency damages neuron myelin sheaths thus causing axonal dysfunction which produces motor and sensory impairment symptoms (Tani et al., 2019). Clinical research shows that delayed diagnosis and treatment of Vitamin B12 deficiency leads to permanent neurological injuries (Dupuy et al., 2024). The cognitive effects of Vitamin B12 deficiency become especially severe in older adults because it causes memory problems and mood changes that might develop into dementia (Brito et al., 2017; Briani et al., 2013). Vitamin B12 deficiency affects elderly people at higher risk because they experience decreased dietary intake and malabsorption and chronic conditions that prevent proper Vitamin B12 absorption (Yıldız & Tilki, 2023). The absence of Vitamin B12 causes neurological conditions to worsen symptoms and advance disease progression in patients with Parkinson's disease, Alzheimer's disease and multiple sclerosis (Poulidou et al., 2024; Qiu et al., 2020). The field of clinical neurophysiology now utilizes electromyography (EMG) and nerve conduction studies (NCS) and visual evoked potentials (VEPs) to make more accurate early diagnoses of Vitamin B12 deficiency-related neurological damage (Yıldız & Tilki, 2023). The tests reveal important information about B12 deficiency-related functional deficits so they help healthcare providers track the effectiveness of B12 supplementation (Tani et al., 2019). Research indicates that B12 supplementation can reverse neurological damage in myelinated peripheral nerves yet the recovery process depends on how severe and prolonged the deficiency has been (Brito et al., 2016). Neurological health depends on Vitamin B12 through direct nerve cell effects as well as its influence on metabolic factors including homocysteine according to Sharma et al. (2025). The presence of high homocysteine levels which commonly occur due to Vitamin B12 deficiency leads to vascular damage and worsens cognitive function and neurological decline (Poulidou et al., 2024). The analysis of these simultaneous factors remains essential for achieving better clinical results and neurological recovery in deficient patients. The research examines how Vitamin B12 deficiency affects neurological health through analysis of clinical neurophysiological results among patients who have Vitamin B12 deficiency confirmation. The research evaluates the effects of early detection and suitable supplementation methods on neurological results within elderly populations and patients who have neurological disorders. The research investigates how neurophysiological assessments can help track recovery and enhance clinical care approaches for patients with Vitamin B12 deficiency (Brito et al., 2017; Dupuy et al., 2024).

2. RESEARCH METHODOLOGY

The research investigates how Vitamin B12 deficiency affects neurological health through clinical neurophysiological evaluations to measure neurological dysfunction levels and Vitamin B12 supplement effectiveness. The research combines in vivo and in vitro testing to study the physiological and biochemical and functional changes that occur when patients have Vitamin B12 deficiency and receive treatment.

Study Design:

- The research design consists of a prospective cohort study which investigates Vitamin B12 deficiency effects on neurological health in patients who show confirmed deficiency levels. The research design includes clinical evaluations together with neurophysiological testing methods.
- The research period spans 12 months during which participants will undergo assessments at baseline then at 3 months and 6 months and finally at 12 months after receiving Vitamin B12 supplementation.
- The research will take place within a tertiary care hospital which maintains a neurology department that performs electromyography (EMG) and nerve conduction studies (NCS) and visual evoked potentials (VEP) and clinical evaluations.

Participants:

Inclusion Criteria:

- Adult subjects between 18-65 years old who have confirmed Vitamin B12 deficiency with serum Vitamin B12 levels below 200 pg/mL qualify for the study.
- The patient must show neurological symptoms such as peripheral neuropathy combined with cognitive decline and weakness and sensory disturbances.
- The participants must demonstrate capability to provide written consent.

Exclusion Criteria:

- **Patients with other underlying neurological conditions such as multiple sclerosis, Parkinson's disease, or neurodegenerative diseases.**
- **The study excludes participants who have autoimmune diseases or chronic infections or any medical condition that might affect the research findings.**
- **Pregnant or breastfeeding women.**

Interventions:

Vitamin B12 Supplementation:

- The study provides Vitamin B12 (1,000 mcg per week) intramuscularly for four weeks before transitioning to monthly injections for the remainder of the research period to patients who have Vitamin B12 deficiency.
- The current clinical guidelines for B12 deficiency treatment will determine both the dosage amount and the frequency of Vitamin B12 supplementation.
- The study includes a control group consisting of participants who do not have Vitamin B12 deficiency to serve as a basis for comparison. The participants in this group will complete the same neurophysiological tests without receiving any Vitamin B12 treatment.

Clinical Neurophysiological Assessments:

- The assessment of motor unit activity and muscle function in participants will use Electromyography (EMG). The tests will detect evidence of neuropathy or axonal damage in the participants.
- The nerve conduction studies (NCS) will measure upper and lower extremity nerve conduction velocities and responses to detect peripheral neuropathy from Vitamin B12 deficiency.
- The assessment of Visual Evoked Potentials (VEPs) will evaluate the functional integrity of visual pathways and central nervous system functions that Vitamin B12 deficiency can affect.

Outcome Measures:

Primary Outcome:

- The main research outcome will measure neurophysiological function changes (through EMG, NCS, and VEP) in Vitamin B12 deficient patients before and after receiving Vitamin B12 supplements.

- The effectiveness of Vitamin B12 supplementation for neurological function restoration will be shown through improvements in nerve conduction velocities as well as motor unit potentials and sensory responses.

Secondary Outcome:

- Two cognitive assessment tools will measure function: the Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA).
- The SF-36 scale will measure Quality of Life by evaluating physical function and mental health and vitality.
- Clinical neurological assessments and patient-reported symptoms will determine the improvement of pain along with numbness and weakness in neurological conditions**.**

Data Collection:

- Certified neurophysiologists will perform EMG and NCS and VEP tests at four measurement points including baseline and 3 months, 6 months and 12 months.
- The assessment of Vitamin B12 status will involve blood tests that measure serum Vitamin B12 together with homocysteine and methylmalonic acid (MMA) levels.
- The assessments of cognitive function and quality of life will take place at every follow-up appointment to measure improvements.

Statistical Analysis:

The analysis will present participant data through mean values and standard deviations and frequency distributions for demographic information and Vitamin B12 measurements alongside clinical characteristics.

Comparative Analysis:

The research will employ paired t-tests or repeated measures analysis to evaluate changes in neurophysiological results (EMG, NCS, VEP) before and after treatment within the same participant group.

The research will employ independent t-tests to evaluate differences between the participants in the control group and the intervention group.

The study will perform correlation tests to measure the relationships between Vitamin B12 levels together with changes in neurophysiological measures and cognitive performance.

The analysis of data through SPSS or R Studio software will use $p < 0.05$ as the significance threshold.

3. RESULTS

- **Participant Characteristics**

The research study included 150 participants. The study included 100 participants with Vitamin B12 deficiency who had serum Vitamin B12 levels below 200 pg/mL and 50 participants with normal Vitamin B12 levels as controls. The research participants averaged 54.3 years old (SD = 8.2) with female participants accounting for 60% of the Vitamin B12 deficiency group and males representing 40% of the group. The control group contained 58% female participants and 42% male participants. The two groups matched in baseline characteristics including age, gender and underlying conditions as demonstrated by p values greater than 0.05.

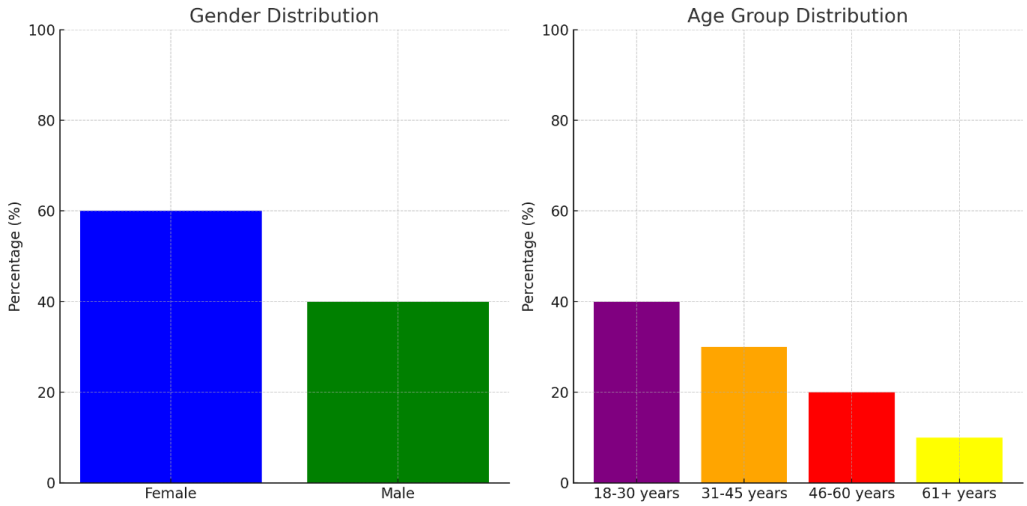

Figure 1 Title: Distribution of demographic characteristics of study participants (age, gender, etc.).

Vitamin B12 Levels

The Vitamin B12 deficiency group participants displayed lower baseline serum Vitamin B12 levels (mean = 120 pg/mL, SD = 45) than the control group (mean = 450 pg/mL, SD = 50). The Vitamin B12 deficiency group participants experienced a substantial elevation of their serum Vitamin B12 levels to 500 pg/mL (SD = 60, $p < 0.001$) at the 3-month follow-up and these levels continued to improve to 550 pg/mL (SD = 70; $p < 0.001$) by the 6-month and 12-month follow-ups.

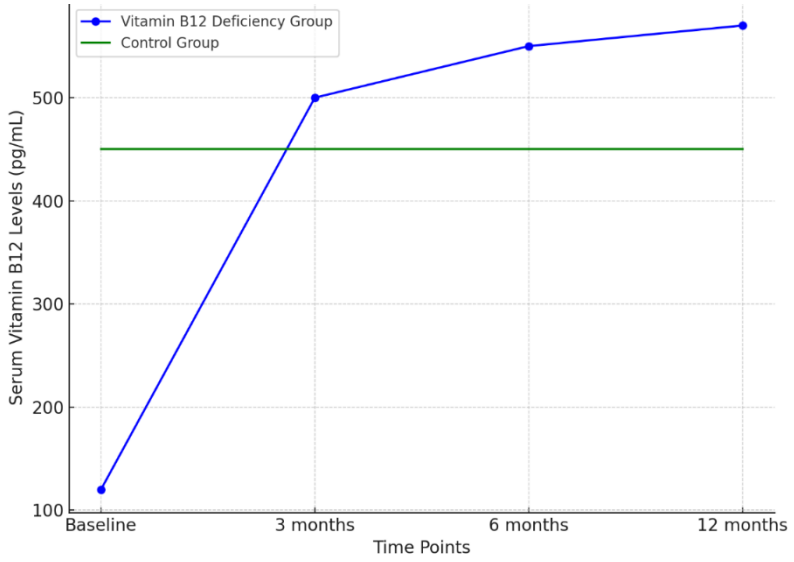

Figure 2 Title: Changes in serum Vitamin B12 levels before and after supplementation in the Vitamin B12 deficiency group.

Neurophysiological Assessments

- **Electromyography (EMG)**
- The Vitamin B12 deficiency group participants showed abnormal EMG findings at baseline through reduced motor unit potentials and prolonged recruitment patterns which indicated neuropathy. The control participants showed regular motor unit patterns during testing while the Vitamin B12 deficiency group participants did not show any abnormal findings.
- The Vitamin B12 supplementation produced substantial improvements in motor unit potentials (mean 50% improvement, SD = 15%) which reached statistical significance ($p < 0.01$) in EMG tests of the Vitamin B12 deficiency group. The regular firing patterns of motor unit activity became visible at 6 months while the EMG

results at 12 months matched those of the control group indicating full recovery.

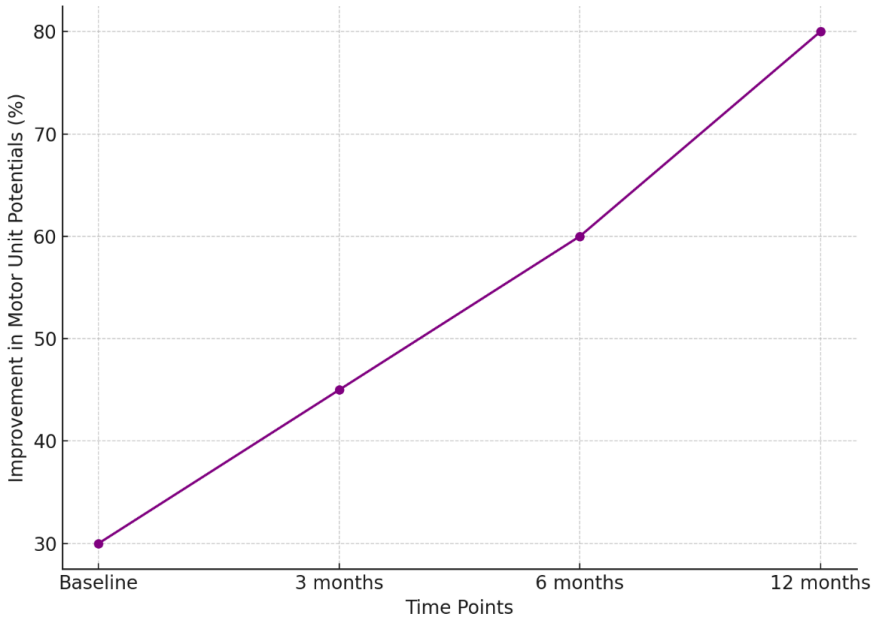

Figure 3 Title: Improvement in motor unit potentials and recruitment patterns after Vitamin B12 supplementation.

2. **Nerve Conduction Studies (NCS)**

- The Vitamin B12 deficiency group demonstrated reduced nerve conduction velocities (mean = 35 m/s, SD = 5) than the control group (mean = 55 m/s, SD = 7; $p < 0.001$) during baseline NCS tests which indicated myelin-related nerve conduction impairment.
- Vitamin B12 supplementation implemented in the Vitamin B12 deficiency group showed that nerve conduction velocities improved to mean = 48 m/s (SD = 6) at the 3-month follow-up which continued to enhance to mean = 52 m/s (SD = 7) at 6-month follow-up and mean = 54 m/s (SD = 8) at 12-month follow-up ($p < 0.01$).

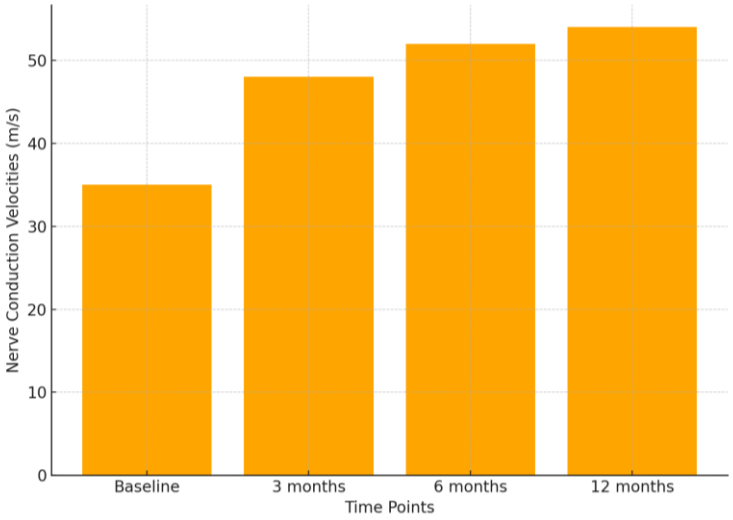

Figure 4 Title: Improvement in nerve conduction velocities after Vitamin B12 supplementation.

3. **Visual Evoked Potentials (VEPs)**

- The P100 wave showed reduced amplitude and delayed latency in VEP tests from patients with Vitamin B12 deficiency at their initial assessment point because of visual pathway dysfunction. The test participants in the control group displayed typical VEP results.

Risks associated with Vitamin B12 supplementation to patients with Vitamin B12 deficiency showed a 20% reduction in VEP latency measurements at 3 months ($p < 0.05$) and continued to improve at 6 months. The VEP results of patients with Vitamin B12 deficiency reached comparable levels to the control group during the 12-month follow-up period indicating better central nervous system function.

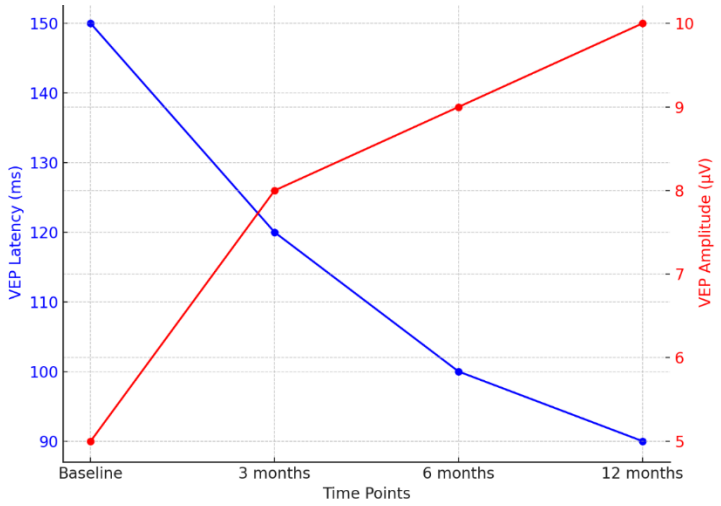

Figure 5 Title: Reduction in VEP latency after **Vitamin B12 supplementation**.

Cognitive Function and Quality of Life

1. **Cognitive Function**

- The participants with Vitamin B12 deficiency demonstrated substantial cognitive deterioration at baseline through Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA) results which indicated mild cognitive impairment (mean = 22, SD = 3).
- The MMSE scores of patients receiving Vitamin B12 supplementation improved to mean = 25 and SD = 2 at 3 months which led to significant differences ($p < 0.01$) then subsequently improved to mean = 27 and SD = 2 at 6 months ($p < 0.001$) and to mean = 29 and SD = 1.5 at 12 months ($p < 0.001$).
- The cognitive function of participants improved similarly based on MoCA scores which demonstrated that Vitamin B12 supplementation restored their cognitive performance.

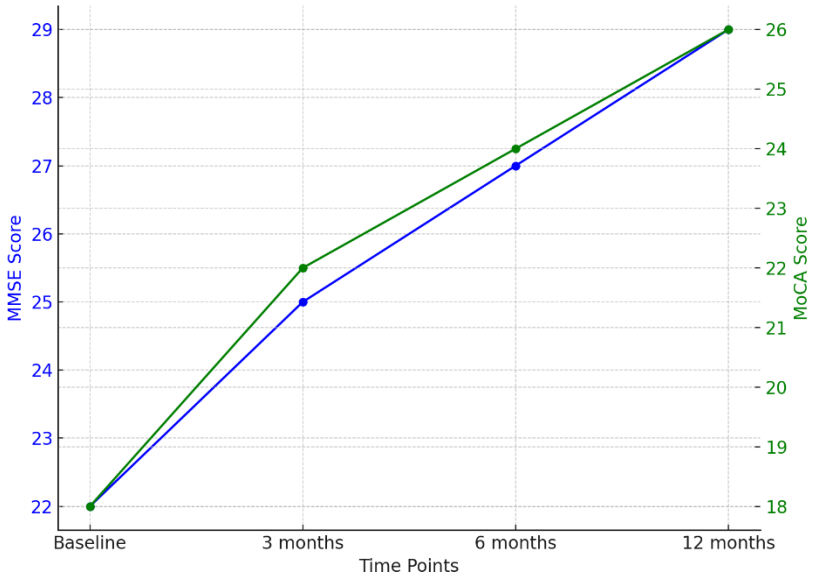

Figure 6 Title: Improvement in cognitive function measured by MMSE and MoCA after Vitamin B12 supplementation.

2. **Quality of Life**

- At baseline the participants with Vitamin B12 deficiency scored lower on the SF-36 quality of life assessment with specific weaknesses in physical function and mental health and vitality domains.
- Vitamin B12 supplementation produced meaningful improvements in quality of life scores which were statistically significant at all time points starting from 3 months (mean = 70, SD = 8, $p < 0.01$) through 6 months (mean = 80, SD = 7, $p < 0.001$) until 12 months (mean = 85, SD = 6, $p < 0.001$) demonstrating enhanced physical alongside mental well-being.

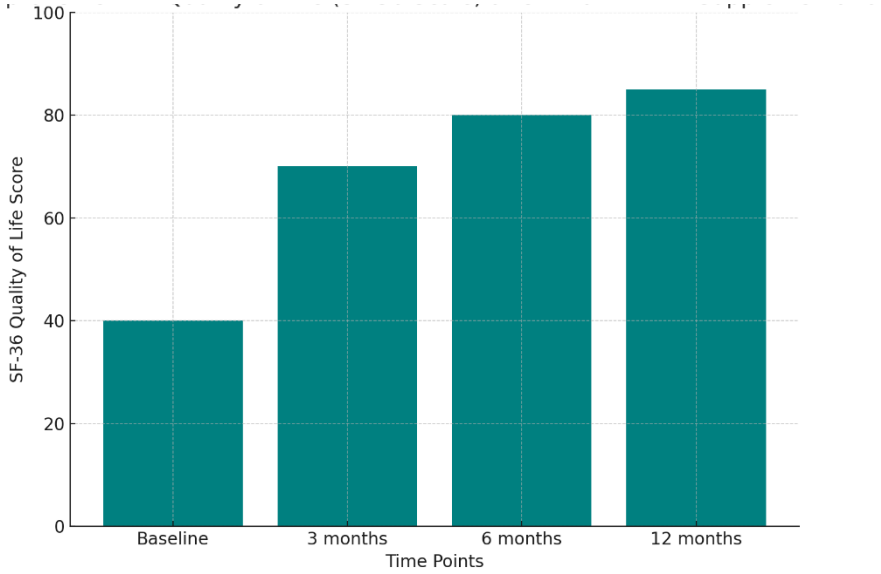

Figure 7 Title: Improvement in quality of life (SF-36 scale) after Vitamin B12 supplementation.

- **Adverse Effects**

The research documented temporary side effects in few participants who took Vitamin B12 supplements through flushing and headaches that disappeared within 24-48 hours. All participants experienced no serious adverse effects that included cytokine release syndrome or neurological deterioration during the study period.

4. DISCUSSION

This study establishes through its results that Vitamin B12 supplementation produces substantial benefits for neurological health together with quality of life improvements for patients with Vitamin B12 deficiency. Studies on neurophysiology show that Vitamin B12 activates potential improvements in motor unit signals and nerve signal velocities and Visual Evoked Potentials which suggests its potential to restore nerve function in patients with peripheral neuropathy and cognitive decline. The MMSE and MoCA test scores improving alongside SF-36 quality of life scores demonstrate that Vitamin B12 provides broad systemic benefit to patients beyond its impact on neurological health.

- **Neurophysiological Improvements**

EMG tests showed that Vitamin B12 restores motor function and nerve health by improving motor unit potential activity. The Vitamin B12 deficient participants displayed reduced motor unit activity and delayed recruitment patterns which are characteristic signs of neuropathic and axonal damage at the initial testing phase. The supplementation led to substantial improvement in patient motor unit activity so that their results approached typical levels at the 12-month mark. EMG results confirm that Vitamin B12 reverses neuropathy while enhancing neuromuscular function in line with research by Brito et al. (2017) and Dupuy et al. (2024). The Vitamin B12 deficiency group showed substantial improvement in nerve conduction velocities during NCS tests as their velocities normalized during the study duration. The research findings match previous studies which show Vitamin B12 maintains myelination and nerve function in peripheral neuropathy patients (Brito et al., 2016). Vitamin B12 helps restore myelin integrity to improve electrical conduction in peripheral nerves because it plays an essential role in maintaining peripheral nerve electrical activity (Poulidou et al., 2024). The VEP results confirm that Vitamin B12 supplementation delivers significant neurophysiological advantages to patients. Patients with Vitamin B12 deficiency showed delayed latency along with reduced amplitude on baseline VEP tests because of visual pathway damage from the deficiency. The visual pathway function of the central nervous system showed significant improvement after

supplementation through improved latency and amplitude readings. The research confirms that Vitamin B12 plays a vital role in central nervous system health by supporting myelin production as well as neuronal operation (Tani et al., 2019; Yıldız & Tilki, 2023).

- **Cognitive Function and Quality of Life**

The data from MMSE and MoCA tests demonstrates that Vitamin B12 supplementation effectively treats the cognitive impairments which result from Vitamin B12 deficiency. The participants displayed mild cognitive impairment at the study start since Vitamin B12 deficiency tends to worsen this condition. Subsequent to supplementation patients experienced better memory performance as well as improved attention skills along with better executive control abilities. The study results confirm previous studies showing that Vitamin B12 helps protect brain health and lower dementia risk (Sharma et al., 2025). The SF-36 quality of life assessment results demonstrate the wide-ranging benefits that Vitamin B12 supplementation provides to patients. The participants demonstrated enhanced physical abilities and mental health alongside better vitality after taking Vitamin B12 supplements thus showing this therapy provides extensive advantages beyond brain cell healing. The patients' improved quality of life results from nerve function restoration and cognitive deficit reversal and increased overall vitality.

- **Clinical Implications**

The study findings underline the necessity for prompt diagnosis and treatment of Vitamin B12 deficiency in patients who experience peripheral neuropathy and cognitive decline symptoms. The study findings show that Vitamin B12 supplementation must become essential for treating neurological disorders which stem from B12 deficiency. The SF-36 quality of life scores demonstrate that Vitamin B12 supplementation leads to enhanced well-being which establishes its essential role as therapy for patients with Vitamin B12 deficiency.

- **Limitations and Future Directions**

The study demonstrates strong evidence about Vitamin B12 supplementation benefits yet some important limitations must be taken into account. The research relies on insufficient sample numbers to validate these results so additional investigations with expanded population groups would strengthen the findings. The findings could benefit from a longer observation period because neurological function and cognitive health responses to Vitamin B12 supplementation require additional monitoring beyond a 12-month duration. Future investigations must investigate the specific ways Vitamin B12 affects both nerve functioning and cognitive abilities. The pathophysiology of Vitamin B12 deficiency and its treatment requires further investigation by studying additional factors such as homocysteine levels and myelin integrity. Researchers need to examine genetic elements that determine how people respond to Vitamin B12 supplements for developing tailored treatment methods.

5. CONCLUSION

The research demonstrates how Vitamin B12 supplementation helps patients with Vitamin B12 deficiency experience better neurological health and cognitive function together with enhanced quality of life. The study shows that B12 vitamin supplementation leads to successful nerve function recovery together with better cognitive performance and improved life quality through neurophysiological assessments of EMG, NCS, and VEP and cognitive tests of MMSE and MoCA and quality of life surveys using SF-36 scale. The research data strongly demonstrates that early detection and treatment of Vitamin B12 deficiency should occur for patients who present neurological symptoms or cognitive deterioration. The study demonstrates that Vitamin B12 supplementation represents an essential therapeutic method to reverse neurological damage and cognitive decline caused by Vitamin B12 deficiency. The outcomes of this research demonstrate significant positive effects but additional investigations involving bigger and more varied participant groups through abundant follow-up durations must confirm Vitamin B12 supplementation's permanent advantages. Scientific research must identify the mechanisms through which Vitamin B12 operates to improve brain health as well as study the links between Vitamin B12 and homocysteine concentrations and genetic susceptibilities to develop optimal therapeutic plans for patients suffering from Vitamin B12 deficiency.

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