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Research Article

Assessment of Nerve Conduction Abnormalities in Peripheral Vascular Disease: A Clinical and Electrophysiological Correlation Using Nerve Conduction Studies and Ankle-Brachial Index in Patients with Chronic Ischemia

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ABSTRACT
Background: Peripheral Vascular Disease (PVD) is a progressive circulatory disorder characterized by arterial narrowing, leading to reduced blood flow to the extremities. Chronic ischemia associated with PVD can cause significant neurological complications, including peripheral neuropathy. However, distinguishing between neuropathy caused by ischemia and other factors, such as diabetes, remains a challenge. Nerve conduction studies (NCS) serve as an objective tool to evaluate peripheral nerve function in PVD patients. This study aimed to assess nerve conduction abnormalities in PVD patients and correlate findings with disease severity.
Methods: A prospective observational study was conducted on 40 patients diagnosed with PVD at Krishna Hospital, Karad, between May 2011 and May 2013. Patients were classified based on the Fountaine Staging System, and Ankle-Brachial Index (ABI) was used to determine the severity of arterial insufficiency. NCS was performed to evaluate motor and sensory nerve conduction velocities (NCV), compound motor action potential (CMAP), and sensory nerve action potential (SNAP). Patients with diabetes mellitus were excluded from the study to eliminate confounding factors. The relationship between PVD severity and nerve conduction abnormalities was analyzed statistically.
Results: The study population comprised 24 males and 16 females, with a mean age of 59 years (range: 42-78 years). The distribution of patients according to Fountaine Staging revealed that 32.5% had ischemic rest pain (Stage III), and 7.5% presented with ulceration or gangrene (Stage IV). ABI values indicated that 67.5% of patients experienced claudication pain (ABI: 0.4–0.85), while 7.5% had rest pain (ABI < 0.4). NCS findings demonstrated significantly reduced motor and sensory NCV in advanced PVD stages, with mean values of 40.92 m/s (motor) and 44.88 m/s (sensory). Furthermore, CMAP and SNAP amplitudes were markedly decreased in patients with severe PVD, suggesting ischemic nerve damage.
Conclusion: This study highlights a strong correlation between PVD severity and nerve conduction abnormalities, indicating that ischemic neuropathy plays a critical role in disease progression. Sensory nerve dysfunction was more pronounced than motor involvement, aligning with findings from previous studies. Early detection of nerve conduction deficits in PVD patients may facilitate timely interventions to prevent further neurological complications. Incorporating routine NCS assessments alongside vascular evaluations could improve diagnostic accuracy and patient outcomes in PVD management. Further studies with larger cohorts are recommended to validate these findings and explore potential therapeutic strategies.

Keywords: Peripheral Vascular Disease, Nerve Conduction Study, Ischemic Neuropathy, Ankle-Brachial Index, Electrophysiology, Fountaine Staging, Peripheral Neuropathy, Chronic Ischemia.

INTRODUCTION

Peripheral Vascular Disease (PVD) is a common circulatory disorder that results from the narrowing or obstruction of blood vessels outside the heart and brain, primarily affecting the arteries that supply the limbs. It is most frequently caused by atherosclerosis, a condition characterized by the buildup of plaque within arterial walls, leading to reduced blood flow and tissue ischemia. Other contributing risk factors include diabetes mellitus, hypertension, hyperlipidemia, smoking, and sedentary lifestyles [1]. As PVD progresses, patients may experience intermittent claudication, ischemic pain, non-healing ulcers, and in severe cases, gangrene. The disease significantly impacts mobility, quality of life, and

overall prognosis, particularly in patients with coexisting metabolic disorders.

While the vascular component of PVD is well understood, its impact on the peripheral nervous system remains an area of growing research interest. Chronic ischemia due to reduced arterial perfusion can lead to nerve dysfunction, manifesting as symptoms such as pain, numbness, tingling, and muscle weakness [2]. However, a major diagnostic challenge lies in differentiating whether these neurological symptoms are a direct consequence of ischemic nerve damage or if they stem from concurrent conditions such as diabetic neuropathy. The overlap between vascular insufficiency and nerve dysfunction necessitates objective evaluation methods to accurately assess nerve involvement in PVD patients [3].

Nerve conduction studies (NCS) serve as a valuable electrophysiological tool for assessing the functional status of peripheral nerves. These studies measure parameters such as nerve conduction velocity, amplitude, and latency, providing critical insights into the presence and extent of neuropathic changes [4]. In patients with PVD, NCS can help identify nerve conduction abnormalities, determine the relationship between vascular insufficiency and nerve damage, and assess whether these changes correlate with the severity of PVD. Furthermore, early detection of nerve dysfunction in PVD patients can aid in appropriate treatment planning, ensuring that both vascular and neurological aspects of the disease are addressed effectively [5].

The severity of PVD can be graded using various clinical and diagnostic tools, including the ankle-brachial index (ABI), Doppler ultrasound, and angiographic imaging. Correlating these findings with NCS results may help establish a clearer understanding of how nerve conduction abnormalities progress alongside vascular impairment. By systematically analyzing nerve conduction parameters in patients with different grades of PVD severity, this study aims to provide valuable insights into the interplay between ischemia and nerve function. The findings from this research may contribute to improved diagnostic accuracy, better risk stratification, and more targeted therapeutic interventions for individuals suffering from PVD [6].

This study seeks to bridge the existing gap in knowledge regarding the electrophysiological impact of PVD on peripheral nerves. Through nerve conduction studies, it will explore the extent to which ischemic changes influence nerve function, ultimately leading to improved patient management strategies. A comprehensive understanding of these mechanisms will not only enhance diagnostic precision but also pave the way for multidisciplinary approaches in the treatment of PVD, ensuring optimal patient care and better long-term outcomes.

MATERIALS AND METHODS

This study was conducted over a period of two years, from May 2011 to May 2013, at Krishna Hospital, Karad. A total of 40 patients diagnosed with Peripheral Vascular Disease (PVD) were included in this study. These patients were selected from the Surgery Department and underwent nerve conduction studies to assess peripheral nerve function. The data were recorded at the time of admission according to a standardized proforma, which included demographic details, medical history, clinical examination, investigations, and nerve conduction study parameters.

Study Population

The study consisted of 40 patients who presented with symptoms suggestive of PVD. Each patient underwent a detailed history-taking and clinical examination, including an assessment of risk factors, symptoms, and grading of disease severity.

Inclusion Criteria

1. Patients diagnosed with chronic Peripheral Vascular Disease based on clinical and investigative findings.

Exclusion Criteria

1. Diabetic patients were excluded from the study to eliminate confounding factors related to diabetic neuropathy.

Classification and Staging of Peripheral Vascular Disease

Patients were classified based on the **Fountaine Staging System**, which is widely used to assess the severity of PVD. The classification is as follows:

- **Stage I**: Asymptomatic
- **Stage IIa**: Mild claudication
- **Stage IIb**: Moderate to severe claudication
- **Stage III**: Ischemic rest pain
- **Stage IV**: Ulceration or gangrene

The **Ankle-Brachial Index (ABI)** was used as an objective measure to assess the severity

of arterial insufficiency in the lower extremities. The ABI was determined by measuring systolic blood pressure at the ankle and comparing it to the brachial systolic pressure.

Ankle-Brachial Index Interpretation

- **Normal**: ABI > 1
- **Mild symptoms**: ABI = 0.90 – 1
- **Claudication pain**: ABI = 0.4 – 0.85
- **Rest pain**: ABI < 0.4

Clinical Assessment

A detailed history was obtained from all patients, focusing on symptoms such as pain, numbness, claudication, rest pain, and ulcer formation. A physical examination was conducted to assess signs of trophic changes, pulses in the lower limbs, ulcer characteristics, and sensory-motor deficits.

History Taking and Examination

Each patient was evaluated using a structured proforma that included:

1. **Demographic Details**: Name, age, gender, IPD/OPD number, address, date of admission, and date of discharge.
2. **Chief Complaints and Present History**: Presence of symptoms like pain, swelling, numbness, discoloration, intermittent claudication, and wound discharge.
3. **Family and Personal History**: History of diabetes mellitus, dietary habits, sleep patterns, bowel and bladder habits, addiction history (smoking, tobacco, etc.), and type of footwear used.
4. **General Examination**: Temperature, pulse rate, blood pressure, and presence of pallor, icterus, clubbing, cyanosis, lymphadenopathy, or edema.
5. **Local Examination**: Assessment of trophic changes, skin color, hair condition, nail integrity, muscle wasting, sweating patterns, and presence of ulcers, gangrene, foot deformities, or sensory deficits. Pulsations were palpated in the dorsalis pedis, anterior tibial, posterior tibial, femoral, and popliteal arteries.
6. **Systemic Examination**: Evaluation of the cardiovascular system (CVS), respiratory system (RS), central nervous system (CNS), and per abdomen (P/A).

Investigations

All patients underwent a battery of laboratory and imaging investigations to assess their overall health status and confirm the diagnosis of PVD. These included:

Blood Investigations

- Complete Blood Count (CBC): Hemoglobin (Hb), Total Leukocyte Count (TLC), Differential Leukocyte Count (DLC)
- Glycemic Parameters: HbA1C (to exclude diabetics), Blood Sugar Levels (Fasting and Postprandial)
- Renal Function Tests: Blood Urea Level (BUL), Serum Creatinine
- Serum Electrolytes: Sodium (Na+), Potassium (K+)
- Coagulation Profile: Bleeding Time (BT), Clotting Time (CT)
- Urine Analysis: Presence of albumin, sugar, and ketones

Radiological and Cardiovascular Assessments

- **Electrocardiogram (ECG)** to assess cardiac status
- **Chest X-ray (PA View)** to evaluate pulmonary status
- **X-ray of Foot/Leg (AP and Lateral Views)** for detecting bony changes and soft tissue involvement

Nerve Conduction Study (NCS)

Nerve conduction studies were performed to evaluate the integrity of sensory and motor nerves in the lower extremities. The following parameters were assessed:

Motor Nerve Conduction Study

- Amplitude of Compound Motor Action Potential (CMAP) (mV)
- Latency (mS)
- Nerve Conduction Velocity (NCV) (m/s)
- F-wave latency (F mean latency)
- Difference in latencies (F-M latency)

Sensory Nerve Conduction Study

- Amplitude of Sensory Nerve Action Potential (SNAP) (μV)
- Latency (mS)
- Nerve Conduction Velocity (NCV) (m/s)

Both the tibial and peroneal motor nerves, as well as the sural and superficial peroneal sensory nerves, were tested. The results were compared with normal reference values to determine the degree of nerve impairment.

Data Collection and Statistical Analysis

All collected data were recorded systematically in the proforma. Patients were grouped based on the severity of PVD according to Fountaine Staging and Ankle-Brachial Index findings. NCS

results were analyzed to identify patterns of neuropathy and correlate them with disease severity. Statistical methods, including mean, standard deviation, and comparative analysis, were used to determine the relationship between PVD severity and nerve conduction abnormalities.

Ethical Considerations

The study was conducted in accordance with ethical guidelines, ensuring patient confidentiality and informed consent before participation. Patients were informed about the purpose of the study, and only those who provided written consent were included.

RESULTS

Demographic Profile

A total of 40 patients diagnosed with Peripheral Vascular Disease (PVD) were included in the study. The age of the patients ranged from 42 to 78 years, with an average age of 59 years. Among them, 24 were male patients, constituting 60% of the study population, while 16 were female patients, making up the remaining 40%. This distribution reflects the known higher prevalence of PVD in males. The demographic details of the study population are summarized in Table 1.

Table 1: Distribution of Patients by Age and Gender

Parameter	Value
Total Patients	40
Age Range	42 - 78 years
Mean Age	59 years
Male Patients	24 (60%)
Female Patients	16 (40%)

Fountaine Staging of Peripheral Vascular Disease

The severity of PVD in patients was classified based on the Fountaine Staging System. The majority of patients, comprising 32.5% of the study group, were in Stage III, experiencing ischemic rest pain. A significant number of patients, 25% of the total, were in Stage I, indicating an asymptomatic presentation, while 20% were in Stage IIa, showing mild claudication. In addition, 15% were in Stage IIb with moderate to severe claudication, and 7.5% were in Stage IV with ulceration or gangrene. The distribution of patients according to disease severity is presented in Table 2.

Table 2: Distribution of Patients Based on Fountaine Staging

Fountaine Stage	Number of Patients	Percentage (%)
Stage I (Asymptomatic)	10	25%
Stage IIa (Mild Claudication)	8	20%
Stage IIb (Moderate to Severe Claudication)	6	15%
Stage III (Ischemic Rest Pain)	13	32.5%
Stage IV (Ulceration/Gangrene)	3	7.5%

Ankle-Brachial Index (ABI) Findings

The Ankle-Brachial Index (ABI) was used to assess the severity of blood flow impairment in the lower extremities. Among the 40 patients, none had an ABI value greater than 1.0, indicating that all patients had some level of arterial compromise. Five patients, representing 12.5% of the study group, had ABI values between 0.90 and 1.0, which correlated with mild symptoms. The majority of patients, comprising 67.5%, had ABI values in the range of 0.4 to 0.85, indicating claudication pain. The remaining 7.5% of patients had an ABI value of less than 0.4, corresponding to rest pain, which is indicative of severe ischemia. The detailed ABI distribution is presented in Table 3.

Table 3: Distribution of Patients Based on ABI Values

ABI Range	Clinical Interpretation	Number of Patients	Percentage (%)
>1.0	Normal	0	0%
0.90 – 1.0	Mild Symptoms	5	12.5%
0.4 – 0.85	Claudication Pain	27	67.5%

<0.4	Rest Pain	3	7.5%

Nerve Conduction Study (NCS) Findings

Nerve conduction studies were performed on all patients to assess the functional status of peripheral nerves. The conduction velocity of both motor and sensory nerves was evaluated. The mean motor nerve conduction velocity (NCV) was found to be 40.92 m/s, with values ranging between 30 and 50 m/s. The mean sensory nerve conduction velocity was 44.88 m/s, with values ranging between 35 and 55 m/s. Patients with more severe PVD stages exhibited lower conduction velocities, indicating a decline in nerve function due to chronic ischemia. The nerve conduction velocities in the study population are detailed in Table 4.

Table 4: Nerve Conduction Velocities (NCV) in PVD Patients

Nerve Type	Mean NCV (m/s)	Range (m/s)
Motor Nerve	40.92	30-50
Sensory Nerve	44.88	35-55

Compound Motor Action Potential (CMAP) and Sensory Nerve Action Potential (SNAP)

The compound motor action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes were also evaluated. The mean CMAP amplitude was 5.02 mV, with values ranging between 2 and 8 mV. The mean SNAP amplitude was 3.17 μV, with a range of 1 to 6 μV. A decline in CMAP and SNAP amplitudes was noted in patients with advanced PVD, suggesting a significant degree of ischemic nerve involvement. Table 5 provides a summary of the CMAP and SNAP findings.

Table 5: CMAP and SNAP Amplitudes in PVD Patients

Parameter	Mean Value	Range
CMAP (mV)	5.02	2-8
SNAP (μV)	3.17	1-6

Correlation between Disease Severity and Nerve Conduction Abnormalities

The results of this study showed a clear correlation between the severity of PVD and nerve conduction abnormalities. Patients in Stage III and IV, who exhibited ischemic rest pain and ulceration/gangrene, had significantly lower NCV values compared to those in Stages I and II. The decline in CMAP and SNAP amplitudes in advanced stages suggests progressive nerve damage due to chronic ischemia. These findings indicate that peripheral nerve function is significantly affected in patients with severe arterial insufficiency, supporting the hypothesis that ischemic neuropathy plays a crucial role in the neurological manifestations of PVD.

DISCUSSION

Overview

Peripheral Vascular Disease (PVD) is a circulatory disorder characterized by narrowed blood vessels, leading to reduced blood flow to the limbs. This diminished perfusion can result in various complications, including peripheral neuropathy. Nerve conduction studies (NCS) are pivotal diagnostic tools in evaluating the functional status of peripheral nerves in PVD patients. The present study investigated the relationship between PVD severity and nerve conduction abnormalities, revealing that patients with advanced stages of PVD exhibited significant reductions in nerve conduction velocities and amplitudes [7]. These findings align with existing literature, underscoring the intricate interplay between vascular insufficiency and nerve dysfunction.

Comparison with Existing Literature

Nerve Conduction Abnormalities in PVD

The observed decline in motor and sensory nerve conduction velocities (NCV) in patients with severe PVD stages is consistent with prior studies. For instance, a study assessed electrophysiological parameters in patients with chronic PVD and found significant reductions in both motor and sensory NCVs compared to control subjects, suggesting that chronic ischemia adversely affects nerve function, leading to peripheral neuropathy [8].

Ankle-Brachial Index (ABI) and Nerve Function

The Ankle-Brachial Index (ABI) is a widely used non-invasive measure to assess the severity of

arterial insufficiency. In the current study, a majority of patients with ABI values indicative of claudication pain (0.4 – 0.85) or rest pain (<0.4) exhibited notable nerve conduction abnormalities. This correlation aligns with findings from a study that reported lower ABI values were associated with impaired nerve function, as evidenced by reduced peroneal and sural nerve conduction velocities [9].

Sensory vs. Motor Nerve Involvement

The study demonstrated that sensory nerve conduction was more profoundly affected in patients with advanced PVD stages. This observation is corroborated by previous research indicating that sensory fibers, due to their higher metabolic demands and longer axonal lengths, are more susceptible to ischemic damage compared to motor fibers. A study emphasized that sensory nerve dysfunction often precedes motor deficits in ischemic neuropathies, reinforcing the current findings.

Electrophysiological Assessments in PVD

Electrophysiological evaluations, including NCS, are instrumental in detecting subclinical neuropathy in PVD patients. The utility of electrodiagnostic testing in precisely locating disease processes affecting the peripheral nervous system, aiding in early diagnosis and management, has been emphasized. The present study's use of NCS to identify nerve conduction abnormalities in asymptomatic or mildly symptomatic patients underscores the importance of such assessments in comprehensive PVD management.

Clinical Implications

The strong association between PVD severity and nerve conduction abnormalities has significant clinical implications. Early detection of neuropathy in PVD patients can facilitate timely interventions, potentially mitigating disease progression and improving quality of life. Moreover, incorporating routine NCS in the evaluation of PVD patients may aid in identifying those at higher risk for neuropathic complications, enabling personalized treatment strategies [10, 11].

Limitations and Future Directions

While the study provides valuable insights, certain limitations must be acknowledged. The sample size was relatively small, and the study population was confined to a single center, potentially limiting the generalizability of the findings. Future research with larger, multi-center cohorts is warranted to validate these results. Additionally, longitudinal studies could elucidate the temporal relationship between PVD progression and nerve conduction changes, offering a deeper understanding of the pathophysiological mechanisms involved.

CONCLUSION

The present study reinforces the intricate relationship between vascular insufficiency and peripheral nerve dysfunction in PVD patients. The observed nerve conduction abnormalities, particularly in advanced disease stages, highlight the necessity for comprehensive vascular and neurological assessments in this population. These findings are in concordance with existing literature, emphasizing the critical need for early detection and intervention to prevent neuropathic complications in PVD patients.

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