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Original Research Article

Comparative Study on Effect of Dyslipidemia on Nerve conduction Variables in Normotensive and Hypertensive Individuals of North India: A Hospital based Cross sectional Study

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Abstract

Background: Hypertension and dyslipidemia commonly coexist and may contribute to peripheral nerve dysfunction through vascular and metabolic injury. This study evaluated the effect of dyslipidemia on nerve conduction parameters in normotensive and hypertensive individuals.

Methods: This observational cross-sectional comparative study was conducted in the Department of Physiology in collaboration with the Department of Medicine at tertiary care centre of north India. A total of 180 subjects aged 18–60 years were enrolled and divided into two groups: 90 normotensive dyslipidemic individuals (controls) and 90 hypertensive dyslipidemic individuals (study group). Clinical examination, anthropometric assessment, blood pressure measurement, lipid profile analysis, and bilateral nerve conduction studies were performed. Motor conduction parameters of median, ulnar, common peroneal, and posterior tibial nerves and sensory parameters of median and sural nerves were assessed using standard techniques. Statistical analysis was performed using SPSS version 20.0, with $p<0.05$ considered significant.

Results: Age, BMI, and lipid profile parameters were comparable between groups, while systolic and diastolic blood pressures were significantly higher in the hypertensive dyslipidemic group. Significant reduction in motor amplitude was observed in bilateral median and posterior tibial nerves in hypertensive dyslipidemic subjects. Motor nerve conduction velocity was significantly reduced in the left ulnar, left common peroneal, and bilateral posterior tibial nerves, while sensory conduction velocity of the right sural nerve was also significantly decreased. Motor nerves were more affected than sensory nerves.

Conclusion: Hypertension with dyslipidemia is associated with early subclinical peripheral neuropathic changes. Nerve conduction studies may serve as a useful non-invasive tool for early detection of neuropathy in such patients.

Keywords: Hypertension, Dyslipidemia, Nerve conduction study, Peripheral neuropathy, Subclinical neuropathy.

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Introduction

Hypertension is considered one of the most common chronic non-communicable diseases worldwide and is a major contributor to morbidity and mortality. Sustained elevation of arterial blood pressure significantly increases the risk of cardiovascular, cerebrovascular, and renal complications, thereby imposing a considerable public health burden.[1] The prevalence of hypertension was estimated at 26.4% in 2000 and is projected to rise to 29.2% by 2025 globally. Hypertension is defined as sustained elevation of blood pressure >140/90 mmHg and is a multifactorial disease influenced by several interacting risk factors. In clinical practice, hypertension and dyslipidaemia frequently coexist, suggesting shared pathophysiological mechanisms such as endothelial dysfunction and obesity.[2] India is currently undergoing a rapid epidemiological transition, with non-communicable diseases emerging as major health concerns. The prevalence of hypertension has increased in both urban and rural populations, including younger age groups.[3] Dyslipidaemia is defined as abnormalities in lipid metabolism characterized by

elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides, along with reduced high-density lipoprotein cholesterol (HDL-C).[4,5] It is highly prevalent in the Indian population and is closely associated with hypertension and diabetes mellitus. Lifestyle modifications, altered dietary habits, and sedentary behavior have contributed to its increasing prevalence. Dyslipidaemia is a recognized risk factor for atherosclerosis and cardiovascular disease; however, its effects are not limited to the vascular system alone. Experimental and clinical studies have demonstrated that altered lipid metabolism may also contribute to peripheral neural dysfunction, including axonal neuropathy and impaired motor nerve function.[6] Proposed mechanisms include oxidative stress, inflammatory injury, ischemia, and dysregulation of local lipid metabolism.[7]

The coexistence of hypertension and dyslipidaemia, often referred to as "lipitension," has been reported in 15–31% of individuals and markedly increases cardiovascular risk.[8,9] Both conditions exert synergistic effects on the vascular endothelium, promoting endothelial dysfunction, vascular remodeling, and accelerated atherogenesis.[8] Chronic hypertension causes microvascular alterations and increased vascular resistance, while dyslipidaemia aggravates vascular injury through lipid peroxidation and inflammation.[4,5] These changes may impair blood supply to peripheral nerves, making them susceptible to ischemic and metabolic injury. Peripheral nerves depend on an intact microvascular network, and lipids form essential structural components of neuronal membranes and myelin sheaths. Therefore, disturbances in lipid metabolism may directly influence axonal integrity and nerve conduction.[10, 11,12]

Nerve conduction studies (NCS) are widely used, non-invasive neurophysiological tests that provide objective assessment of peripheral nerve function. They measure latency, amplitude, duration, and conduction velocity, helping identify early neuropathic changes even before clinical manifestations become evident.[13,14] NCS are also useful in differentiating axonal degeneration from demyelinating neuropathies and in monitoring disease progression. Despite the high prevalence of hypertension and dyslipidaemia and their frequent coexistence, limited studies have evaluated their combined effect on peripheral nerve function, particularly in the North Indian population. Previous studies have reported variable findings due to differences in disease duration, severity, treatment status, and associated metabolic abnormalities.[15] Therefore, the present study aims to compare peripheral nerve conduction parameters in dyslipidaemic normotensive and hypertensive individuals, with emphasis on the influence of hypertension on nerve function in the presence of dyslipidaemia. Early identification of subclinical neuropathy may facilitate timely preventive and therapeutic interventions.

Materials and Method

Approval of Institutional Ethics Committee, SNMC, Agra, was taken prior to commencement of this study. This study was conducted in the Department of Physiology in collaboration with the Department of Medicine at tertiary care centre of north India.

This study was aimed to assess the effect of dyslipidemia on nerve conduction variables in Normotensive and Hypertensive Individuals.

1. Study Design: The present study is an observational Cross-Sectional comparative study.

2. Study duration: The study was conducted from December 2024 to December 2025

3. Sampling technique: Simple Random Technique was used for selection of study subjects.

4. Sample Size calculation: Formula $n = (z_{1-\alpha/2} + z_{1-\beta})^2 \frac{P_1(1-P_1) + P_2(1-P_2)}{(P_1-P_2)^2}$

Where,

- $Z_{1-\alpha/2} = 1.96$ at 95% CI
- $Z_{1-\beta} = 0.84$ 80% power
- P_1 = prevalence of dyslipidemia= 50.75% [16]
- P_2 = prevalence of hypertension with dyslipidemia = 31% [17]

Sample size using this formula was:

$$= (1.95 + 0.84)^2 \frac{(50.7 \times 49.3) + (31 \times 69)}{(50.7-31)^2}$$

$$= 7.84 \times \frac{2499.51 + 2139}{(19.7)^2}$$

$$= \frac{7.84 \times 4638.51}{388.09}$$

$$= 93.7$$

So, our sample size was 90 (approx.) in each group.

Hence total sample size = 180 (90 in each group)

5. Study group:

- Group 1(control group): n=90 Normotensive individuals with dyslipidemia of both sexes of age ≥18 to 60 years.
- Group 2(study group): n= 90 Hypertensive individuals with dyslipidemia of both sexes of age ≥18 to 60 years.

6. Source of Participants: The study subjects were selected from outpatient department (OPD) of medicine. The procedure was explained to the patients in their mother tongue and informed consent was obtained.

Inclusion Criteria: All hypertensive patients of age ≥18 to 60 years who had presented to us with altered lipid levels and all normotensive subjects with altered lipid levels fulfill the criteria and willing to participate in the study were selected.

Criteria for hypertension:

- Patients with blood pressure, systolic ≥ 140 mm of Hg and diastolic ≥ 90 mm Hg.
- New and old, both case of hypertension were included.
- All hypertensive patients were on medication and only patients on antihypertensive drugs were included in the study.

Criteria for dyslipidemia:

- Total lipid: 200-600mg/dl, it includes total cholesterol 150-200mg/dl (LDL- 60 to 130mg/dl, HDL-40 to 60mg/dl, VLDL-10 to 40mg/dl), phospholipids 60-150mg/dl and free fatty acids 5-15mg/dl.
- Any alteration of normal lipid profile was included in the study.

Exclusion criteria:

- Patients with any associated diseases like diabetes mellitus, peripheral vascular diseases, autonomic nervous disorders, Cardiac disorder with pace makers.
- Patients with some other known neurological deformity, traumatic deformity, pregnancy and those who are not willing.
- Patients with complications like, thyroid disorders, CNS disorders, patients on neurotoxic drugs, consuming alcohol.
- Coexisting secondary causes of hypercholesterolemia, hypothyroidism, chronic kidney disease, cholestatic liver disease etc.
- Patient on drugs affecting the lipid profile.

Electrophysiological investigations:

- **Sensory nerve conduction study**: Measurement of SNAP with onset latency, amplitude and conduction velocity of median and sural nerve bilaterally (Upper limb and lower limb).
- **Motor nerve conduction study:** Measurement of CMAP with onset latency, amplitude and conduction velocity of median, ulnar, common peroneal and posterior tibial nerves bilaterally (Upper limb and lower limb).

Biochemical investigations measured were:

Lipid profile:

- Total cholesterol(mg/dl)
- TG (mg/dl)
- LDL-c (mg/dl)
- HDL-c (mg/dl)
- VLDL (mg/dl)

Data collection procedures:

- A detailed history was taken regarding complaints, personal history, past history, family history and treatment history.
- Thorough general examination and systemic examination of the cardiovascular, respiratory, abdomen and central nervous system with neurological examination of limbs was also done and data were collected.

Methodology:

The proposed study was carried out on Hypertensive & normotensive patients with dyslipidemia, who attended the Medicine OPD and fulfilled the inclusion criteria. Patients were briefed regarding the methodology and purpose of the study and informed and written consent was taken. The subjects were explained in brief about the experimental procedure.

Experiment was done in a quiet room, with the subject in supine position, awake and breathing normally. For examination, the subjects were advised to have their meal by 10.00 p.m. on the previous night, to remain free from any physical or mental stress, not to take any sedatives or any drug affecting central nervous system and to have a good sleep at night before the day of examination.

Since, the electrodes were placed over the skin therefore, all the subjects were advised to take a soap bath before coming for examination and were asked to avoid any oil or lotion over skin. The subjects were asked to have a light breakfast and avoid caffeine and attend the research laboratory in the department of physiology at 10:00 am on the day of examination. Patient underwent complete neurological examination.

Recording of NCV:

Recording system: Nerve conduction studies (NCS) are frequently performed to evaluate peripheral nerve disease. We recorded NCV in Median (Motor, Sensory), Ulnar (Motor) and Common Peroneal nerve (Motor), Posterior Tibial nerve (Motor), Sural nerve (Sensory) and F- wave for our studies.

Electrodes:

1. Active
2. Reference
3. Ground

The action potential is measured between active and reference electrodes and the ground electrode serves as a zero-voltage reference point.

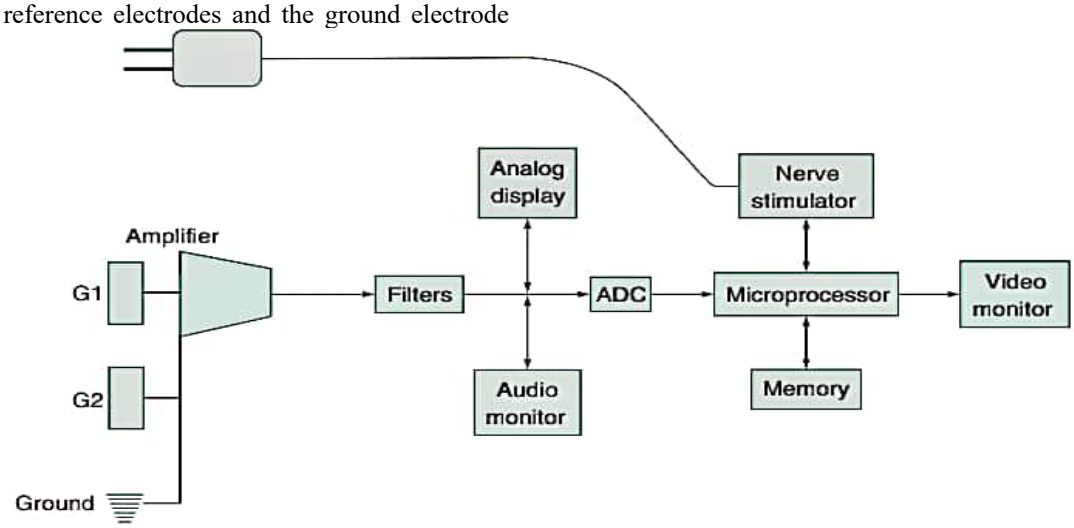

Figure 1: Diagram showing major components of electro diagnostic equipment.

The study was carried out at an ambient room temperature of 25 ◦C. This study was conducted using Neurostim NS-2 by Medicaid Instruments. The procedure was explained to the subjects.

All studies were performed in both upper and lower limbs of each subject comfortably lying-in supine position. Standardized techniques were used to obtain and record action potentials.

Upper limb:

MNCV of median nerve: For motor studies, the median nerve was tested using orthorhombic stimulation.

The active electrodes were positioned at the motor point of the abductor pollicis brevis, which was found halfway between the thumb's metacarpophalangeal joint and the midpoint of the distal wrist crease. The reference electrode was placed 4 cm distal to the thumb. Stimulation of the distal median motor nerve was conducted 8 cm proximal to the active electrode and medial to the flexor carpi radialis tendon, with the distance measured as the shortest path from the active electrode to the midpoint of the distal wrist crease. The stimulations were applied medial and posterior to the flexor carpi ulnaris tendon. The same stimulation points were used for testing both the sensory and motor fibers of the median nerve. [17]

Conduction velocity= $\frac{D}{PL-DL}$m/s

Where, PL is the proximal latency (ms), DL is the distal latency (ms), and D is the distance between proximal and distal stimulation (mm).

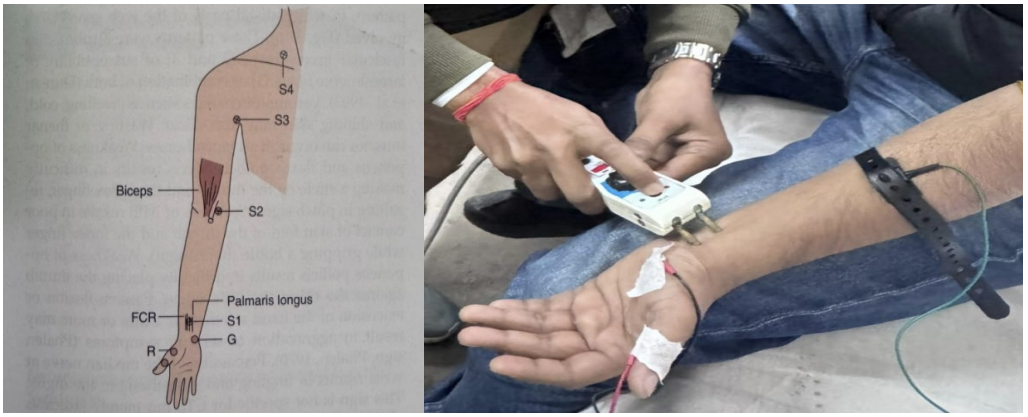

Figure 2: Electrode placement for median motor nerve conduction. S(stimulating), R (recording), G (ground electrodes), FRC (Flexor carpi radialis.

SNCV of median nerve:

For sensory studies, the Median nerve was examined antidromically with the active ring electrode was placed over the index finger at the interphalangeal joint to record responses along the Median nerve. The reference ring electrode was placed 4cm distal to the active electrode. Median nerve stimulations were performed at the wrist joint.

The normal values taken were 56.2±5.8.

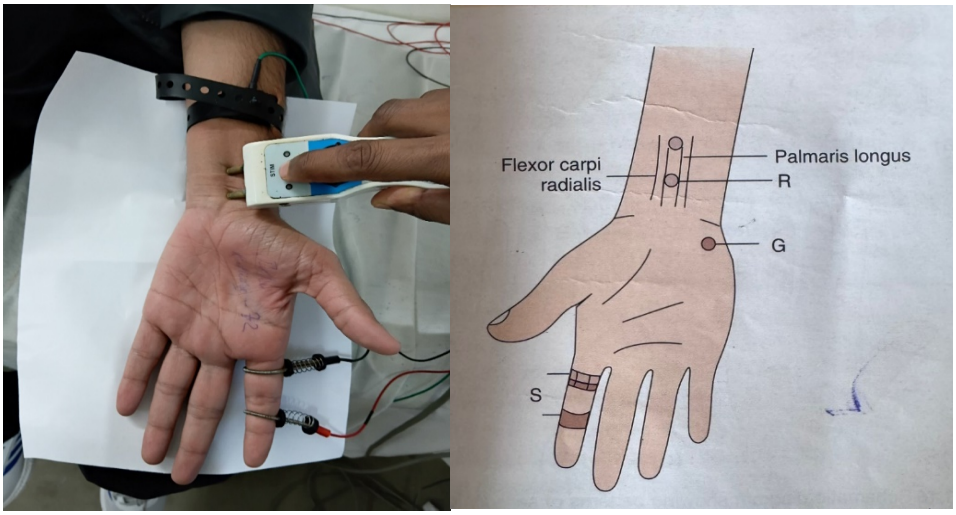

Figure 3: Electrode placement for orthodromic sensory conduction of the Median nerve.

The recording electrode was placed at Abductor digiti minimi and reference electrode was placed 4 cm proximal to the recording electrode, proximal stimulation was given at 3cm proximal to the distal crease at the wrist. Distal stimulation was given by asking the subject to the flex the forearm and stimulation is given at medial epicondyle.

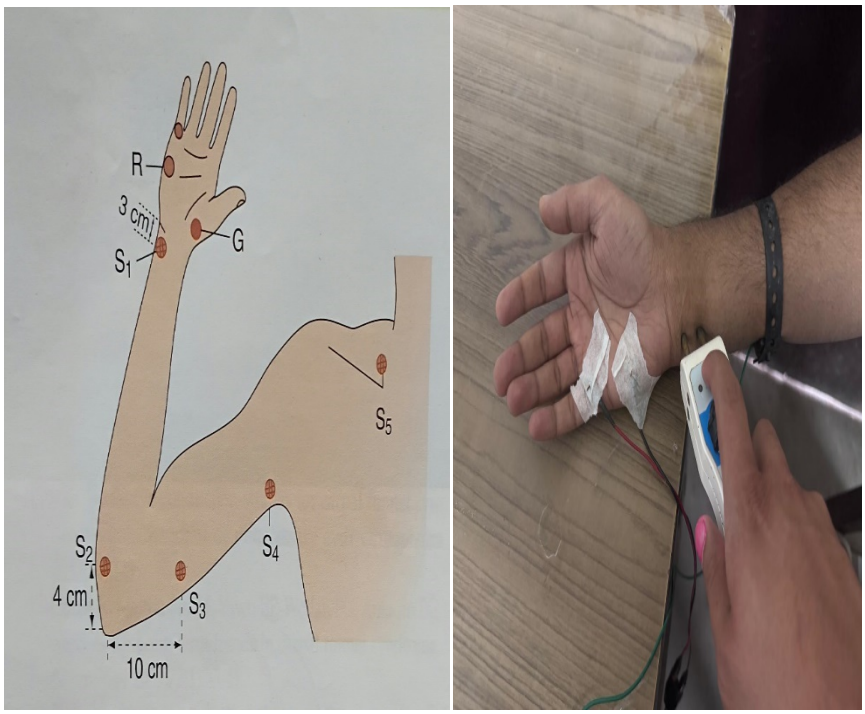

Figure 4: Electrode placement for ulnar motor nerve conduction.

Lower limb:

MNCV of common peroneal nerve: Surface recordings of motor Common peroneal nerve was obtained by placing recording electrodes at Extensor Digitorum Brevis and reference electrode was placed 4cm distal to the recording electrode and first stimulation (S1) was given at the ankle (8 cm proximal to the active recording electrode placed on the Extensor digitorum brevis), and second stimulation was given 2cm. distal to fibular neck for the Peroneal nerve conduction study. Latency and amplitude of MAPs was measured and nerve conduction variables are calculated.

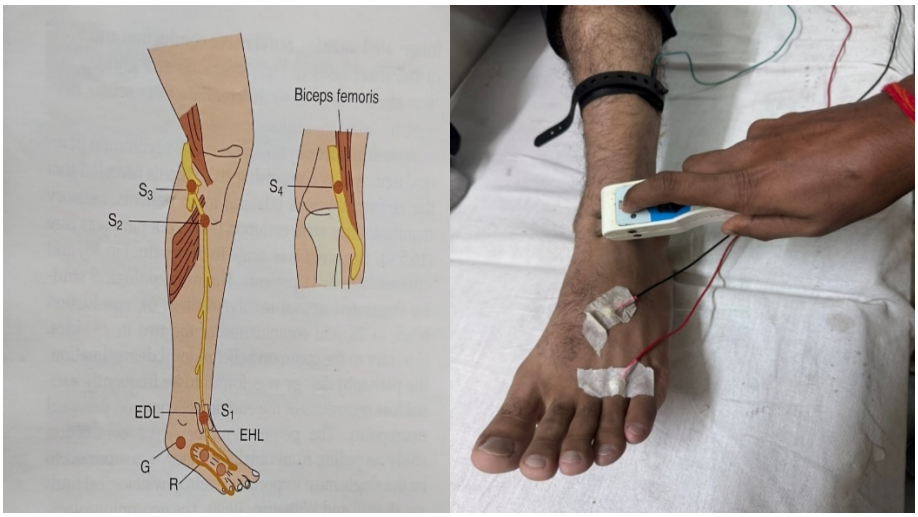

Figure 5: Electrode placement for common peroneal nerve conduction.EHL (extensor hallucis longus), EDL (extensor digitorum longus).

MNCV of posterior tibial nerve: Tibial nerve is the continuation of the median trunk of the sciatic nerve below the popliteal fossa. In the leg, it supplies the Tibialis posterior muscles. At the ankle, the nerve passes under the flexor retinaculum (tarsal tunnel) and divides into the medial and lateral planter nerves after giving a calcaneal branch. The planter nerves pass deep to the abductor hallucis muscle and supply the intrinsic foot muscle.

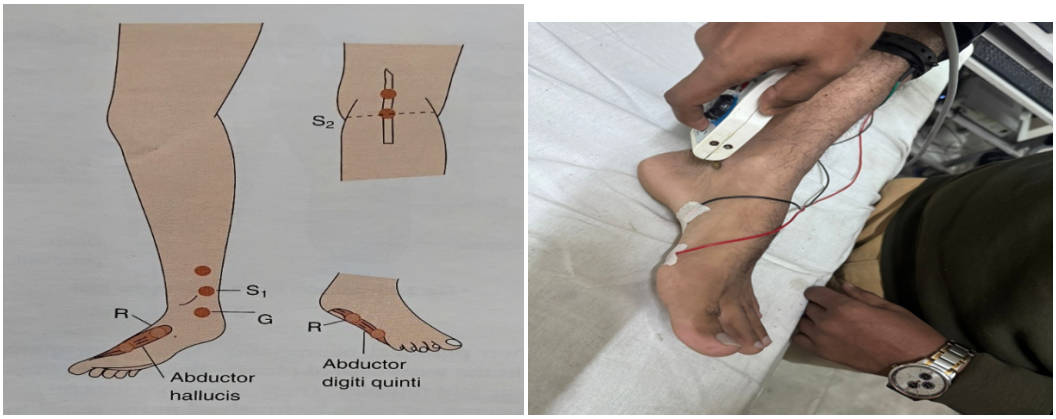

Figure 6: Electrode placement for Posterior tibial nerve.

SNCV of Sural nerve: The surface electrode between lateral malleolus and Tendo Achilles records nerve conduction of the Sural nerve. The nerve is stimulated antidromically 10-16 cm proximal to the recording electrode, distal to the lower border of gastrocnemius at the junction of the middle and lower thirds of the leg. During the recording, the leg should be relaxed and lateral position is convenient.

Sural nerve conduction velocity in healthy volunteers (n = 30) is 50.9± 5.4 m/s and amplitude of SNAP is 18.0 ±10.5 uV. [18]

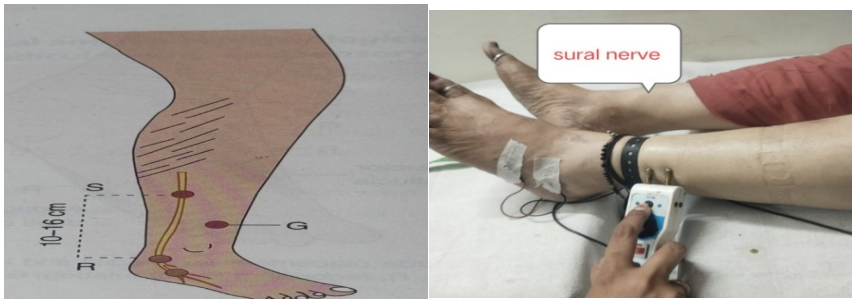

Figure 7: Electrode placement for antidromic sural nerve.

All data was entered into MS excel spreadsheets and analysed with the help of SPSS version 20.0. All values were expressed as Mean ± SD. Differences between the study group (hypertensive dyslipidemia) and control group were examined using the unpaired t test.

Main Pearson's correlation were determined between the dependent and independent variables. A two tailed test (P<0.05)* was considered statistically significant, (P<0.01)* was considered strongly significant, while (P<0.001)** was considered as highly significant.

Results

The mean age in the control group was 46.76 ± 9.76 years, whereas in the study group it was 48.13 ± 9.22 years and the difference in distribution of age in both groups was found to be statistically non-significant (P>0.05).

Among control group, males were 52 (57.8%) and remaining 38 (42.2%) were females. Among study group, males were 58 (64.4%) and remaining 32 (35.6%) were Females.

The total of 110 (61.1%) males and 70 (38.9%) females and difference in distribution of gender in both groups was found to be was statistically non-significant (P>0.05).

Table 1: Comparison of characteristics of study participants of both groups

Variable	Control Group (n=90) (Mean ± SD)	Hypertensive Group (n=90) (Mean ± SD)	P value
Age (Years)	46.76 ± **9.76**	48.13 ± 9.22	0.33
Gender Male	52 (57.8%)	58 (64.4%)	0.358
Female	38 (42.2%)	32 (35.6%)	
Anthropometry Height (cm)	160.53 ± 8.83	164.38 ± 6.99	0.001*
Weight (kg)	65.62 ± 9.02	71.33 ± 9.95	<0.001*
BMI ($Kg/m^{2)}$	25.91 ± 2.23	26.31 ± 2.53	0.26
Blood Pressure – SBP (mmHg)	110.41 ± 5.87	150.26 ± 15.19	<0.001*
DBP (mmHg)	72.59 ± 4.64	91.02 ± 4.36	<0.001*
Lipid Profile	269.80 ± 20.44	271.94 ± 17.38	0.412
Total Cholesterol	178.48 ± 11.85	180.70 ± 12.37	0.214
Low-Density Lipoprotein Cholesterol	29.73 ± 6.08	30.07 ± 5.62	0.701
High-Density Lipoprotein Cholesterol	255.63 ± 27.99	253.43 ± 28.18	0.589
Triglycerides Very low -Density Lipoprotein Cholesterol	55.23 ± 9.58	53.77 ± 9.40	0.294

Mean systolic blood pressure of control group was 110.41 ± 5.87 mmHg and in the study group it was 150.26 ± 15.19 mmHg, while the mean diastolic blood pressure was 72.59 ± 4.64 mmHg in the control group and 91.02 ± 4.36 mmHg in the study group and both differences were statistically highly significant (P <0.001). Mean total cholesterol in the control group was 269.80 ± 20.44 mg/dL and 271.94 ± 17.38 mg/dL in the study group, LDL-C in the control group was 178.48 ± 11.85 mg/dL and 180.70 ± 12.37 mg/dL in the study group, HDL-C in the control group was 29.73 ± 6.08 mg/dL and 30.07 ± 5.62 mg/dL in the study group, triglycerides in the control group were 255.63 ± 27.99 mg/dL and 253.43 ± 28.18 mg/dL in the study group and VLDL in the control group was 55.23 ± 9.58 mg/dL and 53.77 ± 9.40 mg/dL in the study group, showing no statistically significant differences between the groups (P>0.05). (Table 1)

Motor nerve amplitude was significantly reduced in the study group compared to controls for bilateral median nerves and bilateral posterior tibial nerves (p<0.05), indicating possible motor nerve involvement in these nerves.

However, no significant difference was observed in the bilateral common peroneal nerves between the two groups (p>0.05). (Table 2)

Table 2: Comparison of Motor Nerve Amplitude (mV) Between both Groups

Variable	Control (Mean ± SD)	Study (Mean ± SD)	p-value
Motor Left Median	9.37 ± 2.50	8.03 ± 2.10	<0.001*
Motor Right Median	9.78 ± 2.34	8.65 ± 2.11	0.001*
Motor Left CPN	4.85 ± 0.96	4.97 ± 0.73	0.342
Motor Right CPN	4.83 ± 1.10	4.82 ± 0.99	0.942
Motor Left PTN	5.06 ± 0.85	4.78 ± 0.84	0.018*
Motor Right PTN	5.18 ± 0.84	4.87 ± 0.96	0.012*

[+]CPN- Common Peroneal Nerve, [#]PTN – Posterior Tibial Nerve

Table 3: Comparison of Motor Nerve Conduction Velocity (MNCV) (m/s) Between both Groups

Variable	Control	Study	p-value
MNCV Left Median	51.41 ± 9.33	52.74 ± 10.79	0.362
MNCV Right Median	57.80 ± 7.30	59.71 ± 46.44	0.721
MNCV Left Ulnar	56.72 ± 10.81	53.40 ± 9.03	0.028*
MNCV Right Ulnar	58.87 ± 12.02	59.41 ± 8.39	0.742
MNCV Left CPN	51.63 ± 6.58	49.00 ± 8.52	0.021*
MNCV Right CPN	50.43 ± 6.80	50.60 ± 5.44	0.861
MNCV Left PTN	49.46 ± 7.67	43.37 ± 6.71	<0.001*
MNCV Right PTN	42.12 ± 2.69	41.20 ± 3.14	0.041*

[+]CPN- Common Peroneal Nerve, [#]PTN – Posterior Tibial Nerve

Motor nerve conduction velocity (MNCV) was significantly reduced in the study group for the left ulnar, left common peroneal, and bilateral posterior tibial nerves ($p<0.05$), suggesting impaired motor nerve conduction in these nerves. However, no significant differences were observed in bilateral median nerves, right ulnar nerve, and right common peroneal nerve between the two groups ($p>0.05$). (Table 3)

Table 4: Comparison of Sensory Nerve Amplitude (µV) between both Groups

Variable	Control (Mean ± SD)	Study (Mean ± SD)	p-value
Sensory Left Median	19.84 ± 94.94	9.67 ± 1.93	0.421
Sensory Right Median	9.54 ± 1.42	9.94 ± 1.71	0.084
Sensory Left Sural	21.53 ± 2.95	21.13 ± 3.38	0.412
Sensory Right Sural	21.62 ± 2.61	21.82 ± 3.60	0.684

Sensory nerve amplitude showed no statistically significant difference between the control and study groups for bilateral median and sural nerves ($p>0.05$). These findings suggest relative preservation of sensory nerve amplitude in the study population. (Table 4.)

Table 5. Comparison of Sensory Nerve Conduction Velocity (m/s) between both groups

Variable	Control (Mean ± SD)	Study (Mean ± SD)	p-value
Left Median	54.42 ± 48.39	45.50 ± 7.30	0.092
Right Median	47.42 ± 6.90	50.90 ± 42.14	0.486
Left Sural	53.90 ± 48.30	47.40 ± 7.37	0.214
Right Sural	50.02 ± 5.56	47.96 ± 7.03	0.033*

Sensory nerve conduction velocity showed a statistically significant reduction only in the right sural nerve of the study group compared to controls ($p=0.033$). No significant differences were observed in bilateral median nerves and left sural nerve conduction velocities between the two groups ($p>0.05$).(Table 5)

Table 6. Correlation of Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) with Motor and Sensory Nerve Conduction Parameters

Variable	SBP (rho)	DBP (rho)
Motor Left Median	-0.177*	-0.190*
Motor Right Median	-0.186*	-0.166*
SNCV Left Median	-0.240*	-0.276*

Systolic and diastolic blood pressure showed a significant negative correlation with motor median nerve amplitudes and left median sensory nerve conduction velocity ($p<0.05$). These findings indicate that increasing blood pressure is associated with worsening motor and sensory nerve conduction parameters.(Table 6)

Table 7. Correlation of Serum Lipid Parameters (Total Cholesterol, LDL-C, HDL-C, Triglycerides) with Motor Nerve Amplitude Parameters

Variable	Total Cholesterol	LDL-C	HDL-C	Triglycerides
Motor Left Median	-0.131	-0.028	-0.038	0.025
Motor Right Median	-0.104	-0.051	0.028	-0.012

No significant correlation was observed between serum lipid parameters (total cholesterol, LDL-C, HDL-C, and triglycerides) and motor median nerve amplitude parameters. These findings suggest that lipid levels were not significantly associated with motor nerve amplitude changes in the study population. (Table 7)

Discussion

Hypertension is a chronic medical condition characterized by persistent elevation of systemic

arterial blood pressure, leading to cardiovascular and microvascular damage. Dyslipidemia, marked by elevated total cholesterol, LDL cholesterol, triglycerides and reduced HDL cholesterol, contributes to endothelial dysfunction, oxidative stress and inflammation.

Both conditions are important risk factors for peripheral neuropathy through vascular and metabolic mechanisms. Chronic hypertension causes endothelial dysfunction, increased vascular resistance and structural remodeling of the vasa nervorum, resulting in reduced blood supply to peripheral nerves and chronic ischemic injury. However, hypertension alone may not significantly alter nerve conduction, suggesting that associated metabolic factors such as dyslipidemia may play a major role in neuropathic changes.[19] Previous studies on nerve conduction in hypertensive dyslipidemic individuals have reported conflicting findings, and the relationship remains insufficiently explored.

The present study was conducted to evaluate and compare nerve conduction parameters in dyslipidemic normotensive and hypertensive individuals. A total of 180 participants were included. The study group showed significantly higher systolic and diastolic blood pressure compared to controls ($p<0.001$), consistent with findings reported by previous studies as well.[9,15,19] Lipid parameters including total cholesterol, LDL, HDL, triglycerides and VLDL showed no significant intergroup differences, indicating that dyslipidemia was prevalent in both groups, similar to observations by previous studies well. [2,4,5]

Anthropometric analysis showed comparable age distribution between groups, while body weight was significantly higher in hypertensive individuals. BMI showed no significant intergroup difference, although strong negative correlations were observed between BMI and nerve conduction parameters, supporting findings by Deshmane et al. and Naik et al. [20,21] Male predominance was observed in both groups, in agreement with previous studies. [1,3,22]

Motor nerve conduction studies revealed significantly reduced CMAP amplitudes in bilateral median nerves and posterior tibial nerves in hypertensive dyslipidemic individuals, indicating early motor axonal involvement. Similar findings were reported by previous studies as well. [6,11,] Significant reductions in motor nerve conduction velocity were also observed in ulnar, common peroneal and posterior tibial nerves, particularly in lower limbs, suggesting demyelination and impaired nerve conduction. [14,20] Sensory nerve studies showed minimal amplitude changes, although sural SNCV demonstrated significant slowing, indicating early sensory involvement.

Negative correlations were observed between SBP, DBP and nerve conduction parameters, suggesting progressive neural impairment with increasing blood pressure.[15,16,19] Lipid parameters showed weak correlations with nerve amplitudes, indicating indirect metabolic effects on neuropathy. [4,5] Overall, the findings suggest that hypertension and dyslipidemia together contribute to early peripheral nerve dysfunction, particularly affecting lower limb motor nerves, highlighting the importance of early detection and metabolic control to prevent neuropathic progression.

Conclusion

This study evaluated the effect of dyslipidaemia on nerve conduction variables in normotensive and hypertensive individuals of North India. Dyslipidaemia was associated with reduced nerve conduction velocity, prolonged latency, and decreased amplitude, indicating impaired peripheral nerve function and early subclinical neuropathic changes.

These abnormalities were more pronounced in hypertensive individuals, suggesting a combined detrimental effect of hypertension and dyslipidaemia on peripheral nerves. Nerve conduction studies are useful for early detection of subclinical neuropathy and should be routinely considered in hypertensive patients. Early screening and management of modifiable risk factors such as dyslipidaemia, hypertension, obesity, and smoking are essential to reduce neuropathy-related morbidity.

Limitations:

The cross-sectional design limited causal interpretation between hypertension, dyslipidaemia, and nerve conduction abnormalities. Only selected motor and sensory nerves were assessed, while advanced electrophysiological techniques such as F-wave, H-reflex, and small fiber neuropathy evaluation were not included, possibly missing subtle dysfunction. Additionally, factors like duration and severity of hypertension and dyslipidaemia, along with the effects of medications, were not analyzed separately, which may have influenced the findings.

References

1. Zhou B, Perel P, Mensah GA, Ezzati M. Global epidemiology, health burden and effective interventions for elevated blood pressure and hypertension. Nature Reviews Cardiology. 2021 Nov;18(11):785-802.
2. Chen S, Cheng W. Relationship between lipid profiles and hypertension: a cross-sectional

study of 62,957 Chinese adult males. Frontiers in public health. 2022 May 18;10:895499.
3. Gupta R, Xavier D. Hypertension: The most important non-communicable disease risk factor in India. Indian heart journal. 2018 Jul 1;70(4):565-72.
4. Goldberg IJ, Eckel RH, McPherson R. Triglycerides and heart disease: still a hypothesis? Arteriosclerosis, thrombosis, and vascular biology. 2011 Aug;31(8):1716-25.
5. Nelson RH. Hyperlipidemia as a risk factor for cardiovascular disease. Primary care. 2012 Dec 4;40(1):195.
6. Iqbal Z, Bashir B, Ferdousi M, Kalteniece A, Alam U, Malik RA, Soran H. Lipids and peripheral neuropathy. Current opinion in lipidology. 2021 Aug 1;32(4):249-57.
7. Wu S, Cao X, He R, Xiong K. Detrimental impact of hyperlipidemia on the peripheral nervous system: A novel target of medical epidemiological and fundamental research study:/1. Neural regeneration research. 2012 Feb 15;7(5):392-9.
8. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith Jr SC, Spertus JA. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005 Oct 25;112(17):2735-52.
9. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nature reviews nephrology. 2020 Apr;16(4):223-37.
10. Virdis A, Ghiadoni L, Giannarelli C, Taddei S. Endothelial dysfunction and vascular disease in later life. Maturitas. 2010 Sep 1;67(1):20-4.
11. Vincent AM, Callaghan BC, Smith AL, Feldman EL. Diabetic neuropathy: cellular mechanisms as therapeutic targets. Nature Reviews Neurology. 2011 Oct;7(10):573-83.
12. Wiggin TD, Sullivan KA, Pop-Busui R, Amato A, Sima AA, Feldman EL. Elevated triglycerides correlate with progression of diabetic neuropathy. Diabetes. 2009 Jul 1;58(7):1634-40.
13. Preston DC, Shapiro BE. Electromyography and Neuromuscular Disorders: Clinical-Electrophysiologic Correlations. 3rd ed. Philadelphia: Elsevier Saunders; 2013.
14. Kimura J, Strakowski JA. Electrodiagnosis in diseases of nerve and muscle: principles and practice. Oxford university press; 2025 Feb 26.
15. Gupta V, Gupta R, Gaur K, et al. Nerve conduction changes in patients with essential hypertension. J Assoc Physicians India. 2015;63(3):30–34.
16. Taddei S, Virdis A. Endothelial dysfunction in hypertension. J Clin Hypertens (Greenwich). 2021;23(5):1001–1008.
17. Yuan Y, Chen W, Luo L, Xu C. Dyslipidemia: causes, symptoms and treatment. Int J Trend Sci Res Dev. 2021;5(2):1013-1016.
18. Elsaied H, Sedeek M, Fehr A, Fadeil MR. The Relationship between Dyslipidemia, Antioxidants and Nerve Conduction Velocity in Overweight and Obese Persons in Aswan. IJCEP. 2018;5(3):136-40.
19. Misra UK, Kalita J, Yadav RK, Peripheral neuropathy in RA. J Assoc Physicians India. 2005; 53:39-78.
20. Lloyds A, Subhash S, Swornila DL, Justus AM. Effect of hypertension on motor nerve conduction variables. J Evid Based Med Healthc. 2016;3(75):4091–4093.
21. Deshmane S, Khan ST. Comparative study of nerve conduction velocity in normal and obese individuals. IOSR J Dent Med Sci. 2016;15(3):54-57.
22. Naik BM, Pal P, Pal GK, Balakumar B, Dutta TK. Assessment of motor nerve conduction in healthy obese Indian population. Int J Clin Exp Physiol. 2014;1(4):277-282
23. Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW, MacLaughlin EJ. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2018 May 15;71(19):e127-248.