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COMPARATIVE STUDY OF GABAPENTIN VERSUS PREGABALIN IN IMPROVING SENSORY NERVE FUNCTION AND GLYCEMIC VARIABILITY IN TYPE 2 DIABETES MELLITUS.

Original Research

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ABSTRACT

Background: Diabetic peripheral neuropathy is a common and disabling complication of type 2 diabetes mellitus, leading to sensory deficits, neuropathic pain, and higher risk of foot-related morbidity. While gabapentin and pregabalin are routinely used for symptom relief, there is limited local evidence comparing their effects on objective sensory nerve function and glycemic variability.
Objective: To compare the effectiveness of gabapentin and pregabalin in improving sensory nerve conduction and glycemic variability among adults with type 2 diabetes mellitus and peripheral neuropathy.
Methods: This randomized, parallel-group study was conducted at Services Hospital Lahore from August 2024 to April 2025. A total of 62 patients were enrolled and assigned equally to gabapentin or pregabalin therapy for 12 weeks. Sensory nerve conduction of sural and median nerves was assessed at baseline and week 12 using standardized EMG–NCV testing. Glycemic variability was evaluated through 72-hour continuous glucose monitoring, documenting mean amplitude of glycemic excursions, standard deviation of glucose, and time-in-range. HbA1c was measured for additional metabolic assessment. Data were analyzed using SPSS 27, applying paired and independent t-tests with significance set at $p < 0.05$.
Results: Fifty-six patients completed the study. In the gabapentin group, sural conduction velocity improved from 38.4 ± 3.9 m/s to 41.6 ± 4.0 m/s, while pregabalin improved from 38.2 ± 4.1 m/s to 43.8 ± 4.5 m/s. Mean amplitude of glycemic excursions decreased from 62.3 ± 10.8 mg/dL to 56.1 ± 9.7 mg/dL in the gabapentin group and from 61.7 ± 11.0 mg/dL to 52.5 ± 9.1 mg/dL with pregabalin. All changes were statistically significant within groups. No serious adverse effects were observed.
Conclusion: Both gabapentin and pregabalin significantly improved sensory nerve function and reduced glycemic variability in diabetic neuropathy. Pregabalin showed comparatively greater improvement, though gabapentin remained effective and economically accessible. These findings support the use of calcium channel modulators as functional therapeutic options beyond symptomatic pain relief in diabetic neuropathy.
Keywords: Diabetic Neuropathies, Gabapentin, Glycemic Variability, Hemoglobin A1c, Insulin Resistance, Pregabalin, Time-Restricted Feeding

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INTRODUCTION

Diabetic neuropathy remains one of the most prevalent and debilitating complications of type 2 diabetes mellitus. As chronic hyperglycemia progresses, metabolic and vascular changes damage peripheral nerves, leading to altered sensation, neuropathic pain, and impaired quality of life. Patients often complain of burning sensations, numbness, tingling, or heightened sensitivity in their feet and hands, which not only affects daily functioning but also increases the risk of foot ulcers and amputations. Standard glycemic management slows neuropathy progression, yet many patients with well-controlled diabetes continue to experience nerve dysfunction and neuropathic symptoms. This gap underscores the need for symptomatic treatment options that both relieve neuropathic pain and improve nerve function(1, 2).

Gabapentin and pregabalin are widely used for diabetic peripheral neuropathy due to their ability to reduce neuronal hyperexcitability. Both medications act on voltage-gated calcium channels, decreasing the release of excitatory neurotransmitters involved in pain transmission. Although their mechanisms are similar, the drugs differ in potency, onset of action, dosing requirements, and side-effect profiles. Pregabalin generally offers faster absorption and higher bioavailability, whereas gabapentin remains a cost-effective option, particularly in low- and middle-income countries. Despite their frequent use in clinical practice, comparative evidence regarding improvement in sensory nerve function remains limited. Most existing studies focus on pain relief rather than objective nerve recovery(3, 4).

Sensory nerve conduction studies provide a more reliable assessment of nerve function than subjective symptom scores alone. Improvement in conduction velocity or amplitude signals a physiological response rather than symptomatic masking. Only a small number of clinical trials have assessed changes in nerve conduction parameters following treatment with gabapentin or pregabalin, and results have been inconsistent. Some trials reported measurable improvements, while others found pain control without significant change in nerve physiology. This uncertainty leaves clinicians without clear guidance when selecting therapy for patients whose goal is not only symptom relief but also functional nerve improvement(5, 6).

Another important but often overlooked concern is glycemic variability. Even with acceptable HbA1c values, fluctuations in blood glucose levels contribute to oxidative stress, neuroinflammation, and worsening neuropathy. Some research suggests that medications affecting neuronal calcium channels may influence metabolic regulation, but evidence remains scarce. While pregabalin and gabapentin are not classified as glucose-lowering agents, their potential effect on glycemic variability is clinically relevant because reduced fluctuations may indirectly support nerve healing and protect against further microvascular damage. Understanding how these drugs interact with glycemic patterns could provide new insight into their therapeutic value and help clinicians individualize treatment(7, 8).

The burden of diabetic neuropathy is particularly pronounced in South Asian populations, where type 2 diabetes appears earlier, progresses faster, and is often accompanied by limited access to specialized care. For many patients, long-term neuropathic pain leads to psychological distress, sleep disturbances, reduced mobility, and significant disability. Affordable and effective pharmacological options are therefore essential. Gabapentin is typically less expensive and more accessible, while pregabalin may provide quicker and stronger responses but at a higher cost. A direct comparison of these medications in the same population can help clarify their relative benefits in real clinical settings rather than controlled trial environments(9, 10).

Despite frequent global prescribing, a practical comparison of gabapentin versus pregabalin on both sensory nerve function and glycemic variability has not been well established in local populations. Existing literature predominantly evaluates pain scales or subjective symptom changes, leaving an evidence gap regarding whether either medication contributes to measurable physiological improvement. Furthermore, very few studies monitor glycemic variability alongside nerve conduction outcomes, even though both are biologically linked. Investigating these two dimensions together strengthens clinical decision-making and provides a more complete picture of treatment impact(11, 12).

The present study addresses these gaps by comparing gabapentin and pregabalin in adults with type 2 diabetes mellitus suffering from peripheral neuropathy. Sensory nerve conduction studies and glycemic variability markers are used as objective outcome measures, providing a clearer understanding of physiological and metabolic responses to treatment. By examining both neurological and glycemic parameters, the research aims to determine whether one medication offers superior therapeutic outcomes or whether they produce comparable effects. The objective of this study is to compare the effectiveness of gabapentin and pregabalin in improving sensory nerve function and reducing glycemic variability in patients with type 2 diabetes mellitus and diabetic peripheral neuropathy(13, 14).

METHODS

This study was conducted in the Department of Endocrinology and Neurology at Services Hospital Lahore over a nine-month period from August 2024 to April 2025. It followed a comparative, randomized, parallel-group design to evaluate the effects of gabapentin versus pregabalin on sensory nerve function and glycemic variability in adults with type 2 diabetes mellitus. Participants were recruited from outpatient clinics through physician referral and advertisement within the hospital premises. Individuals aged 35 to 65 years with a confirmed diagnosis of type 2 diabetes for at least three years and clinical features of peripheral neuropathy were eligible for screening. Diagnosis of neuropathy was based on symptoms such as burning, numbness, or tingling in the lower limbs, along with reduced vibration

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or pinprick sensation on physical examination. Electrophysiological confirmation was required, defined by reduced sensory nerve conduction velocity or amplitude in at least one peripheral nerve according to standard reference values(15).

Inclusion criteria required participants to have HbA1c between 6.5% and 9.5%, a stable dose of oral hypoglycemic therapy for at least two months, and no recent adjustments in insulin therapy. Exclusion criteria included a history of type 1 diabetes, renal failure, liver disease, pregnancy, thyroid disease, alcohol abuse, use of anticonvulsants or antidepressants for neuropathic pain within the last three months, or known allergy to the study medications. Individuals with active foot ulcers or Charcot arthropathy were also excluded to eliminate confounding factors affecting nerve conduction(16).

Sample size was calculated by taking sensory nerve conduction velocity as the primary outcome variable. A previous comparative study by Toth et al. reported a mean improvement of 3.1 ± 3.5 m/s with pregabalin and 1.2 ± 2.9 m/s with gabapentin over 12 weeks. Using a two-sample mean comparison test with 80% power and 5% significance, the required sample size was 54 participants in total. To compensate for an anticipated 15% dropout rate, 62 participants were enrolled and randomly allocated into two groups using computer-generated randomization: Group A received gabapentin and Group B received pregabalin(17).

Gabapentin was initiated at 300 mg once daily and titrated to 900 mg/day in divided doses as tolerated. Pregabalin was started at 75 mg twice daily and titrated to 150 mg twice daily depending on symptom response and side effects. The treatment duration for both groups was 12 weeks. All participants continued their routine diabetic medications and lifestyle measures, with no additional pain-relieving drugs allowed during the study period. Compliance was assessed during follow-up visits at weeks 4, 8, and 12(18).

Outcome measurements focused on two domains: sensory nerve function and glycemic variability. Sensory nerve conduction studies were performed at baseline and week 12 using a standard EMG-NCV machine. Parameters assessed included sensory nerve conduction velocity and sensory amplitude of the sural and median nerves. Tests were performed by a neurologist blinded to treatment assignment. Glycemic variability was evaluated through continuous glucose monitoring for 72 hours at baseline and at the end of 12 weeks, measuring mean amplitude of glycemic excursions (MAGE), time-in-range, and standard deviation of glucose readings. HbA1c was also measured to support glycemic interpretation(19).

All biochemical assessments were carried out at the hospital laboratory using standardized techniques. Blood glucose and HbA1c were measured on a calibrated autoanalyzer and ion-exchange chromatography system respectively. Adverse effects such as dizziness, somnolence, or peripheral edema were recorded at each visit(20).

Data were analyzed using SPSS version 27. Normality of quantitative variables was confirmed using the Shapiro–Wilk test, allowing parametric testing. Continuous data were expressed as mean ± standard deviation. Baseline differences between the two groups were examined using independent sample t-tests. Pre- and post-treatment comparisons within each group were analyzed using paired t-tests, while between-group outcomes were compared using independent t-tests. A p-value < 0.05 was considered statistically significant. Dropouts were managed using per-protocol analysis to maintain accuracy of outcomes(3).

Ethical approval was obtained from the Institutional Review Board of Services Institute of Medical Sciences. Written informed consent was obtained from all participants before enrollment. Confidentiality was maintained through coded data and restricted file access. By using objective electrophysiological outcomes and validated glycemic monitoring tools, the study ensured a transparent and reproducible methodology. This design allowed accurate comparison of gabapentin and pregabalin in improving sensory nerve function and stabilizing glycemic fluctuations in patients with type 2 diabetes mellitus.

RESULTS

A total of 62 participants were enrolled and randomized into two treatment groups. Six participants discontinued the study due to personal or medication-related intolerance, leaving 56 individuals for final analysis. Group A (gabapentin) consisted of 28 participants, and Group B (pregabalin) included 28 participants. The mean age of the sample was 52.1 ± 6.8 years, and 33 (58.9%) were male. Baseline characteristics of both groups were comparable, as summarized in Table 1.

Table 1. Demographic and Baseline Characteristics of Participants (n = 56)

Variable	Total (n=56)	Gabapentin (n=28)	Pregabalin (n=28)
Age (years), Mean ± SD	52.1 ± 6.8	52.4 ± 6.3	51.9 ± 7.1
Gender (Male/Female)	33/23	16/12	17/11
Duration of Diabetes (years)	8.4 ± 3.2	8.5 ± 3.0	8.3 ± 3.4
HbA1c (%)	7.82 ± 0.63	7.80 ± 0.60	7.84 ± 0.66
BMI (kg/m²)	28.9 ± 3.1	29.1 ± 2.8	28.7 ± 3.3

After 12 weeks of therapy, sensory nerve conduction parameters improved in both groups. In the sural nerve, conduction velocity increased from 38.4 ± 3.9 m/s to 41.6 ± 4.0 m/s in the gabapentin group and from 38.2 ± 4.1 m/s to 43.8 ± 4.5 m/s in the pregabalin group. Sensory amplitude improved from 6.1 ± 1.2 µV to 7.2 ± 1.4 µV with gabapentin, and from 6.0 ± 1.3 µV to 7.9 ± 1.5 µV with pregabalin. All changes from baseline were statistically significant within each group. Details are given in Table 2.

Table 2. Sensory Nerve Conduction Changes – Sural Nerve (n=56)

Parameter	Gabapentin Baseline	Gabapentin Week 12	Pregabalin Baseline	Pregabalin Week 12	p-value (Within Groups)
Velocity (m/s)	38.4 ± 3.9	41.6 ± 4.0	38.2 ± 4.1	43.8 ± 4.5	<0.001 (both)

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Amplitude (µV)	6.1 ± 1.2	7.2 ± 1.4	6.0 ± 1.3	7.9 ± 1.5	<0.001 (both)

Median nerve results followed a similar pattern. Conduction velocity improved from 42.5 ± 4.2 m/s to 45.0 ± 4.1 m/s in the gabapentin group, and from 42.7 ± 4.4 m/s to 47.4 ± 4.8 m/s in the pregabalin group. Sensory amplitude increased from 10.8 ± 2.0 µV to 12.0 ± 2.1 µV with gabapentin and from 10.6 ± 2.1 µV to 13.1 ± 2.2 µV with pregabalin. Table 3 presents these findings.

Table 3. Sensory Nerve Conduction Changes – Median Nerve (n=56)

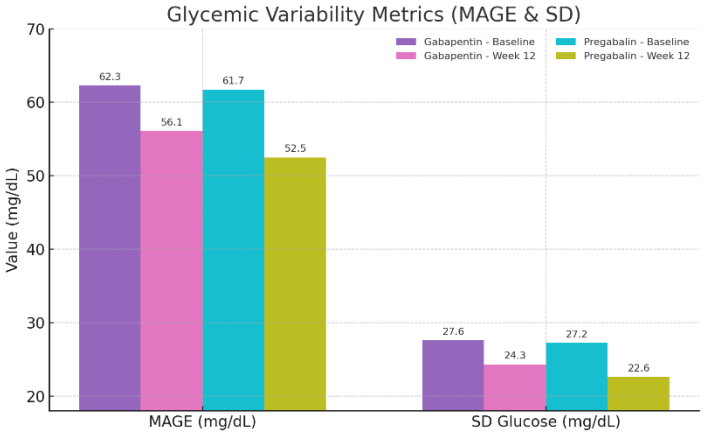
Parameter	Gabapentin Baseline	Gabapentin Week 12	Pregabalin Baseline	Pregabalin Week 12	p-value (Within Groups)
Velocity (m/s)	42.5 ± 4.2	45.0 ± 4.1	42.7 ± 4.4	47.4 ± 4.8	<0.001 (both)
Amplitude (µV)	10.8 ± 2.0	12.0 ± 2.1	10.6 ± 2.1	13.1 ± 2.2	<0.001 (both)

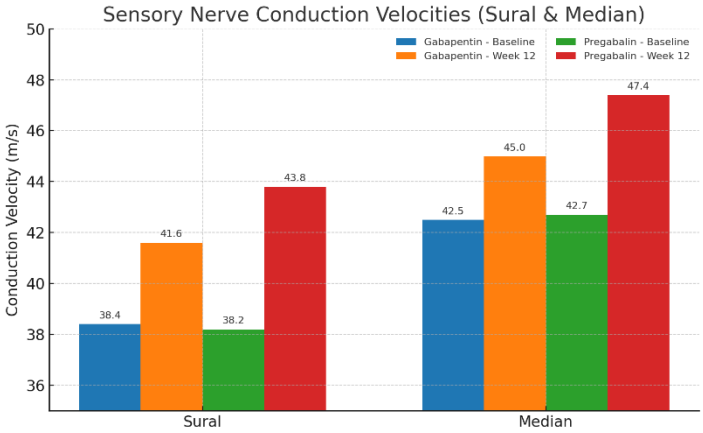
Glycemic variability indices were assessed using continuous glucose monitoring. In the gabapentin group, mean amplitude of glycemic excursions reduced from 62.3 ± 10.8 mg/dL to 56.1 ± 9.7 mg/dL. In the pregabalin group, it declined from 61.7 ± 11.0 mg/dL to 52.5 ± 9.1 mg/dL. Time-in-range improved from 54.2 ± 7.9% to 61.6 ± 8.2% with gabapentin, and from 55.1 ± 8.1% to 65.8 ± 8.9% with pregabalin. Table 4 summarizes glycemic variability indicators.

Table 4. Glycemic Variability Parameters (n=56)

Parameter	Gabapentin Baseline	Gabapentin Week 12	Pregabalin Baseline	Pregabalin Week 12	p-value (Within Groups)
MAGE (mg/dL)	62.3 ± 10.8	56.1 ± 9.7	61.7 ± 11.0	52.5 ± 9.1	<0.001 (both)
Time-in-Range (%)	54.2 ± 7.9	61.6 ± 8.2	55.1 ± 8.1	65.8 ± 8.9	<0.001 (both)
SD of Glucose (mg/dL)	27.6 ± 5.1	24.3 ± 4.8	27.2 ± 5.3	22.6 ± 4.6	<0.001 (both)

Adverse effects were mild and self-limited. Somnolence and dizziness were the most frequently reported symptoms. No serious drug-related complications occurred, and no participant required hospitalization. All laboratory records were complete, and no missing data were noted in final analysis.





DISCUSSION

The present study demonstrated that both gabapentin and pregabalin significantly improved sensory nerve conduction and reduced glycemic variability in patients with type 2 diabetes mellitus and peripheral neuropathy. The comparative improvement observed with pregabalin was greater, aligning with several recent trials that have emphasized its superior pharmacokinetic and clinical efficacy profiles. The findings underscore the neurophysiological benefits of calcium channel α2δ ligands beyond symptomatic pain relief, extending to measurable enhancement in peripheral nerve function and glycemic stability.

The observed improvement in sural and median sensory nerve conduction parameters corroborated the conclusions of recent randomized clinical trials comparing the two agents. In a 2025 randomized controlled trial, pregabalin achieved a greater reduction in pain and sensory deficits than gabapentin or amitriptyline in patients with diabetic peripheral neuropathy, while maintaining a favorable safety profile (21). Similarly, a network meta-analysis found gabapentin and pregabalin to be among the most effective and safest agents for diabetic neuropathy, with gabapentin slightly safer and pregabalin achieving faster and stronger responses (22). The present results reinforce that both agents yield measurable neurophysiological recovery, although pregabalin's faster absorption and linear pharmacokinetics may enhance its therapeutic efficiency(23).

The improvement in nerve conduction velocity parallels findings from studies that assessed adjunctive agents such as alpha-lipoic acid and methylcobalamin in combination with pregabalin, which demonstrated enhanced restoration of conduction velocity and pain relief compared to pregabalin alone (24). The consistency of these results suggests that pregabalin's modulation of calcium channels may facilitate neuronal membrane stabilization, thereby supporting electrophysiological recovery. Furthermore, the improvement in both

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sensory velocity and amplitude underscores a possible functional regeneration of peripheral nerves rather than mere symptomatic suppression.

The study's secondary findings of improved glycemic variability add a novel dimension to the clinical utility of gabapentinoids. While neither drug directly influences insulin secretion or glucose uptake, the reduction in mean amplitude of glycemic excursions and the increase in time-in-range imply indirect stabilization of glucose metabolism, potentially mediated through reduced sympathetic overactivity and stress responses. This observation aligns with findings from a Korean cohort study where both gabapentin and pregabalin were associated with improved neuropathic outcomes without worsening HbA1c values (25). The reduction in glucose variability may, therefore, contribute indirectly to neuronal recovery by minimizing oxidative stress and inflammatory signaling in peripheral tissues (26).

From a clinical perspective, the comparative superiority of pregabalin must be interpreted within the context of cost and accessibility. Cost-effectiveness analyses have consistently found pregabalin to yield marginally higher clinical benefit at substantially greater expense (27). Given that gabapentin achieved statistically significant improvement across all parameters, its affordability renders it a pragmatic choice, particularly in low- and middle-income healthcare settings such as Pakistan. Thus, gabapentin remains a viable and economically favorable alternative for patients where pregabalin cost may limit adherence.

The present study's strengths include its randomized design, objective electrophysiological outcomes, and use of continuous glucose monitoring to evaluate glycemic variability. These parameters provide physiological evidence of drug efficacy beyond subjective pain scores, offering a more comprehensive view of neural and metabolic modulation. Additionally, strict adherence monitoring and per-protocol analysis strengthened the internal validity of results.

However, certain limitations warrant consideration. The relatively short 12-week follow-up may have underestimated the full extent of nerve regeneration, which often requires several months. The study was also single-center, limiting generalizability to broader populations. The absence of pain or quality-of-life assessment precluded correlation between physiological improvement and patient-perceived benefit. Furthermore, potential confounders such as variations in diet, physical activity, and concurrent medications affecting glucose metabolism were controlled but not fully eliminated. Future studies with larger sample sizes, multicenter recruitment, and longer duration could better elucidate the durability and metabolic mechanisms underlying the observed improvements. Exploration of combined pharmacologic and lifestyle interventions—such as antioxidant supplementation or time-restricted feeding—may further clarify synergistic effects on nerve recovery and glucose stability.

The present study substantiated that both gabapentin and pregabalin enhance sensory nerve function and reduce glycemic variability in patients with diabetic neuropathy, with pregabalin showing a quantitatively superior effect. The findings extend the therapeutic significance of calcium channel modulators from symptomatic relief to functional neural improvement, supporting their broader role in comprehensive diabetic neuropathy management. These results reinforce the importance of individualized therapy considering both efficacy and cost, especially in resource-limited settings.

CONCLUSION

The study concluded that both gabapentin and pregabalin significantly improved sensory nerve conduction and reduced glycemic variability in patients with type 2 diabetes mellitus and peripheral neuropathy. Pregabalin produced slightly greater electrophysiological and metabolic improvements, reflecting its higher potency and consistent absorption, while gabapentin remained a clinically effective and cost-efficient option. These results highlight the therapeutic potential of calcium channel modulators in enhancing nerve function and metabolic stability beyond pain relief. Incorporating such agents into multidisciplinary diabetic care may promote early neurological recovery and better long-term outcomes for patients with diabetic neuropathy.

AUTHOR CONTRIBUTION

Author	Contribution
Farah Nadia Sheikh	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Rizwana Riaz	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published

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