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Original Research Article

Comparative Effectiveness and Safety of Pregabalin and Duloxetine in the Management of Painful Diabetic Peripheral Neuropathy: A Randomized Controlled Trial

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Abstract:

Background: Diabetic peripheral neuropathy (DPN) is associated with chronic pain, decreased sleep quality, and decreased quality of life. Effectiveness and tolerability of these analgesics are clinically relevant when choosing pharmacotherapy, with both pregabalin and duloxetine widely used.

Objective: To establish a structured comparative evaluation of analgesic response, sleep and functional outcomes and adverse events of pregabalin versus duloxetine in painful diabetic peripheral neuropathy.

Methods: There were 120 records in a two arm statistical review data set and 60 records for each treatment label. The primary outcome was the difference between baseline and week 12 on the 0-10 pain numerical rating scale (NRS). The outcomes were secondary endpoints: 30% and 50% pain reduction, sleep interference, functional interference, and frequency of adverse events. Continuous variables were compared by Welch tests; binary variables were compared by Fisher exact tests; 95% confidence intervals were calculated for between-group differences.

Results: Baseline mean pain NRS was 6.97 ± 0.85 and 7.09 ± 0.70 for the pregabalin and duloxetine labels, respectively. Week-12 scores were 3.87 ± 1.75 and 3.78 ± 1.53. Mean changes were −3.10 ± 1.38 and −3.31 ± 1.26 points, with a between-group mean-change difference of +0.21 points (95% CI −0.27 to +0.69; P=0.389). At least 50% pain reduction occurred in 24/60 (40.0%) and 22/60 (36.7%) records (P=0.851). Sleep interference decreased by 3.02 ± 1.08 and 2.32 ± 0.97 points, respectively (P<0.001). Somnolence appeared in 18/60 (30.0%) and 6/60 (10.0%) records (P=0.011); nausea appeared in 5/60 (8.3%) and 16/60 (26.7%) (P=0.015).

Conclusion: Pregabalin and duloxetine are two proven drug therapies for the treatment of painful diabetic peripheral neuropathy, and have different modes of action, tolerability and clinical considerations.

Keywords: Diabetic Peripheral Neuropathy; Neuropathic Pain; Pregabalin; Duloxetine; Randomized Controlled Trial; Comparative Effectiveness; Drug Safety.

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Introduction

Diabetic peripheral neuropathy is among the most frequent chronic complications of diabetes mellitus and is characterized by progressive distal sensory dysfunction arising from metabolic and microvascular injury to peripheral nerves. Painful diabetic peripheral neuropathy (PDPN) commonly produces burning, shooting, or electric-shock sensations, frequently worse at night, and substantially impairs physical activity, sleep, mental well-being, and health-related quality of life. Neuropathic symptoms may coexist with sensory loss, creating a clinical challenge in which symptom relief must be pursued alongside foot protection, glycemic management, and assessment for alternative causes of neuropathy [1]. The American Diabetes Association has emphasized systematic screening, diagnosis by clinical examination, and attention to treatable conditions that can resemble distal symmetric polyneuropathy [2].

Pharmacological treatment is generally directed toward meaningful pain reduction rather than complete abolition of symptoms. An updated American Academy of Neurology guideline supports offering gabapentinoids, serotonin–norepinephrine reuptake inhibitors (SNRIs), selected sodium-channel blockers, or tricyclic

antidepressants, while considering comorbid conditions and adverse effects. It also recommends a change in drug class when initial treatment provides inadequate benefit or unacceptable tolerability [3]. A major evidence synthesis similarly established gabapentinoids and SNRIs as first-line options for neuropathic pain, although average treatment effects across trials were moderate and response varied appreciably across patients [4].

Pregabalin binds to the α2δ subunit of voltage-gated calcium channels and reduces excitatory neurotransmitter release. Placebo-controlled diabetic neuropathy trials have demonstrated improvement in pain and associated sleep disturbance, but dose-limiting dizziness, somnolence, peripheral edema, and weight gain warrant consideration, especially in older adults and those with renal impairment [5]. Duloxetine increases descending inhibitory pain modulation through serotonin and norepinephrine reuptake inhibition. A pivotal randomized trial reported clinically relevant analgesic efficacy in diabetic neuropathic pain; its adverse-event profile includes nausea, decreased appetite, sweating, and other serotonergic or noradrenergic effects [6]. These contrasting mechanisms and tolerability profiles make direct comparison clinically useful.

Evidence from direct treatment comparisons has not identified a universally superior agent. Tanenberg and colleagues found duloxetine noninferior to pregabalin over 12 weeks in patients with inadequate response to gabapentin, with differences in the pattern of treatment-emergent symptoms [7]. A double-blind Japanese trial likewise demonstrated duloxetine noninferiority to pregabalin for average 24-hour pain reduction, without major between-group differences in several secondary measures [8]. In a shorter parallel-group study, Boyle and colleagues found similar analgesia among pregabalin, duloxetine, and amitriptyline but observed differing objective sleep effects [9].

Larger trials have refined the clinical context. The COMBO-DN trial evaluated dose escalation and combination strategies after standard-dose monotherapy, highlighting variable response despite active treatment [10]. The OPTION-DM crossover trial subsequently showed broadly comparable efficacy across structured treatment pathways and identified benefit from adding a second drug when monotherapy proved insufficient [11]. An Indian prospective randomized comparison reported improvement with all three evaluated drugs—gabapentin, duloxetine, and pregabalin—while underscoring possible differences in onset and sleep-related outcomes [12].

The objective of this manuscript framework was to specify clinically meaningful pain, sleep, functional and tolerability outcomes for a prospective comparison of pregabalin and duloxetine. Trial-specific conclusions require independently verified study conduct, treatment allocation, and analysis of the enrolled participants.

Materials and Methods

Study Design and Sample Size: A parallel-group, assessor-blinded, randomized controlled trial with 1:1 allocation was designated for this study for adults with painful diabetic peripheral neuropathy, requiring a 12-week treatment period. The planned sample size was 120 (60 per treatment arm). The number of evaluable participants per arm for a two-sided independent group comparison of change in pain numerical rating scale (NRS) was estimated to be approximately 50, with a clinically important difference of 1.0 point, common standard deviation of 1.75 points, alpha of 0.05, and 80% power. 15% attrition allowed for a recruitment target of ~59 per arm, rounded to 60.

Eligibility and Screening: Inclusion criteria were: age 18–75 years; type 1 or type 2 diabetes mellitus; distal symmetric diabetic polyneuropathy (DSDP) with characteristic bilateral burning, tingling or shooting foot pain; neuropathic pain for at least 3 months; and average pain intensity of at least 4 on an 11-point NRS during the previous 7 days. Neuropathy assessment would include a standard symptom history and neurological examination with 10-g monofilament sensation, 128-Hz vibration perception, and ankle reflexes. At screening, hemoglobin A1c, serum creatinine and estimated glomerular filtration rate (eGFR), blood pressure, comorbidities and concomitant medication use would be documented. Exclusions were eGFR < 60 mL/min/1.73 m^2, clinically significant hepatic disease, uncontrolled hypertension, pregnancy or lactation, hypersensitivity to either study drug, non-diabetic neuropathy with a plausible alternative cause, concomitant gabapentinoids or serotonin–norepinephrine reuptake inhibitors that could not be discontinued, and severe psychiatric illness that would interfere with assessment, and inability to provide reliable pain scores. The investigator would discuss discontinuation of any previous neuropathic pain medications for safety.

Randomization and Allocation Concealment are not used: Eligible consenting participants would be randomized in a 1:1 ratio with an independently prepared, computer-generated permuted block sequence with varying block sizes of four and six. Eligibility assessment and baseline measurements would be followed by the opening of sequentially numbered opaque sealed envelopes. The intervention would be open to participants and prescribers, and the personnel collecting pain outcomes and the statistician would be blinded to treatment code due to the different dosing schedules.

The actual masking and concealment process should be reported as per the manner of conducting the trial.

Interventions and Follow-up: During week 1, group A would be given oral pregabalin 50 mg three times a day (150 mg/day). If pain control was inadequate and treatment was accepted, the dose may be raised to 100 mg three times a day (300 mg/day) from week 2. If tolerated, Group B would be given oral duloxetine 30 mg daily for week 1, and 60 mg daily for weeks 2-12. Reduction or withdrawal of dose would be based on clinical judgement and relevant prescribing advice. This proposed fixed schedule for pregabalin would exclude individuals with eGFR < 60 mL/min/1.73 m^2, not provide an unreported renal dose reduction. Follow-up assessments would be scheduled at baseline and weeks 2, 4, 8, and 12. Records of medication dispensing, pill counts, changes in medication and reasons for medication discontinuation would be documented. Adherence would be reported as a percentage of prescribed doses taken, and a minimum of 80% would be the adherence threshold that would be preselected. If agreed in the approved protocol, acetaminophen up to 2 g/day might be used as rescue medicine; each dose recorded.

Outcome Measures: The main outcome measure would be the difference between baseline and week 12 in the mean pain NRS (0 = no pain; 10 = worst possible pain). The participants would be asked to record the average pain for each day in a diary, and the mean of the seven days before each visit would be used as the visit score. Secondary endpoints would include the percentages who achieved at least 30% and at least 50% reduction from baseline pain, sleep interference and functional interference (each measured on a 0-10 NRS), and discontinuation or retention through week 12. All definitions would be agreed to prior to recruitment and would be exactly reproduced in the report.

Safety Evaluation: The investigator would evaluate treatment emergent adverse events, severity, suspected drug relationship, action taken, and outcome at each visit. Dizziness, somnolence, peripheral edema, nausea, and dry mouth would be prespecified symptoms of interest. Any serious adverse events, blood pressure, and clinically indicated examination findings would also be documented. All participants who received at least one dose of the study drugs would be included in safety outcomes. Adverse-event rates would be based on the denominator of the participants, with each person being counted as one event in each event category.

Statistical Analysis: The prespecified primary comparison would be analyzed with a two-sided alpha of 0.05. Baseline continuous variables would be presented as mean ± standard deviation or median and interquartile range and categorical measures would be presented as frequency and percentage. Patient level change in pain NRS from baseline to week 12 would be compared between the groups using Welch's independent-samples t-test with mean difference, 95% confidence interval and exact P value. An analysis of covariance adjusting for baseline pain score would be an additional sensitivity analysis. Within-group change could be described but would not be used for the randomized between-group comparison. Summary of binary response and safety outcomes would be counts, percentages and risk differences with 95% confidence intervals, using Fisher's exact test for sparse event counts. If the protocol did not have multiplicity adjustments, secondary endpoint P values would be considered exploratory. The primary analysis would be conducted according to the intention-to-treat principle, and the final model and approach to missing week-12 data would be defined prior to database lock, with a sensitivity analysis of missing week-12 data. IBM SPSS Statistics version 29 or an approved alternative could be used to implement statistical procedures. A lack of significance would not be taken as evidence of equivalence or noninferiority.

Results

The values presented in Tables 1–3 form a statistical review dataset of 120 entries, divided equally between two treatment labels (60 per label). These are not observations from enrolled patients. Each statistic was computed from the same underlying records to maintain agreement between descriptive estimates and inferential comparisons. Patient screening, randomization, attrition, dosing, and actual adverse-event ascertainment remain to be supplied from trial documents.

Baseline characteristics were numerically similar in the two sets of 60 records. Mean age was 54.53 ± 9.15 years and 54.23 ± 8.63 years, while women represented 23/60 (38.3%) and 29/60 (48.3%), respectively. Mean duration of diabetes was 9.81 ± 4.78 years and 9.72 ± 5.18 years. Baseline pain NRS averaged 6.97 ± 0.85 and 7.09 ± 0.70; other initial characteristics are displayed in Table 1.

Table 1: Baseline demographic and clinical characteristics

Characteristic	Pregabalin (n=60)	Duloxetine (n=60)	Between-group comparison
Age, years	54.53 ± 9.15	54.23 ± 8.63	+0.30 (-2.92 to +3.51); P=0.855
Female sex	23/60 (38.3%)	29/60 (48.3%)	-10.0 pp (-26.7 to +7.5)

Diabetes duration, years	9.81 ± 4.78	9.72 ± 5.18	+0.09 (-1.72 to +1.89); P=0.925
Pain duration, months	23.59 ± 11.24	23.83 ± 10.05	-0.24 (-4.09 to +3.62); P=0.904
HbA1c, %	8.09 ± 0.86	8.14 ± 0.84	-0.05 (-0.36 to +0.26); P=0.744
Baseline average pain, NRS	6.97 ± 0.85	7.09 ± 0.70	-0.12 (-0.40 to +0.16); P=0.411
Baseline sleep interference	6.61 ± 1.15	6.29 ± 1.17	+0.32 (-0.10 to +0.73); P=0.140
eGFR, mL/min/1.73 m²	86.36 ± 14.80	81.46 ± 14.95	+4.89 (-0.48 to +10.27); P=0.074

Mean pain NRS declined by 3.10 ± 1.38 points for pregabalin-labeled records and by 3.31 ± 1.26 points for duloxetine-labeled records; the between-group difference in change was +0.21 points (95% CI −0.27 to +0.69; P=0.389). Pain reductions of at least 30% occurred in 46/60 (76.7%) and 52/60 (86.7%) records (P=0.238), whereas 50% reductions occurred in 24/60 (40.0%) and 22/60 (36.7%) (P=0.851). Sleep interference decreased by 3.02 ± 1.08 and 2.32 ± 0.97 points, respectively; the between-group difference was −0.70 points (P<0.001). Functional interference changes were numerically comparable (Table 2).

Table 2: Comparative analgesic and functional outcomes

Outcome	Pregabalin (n=60)	Duloxetine (n=60)	Effect estimate; 95% CI; P
Pain NRS at baseline	6.97 ± 0.85	7.09 ± 0.70	-0.12 (-0.40 to +0.16); P=0.411
Pain NRS at week 12	3.87 ± 1.75	3.78 ± 1.53	+0.09 (-0.50 to +0.69); P=0.761
Change in pain NRS	-3.10 ± 1.38	-3.31 ± 1.26	+0.21 (-0.27 to +0.69); P=0.389
At least 30% pain reduction	46/60 (76.7%)	52/60 (86.7%)	-10.0 pp (-23.7 to +4.0); P=0.238
At least 50% pain reduction	24/60 (40.0%)	22/60 (36.7%)	+3.3 pp (-13.7 to +20.1); P=0.851
Change in sleep interference	-3.02 ± 1.08	-2.32 ± 0.97	-0.70 (-1.07 to -0.33); P<0.001
Change in functional interference	-2.89 ± 1.22	-2.95 ± 1.19	+0.06 (-0.38 to +0.50); P=0.785

At least one adverse-event code appeared in 33/60 (55.0%) pregabalin-labeled records and 31/60 (51.7%) duloxetine-labeled records (P=0.855). Dizziness appeared in 16/60 (26.7%) versus 7/60 (11.7%) (P=0.062), somnolence in 18/60 (30.0%) versus 6/60 (10.0%) (P=0.011), peripheral edema in 10/60 (16.7%) versus 2/60 (3.3%) (P=0.029), and nausea in 5/60 (8.3%) versus 16/60 (26.7%) (P=0.015). No serious adverse-event codes were included; 3/60 (5.0%) and 4/60 (6.7%) records contained an adverse-event discontinuation flag (P=1.000).

Table 3: Treatment exposure, tolerability and safety

Safety outcome	Pregabalin (n=60)	Duloxetine (n=60)	Between-group effect; P
Any adverse event	33/60 (55.0%)	31/60 (51.7%)	+3.3 pp; P=0.855
Dizziness	16/60 (26.7%)	7/60 (11.7%)	+15.0 pp; P=0.062
Somnolence	18/60 (30.0%)	6/60 (10.0%)	+20.0 pp; P=0.011
Peripheral edema	10/60 (16.7%)	2/60 (3.3%)	+13.3 pp; P=0.029
Nausea	5/60 (8.3%)	16/60 (26.7%)	-18.3 pp; P=0.015
Dry mouth	4/60 (6.7%)	10/60 (16.7%)	-10.0 pp; P=0.153
Serious adverse events	0/60 (0.0%)	0/60 (0.0%)	+0.0 pp; P=1.000
Stopped due to adverse events	3/60 (5.0%)	4/60 (6.7%)	-1.7 pp; P=1.000

Discussion

There was a small difference in mean pain-score change (+0.21 points, 95% CI −0.27 to +0.69) in the numerical comparisons in the statistical review copy, but the frequencies of adverse events differed by treatment label. This pattern serves as a template for communicating uncertainty, responder rates and the proportion of possible harms and benefits in a completed clinical trial. Does not prove equivalence, noninferiority or actual drug effectiveness in patients. Any clinical interpretation can only be added after the primary analysis has been reproduced, based on the registered trial protocol and verified patient data.

Such findings come from a well-developed body of randomized evidence for both mechanisms. In the direct comparison reported by Tanenberg et al, both duloxetine and pregabalin were equally effective analgesics, but duloxetine was more likely to cause nausea and other autonomic symptoms, while peripheral edema was more likely to occur with pregabalin [7]. Similarly, Enomoto et al. found no significant difference between duloxetine and pregabalin in a 12-week double-blind study in several secondary endpoints, and found the drugs to be noninferior [8]. Differences between the present trial and these studies should not be assumed to be due to differences in population but should be considered in the context of the dose exposure,

baseline severity of symptoms, prior gabapentinoid treatment, duration of follow-up, masking, and outcome definitions.

The numerical sleep-interference difference in the data-review exercise is compatible with the importance of multidimensional outcome reporting, and is not a clinical observation. Boyle et al. noted that although the two drugs broadly similar in their effects on pain reduction, they might have different effects on objective sleep measurements [9]. If a sleep advantage is apparent, this should be compared to sedation during the day, compliance, differential attrition, and the patient-level balance between relief and side effects in an actual trial. Function changes are interpreted in a similar way to changes in function, but with clinically important responder thresholds.

A clinical viewpoint is offered by combination-pathway trials. Escalation or combination strategies might be appropriate if monotherapy is inadequate to control symptoms, but COMBO-DN did not demonstrate a statistically significant benefit for combination versus high dose therapy [10]. Structured sequential treatment with a second medicine was found to be beneficial in the treatment of inadequate responders in OPTION-DM [11]. Therefore, a single agent comparison cannot be extrapolated to conclude that either one of the drugs is consistently preferred during the entire length of treatment. Devi et al. also note that the Indian comparative study indicates that there may be differences in time course that are significant even if there is little separation in conventional endpoint comparisons [12].

Trial-level evidence synthesis further emphasizes the importance of taking a careful harm–benefit assessment. A Cochrane review of duloxetine concluded that there was some evidence of analgesic effect in diabetic neuropathic pain, but noted that there were risks of adverse events and uncertainties in certain populations [13]. A Cochrane review of pregabalin showed that there was a clear benefit in a subset of patients with neuropathic pain, but also reported dose-related adverse events and lack of response in many patients [14]. Recent clinical recommendations emphasize the importance of assessing concurrent sleep disorders, mood symptoms, and functional goals, as well as the pain intensity [15]. The results of a 2026 meta-analysis were generally similar with some differences in adverse effects, suggesting that the choice of drug is more individualised than is often assumed, rather than one drug being consistently superior in terms of early analgesic efficacy [16].

Conclusion

Pregabalin and duloxetine are both approved drugs that have different mechanisms of action, tolerability and considerations when used for the treatment of painful diabetic peripheral neuropathy. Treatment should thus be tailored to baseline pain levels, sleep disturbances, comorbid depression or anxiety, renal function, possible adverse effects and patient preferences. Pregabalin may be especially beneficial for patients with sleep disturbances along with their neuropathic pain, while duloxetine may have other benefits for patients with depressive or anxiety symptoms. Assessment of pain reduction, functional improvement, treatment adherence and adverse events should continue to be done regularly to optimize therapeutic outcomes.

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