

## Original Research Article

# Assessment of quality of life in patients with trigeminal neuralgia receiving pregabalin as add-on therapy to carbamazepine

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**Received:** 26 August 2026

**Revised:** 07 October 2026

**Accepted:** 26 October 2026

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## ABSTRACT

**Background:** Trigeminal neuralgia significantly affects patients' quality of life due to recurrent, severe pain. This study aimed to compare the quality of life in patients with trigeminal neuralgia receiving pregabalin as add-on therapy to carbamazepine.

**Methods:** A prospective, open-label, randomized comparative study was conducted on 50 patients from November 2015 to September 2016. The patients were divided into two groups (n=25 each) and treated for 12 weeks. Group I received carbamazepine 200 mg/day, titrated up to 1000 mg/day, while Group II received pregabalin 75 mg OD plus carbamazepine 200 mg/day, with pregabalin titrated up to 300 mg/day. Quality of life was assessed using PDI at weeks 4, 8 and 12.

**Results:** Intragroup analysis showed a statistically significant reduction in mean PDI scores ( $p < 0.05$ ) from baseline at 4, 8, and 12 weeks in both groups, indicating improved quality of life. Intergroup analysis showed no significant difference at 4 weeks ( $p = 0.15$ ), whereas pregabalin added to carbamazepine produced significantly better quality of life at 8 and 12 weeks ( $p < 0.05$ ).

**Conclusions:** Present study suggested that pregabalin as an add on therapy was found to cause significant improvement in quality of life & reduction in pain scoring at 8<sup>th</sup> and 12<sup>th</sup> weeks.

**Keywords:** Carbamazepine, Pregabalin, Trigeminal neuralgia, Pain disability index

## INTRODUCTION

Trigeminal neuralgia (TN) which is also known as Fothergill disease or Tic douloureux disease is a form of neuropathic pain characterized by the occurrence of abrupt pain which is generally one-sided, severe, brief, sharp and recurrent in the distribution area of one or several branches of the V<sup>th</sup> nerve.<sup>1</sup> Pain is commonly evoked by trivial stimuli including washing, shaving, smoking, talking and brushing the teeth but may also occur spontaneously.<sup>2</sup>

The International Headache Society (IHS) in year 2013 defined strict clinical criteria for trigeminal neuralgia

diagnosis.<sup>2</sup> According to these criteria a diagnosis can be made when there are at least three attacks of unilateral facial pain i.e., occurring in one or more division of the trigeminal nerve with no radiation beyond the trigeminal distribution. Pain with atleast three of the following four characteristics i.e., recurring in paroxysmal attacks lasting from a fraction of a second to two minutes, severe intensity, electric shock-like shooting, stabbing pain, precipitated by innocuous stimuli to the affected side of face. Trigeminal neuralgia is further divided as typical and atypical.<sup>3</sup> In Typical type there is an idiopathic episodic pain lasting for several seconds, with pain-free intervals between the attacks whereas in atypical type there is

continuous or repeated pain between transient paroxysms.<sup>3</sup> The prevalence of trigeminal neuralgia in the general population is 0.015% and overall incidence ranges from 12.6 to 27 per 100,000/year which increases with the advancing age.<sup>4,5</sup> Middle aged and elderly persons are primarily affected; higher incidence is seen in women with 5.9 cases per 100,000 in females as compared with men with 3.5 cases per 100,000 in males.

The diagnosis of trigeminal neuralgia is purely clinical and is made on the basis of characteristic pain in the trigeminal nerve distribution. Patients with trigeminal neuralgia suffer pain episode for months or years before the condition is finally diagnosed and unfortunately episodes of TN have a devastating impact on patients Quality of life. A timely accurate diagnosis of trigeminal neuralgia is therefore particularly important, because a variety of specific treatments can greatly reduce or totally eliminate trigeminal neuralgia pain symptoms in most patients. There have been surprisingly few studies relating to quality-of-life outcomes in TN. A couple of long-term cohort studies have demonstrated that many TN patients suffer depression, which can be alleviated following curative surgical procedures.<sup>6</sup>

The first-line of treatment is always medical therapy. Of the drugs currently used to treat trigeminal neuralgia, most of them are anticonvulsants. Additionally, only a handful of these drugs have been investigated in small randomized control trials for the treatment of trigeminal neuralgia and many of these trials have methodological flaws and are outdated.<sup>7</sup> Carbamazepine is the most studied medication for treatment of trigeminal neuralgia and is therefore the drug of choice. It is a sodium channel blocker and promotes the inactivated state of voltage activated sodium ion channels.

Pregabalin is a calcium channel blocker which shows specific affinity for the  $\alpha 2\delta$  auxiliary subunits of voltage dependent calcium channels. The pregabalin exhibits analgesic, anxiolytic and anticonvulsant properties. It is structurally related to gabapentin and absorbed orally. It is not bound to plasma protein and is excreted unchanged mainly in urine without undergoing metabolism, its half-life is approximately 6 hour. In randomized, placebo-controlled clinical trials pregabalin has demonstrated efficacy in reducing pain in patients with diabetic neuropathy and post herpetic neuralgia thereby significantly improving affective symptoms, sleep and quality of life.<sup>8</sup> Furthermore the pharmacokinetic profile of pregabalin allows for easy management and rapid dose escalation to therapeutic dosages.

Presently, surgical treatment options for trigeminal neuralgia are generally explored only when patients are refractory to medical management. A patient is said to be refractory when he/she cannot bear the adverse effects of the medication, experience breakthrough pain or cannot

take the medications because he/she is medically complex patients with polypharmacy for other conditions.<sup>9</sup>

The present study has been planned in the view of the fact that, trigeminal neuralgia is common neuropathic pain disorder. Also, it has a potential risk for causing depression and poor quality of life if left untreated. Despite the fact that many standard drugs such carbamazepine and other anticonvulsants are available for treatment of trigeminal neuralgia but still, there is a search for an ideal analgesic with minimal side effects, maximal analgesia and improved patient compliance. Pregabalin is a commonly used therapy currently recommended as first line treatment for a number of neuropathic pain conditions such as diabetic peripheral neuropathy, postherpetic neuralgia, spinal cord injury etc. Pregabalin has been used as an add-on therapy along with NSAIDs, opioids, antiepileptic drugs and antidepressants drugs in uncontrolled neuropathic pain and its administration resulted in significant reduction in pain and improvement in the psychological well-being. So, a comparative study was planned where combination of carbamazepine and pregabalin was compared with carbamazepine as a standard line of drug.

## METHODS

This was a prospective, open label, randomized, comparative clinical study conducted by the Department of Pharmacology in collaboration with the Department of Neurology at Pt. B.D. Sharma PGIMS, Rohtak. In present study patients of either sex of more than 18 yrs of age attending the OPD in Neurology department with facial pain of trigeminal neuralgia were selected. The study was conducted over a period of 1 year and 50 patients were included. The Study was in accordance with the principles of good clinical practice (ICH-GCP) and declaration of Helsinki. The study was conducted after obtaining ethical clearance from institutional ethical committee (IEC). An informed consent was obtained from all the patients enrolled in this study. The eligible patients were randomly divided into two study groups i.e. Group I and Group II with the help of computer-generated random numbers. Each study group had 25 patients and were found to be comparable at the time of their initial visit with regard to demographic parameters such as age, gender, side involved and other parameters (as shown in Table 1). Patients were allocated to receive one of the 2 different treatments in an open fashion.

Group I (n=25) received tablet carbamazepine as a monotherapy initially 200 mg daily per orally in divided doses and gradually built up as per clinical response with maximum titrated dose upto 1000 mg/day. Group II (n=25) received capsule pregabalin 75 mg OD and tablet carbamazepine 200 mg daily per orally in divided doses and dose gradually built up as per clinical response with maximum titrated dose upto 300mg/day for pregabalin for a period of 12 weeks and subjected to clinical assessment for safety and efficacy of drug (effect on quality of life).

During the study, patients were not permitted to take any non-study drugs. Inclusion criteria were diagnosed cases of trigeminal neuralgia, patients of either gender of more than 18 years of age, patients who are ready to give written informed consent. Exclusion criteria were patients with history of psychiatric illness, patients with severe hepatic and renal disorders and other co-morbid conditions, pregnant and lactating women, history of known hypersensitivity to pregabalin and carbamazepine, patients who refused to give informed consent. Quality of life assessment was determined by Pain disability index (PDI). This index was developed at St. Louis University Medical Center which was designed to measure the degree to which aspects of life are disrupted by chronic pain. In other words, one would like to know how much pain is preventing you from doing what you would normally do or from doing it as well as you normally would. There are 7 categories of life activity listed and patients responded to each category indicating the overall impact of pain in one's life, not just when pain is at its worst. Patients will circle the number on the scale that describes the level of disability ones typically experience.

In each category a score of 0 means no disability at all, and a score of 10 signifies that all of the activities in which you would normally be involved have been totally disrupted or

prevented by your pain. So, this scale can be used to evaluate patients initially to monitor them over time and to

judge the effectiveness of interventions. Measures of disability related to pain: family and home responsibilities: activities related to home and family; recreation: hobbies sports and other leisure time activities; social activity: participation with friends and acquaintances other than family members; occupation: activities partly or directly related to work including housework or volunteering; sexual behavior: frequency and quality of sex life; self-care: personal maintenance and independent daily living (bathing and dressing); life-support activity: basic life-supporting behaviors (eating, sleeping and breathing).

### Data analysis

Data was expressed as Mean $\pm$ SEM. Both intragroup and intergroup statistical analysis was done. Intragroup analysis was done by using ANOVA. Intergroup analysis was done by using unpaired 't' test. A p value of less than 0.05 was considered as statistically significant.

## RESULTS

### PDI scoring in GP I

Intragroup analysis (Table 2 and Figure 4): at baseline mean PDI score was 48.08 $\pm$ 1.03. There was clinically significant reduction in mean pain disability index scoring at week 4 (43.52 $\pm$ 1.07), 8 (40.24 $\pm$ 0.95) and 12 week (37.44  $\pm$  0.94), as compared to baseline.

**Table 1: Demographic profile of study participants in both the groups (n=25 in each group).**

| Demographic profile                   | Group I<br>(n=25) | Group II<br>(n=25) |
|---------------------------------------|-------------------|--------------------|
| <b>Age in years</b>                   | 52.9 $\pm$ 3.59   | 51.3 $\pm$ 3.33    |
| <b>Gender</b>                         |                   |                    |
| Male                                  | 19 (76%)          | 18 (72%)           |
| Female                                | 06 (24)           | 07 (28%)           |
| <b>Side of face involved</b>          |                   |                    |
| Right side                            | 16 (64%)          | 18 (72%)           |
| Left side                             | 09 (36%)          | 07 (28%)           |
| Vascular loop around Trigeminal nerve | 4 (16%)           | 6 (24%)            |
| Drug allergy                          | NO                | NO                 |

Age is expressed as Mean $\pm$ SEM (standard error of mean) while categorical values are expressed as actual number of patients and their percentage.

**Table 2: Intragroup comparison of pain disability index in group I (n=25).**

| Time interval               | Group 1          |                      |
|-----------------------------|------------------|----------------------|
|                             | Mean $\pm$ SEM   | Change from baseline |
| <b>Baseline</b>             | 48.08 $\pm$ 1.03 | ----                 |
| <b>4<sup>th</sup> week</b>  | 43.52 $\pm$ 1.07 | 4.56*                |
| <b>8<sup>th</sup> week</b>  | 40.24 $\pm$ 0.95 | 7.84*                |
| <b>12<sup>th</sup> week</b> | 37.44 $\pm$ 0.94 | 10.64*               |

\*p<0.05 indicates significant values at 4,8 and 12 week. All values are expressed as Mean $\pm$ SEM. Gradual decrease in pain disability index score with initiation of treatment at the end of 4, 8 and 12 week signifies improvement in quality of life.

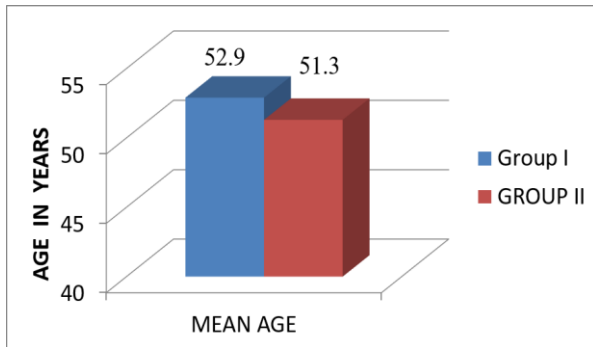


**Figure 1: Study population-age characteristics.**

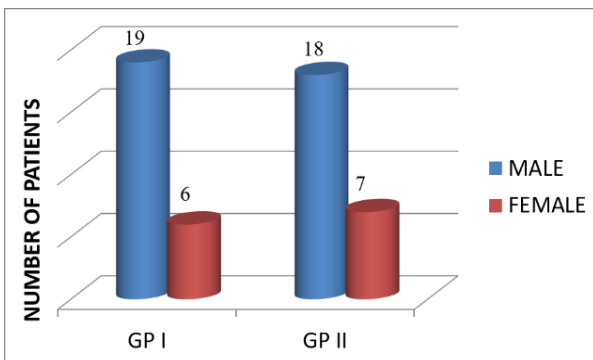


**Figure 2: Sex distribution in two groups.**

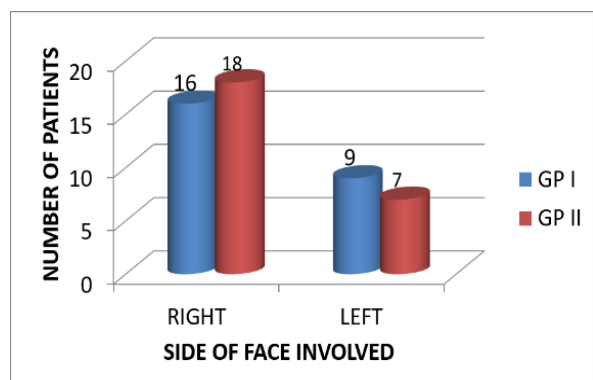


**Figure 3: Facial pain in two groups.**

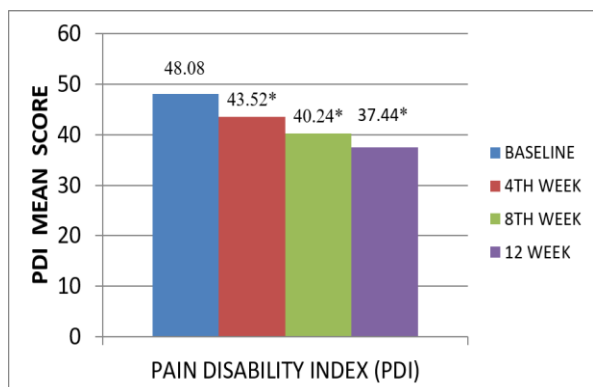


**Figure 4: PDI scoring in group I.**

### PDI scoring in GP II

Intragroup analysis (Table 3 and Figure 5): at baseline mean PDI score was  $47.80 \pm 0.53$ . There was statistically significant reduction in mean pain disability index score at week 4 ( $41.60 \pm 0.51$ ), 8 ( $37.20 \pm 0.52$ ) and 12 ( $33.12 \pm 0.60$ ), when compared to baseline.

**Table 3: Intragroup comparison of pain disability index in group II (n=25).**

| Time interval         | Group II   |                      |
|-----------------------|------------|----------------------|
|                       | Mean±SEM   | Change from baseline |
| Baseline              | 47.80±0.53 | -----                |
| 4 <sup>th</sup> week  | 41.60±0.51 | 6.20*                |
| 8 <sup>th</sup> week  | 37.20±0.52 | 10.60*               |
| 12 <sup>th</sup> week | 33.12±0.60 | 14.68*               |

\* $p < 0.05$  indicates significant values at 4, 8 and 12 week. All values are expressed as Mean±SEM. Gradual decrease in pain disability index score with initiation of treatment at the end of 4, 8 and 12 week signifies improvement in Quality of life

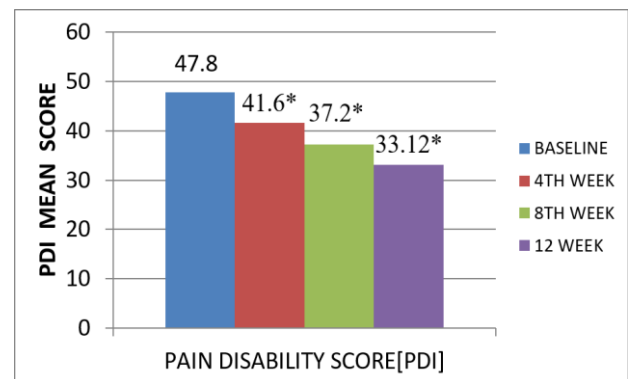


**Figure 5: PDI scoring in group II.**

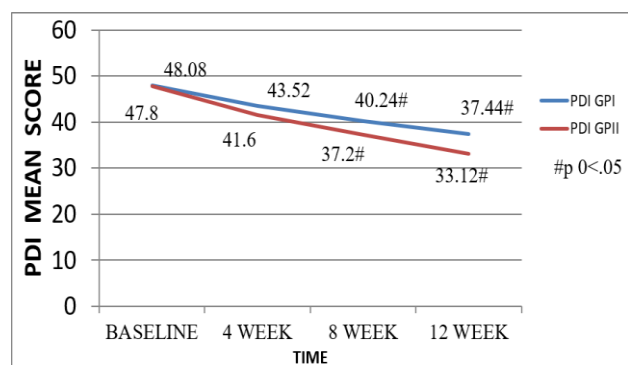


**Figure 6: Profile plot of QOL scoring between two groups.**

Intergroup analysis (Table 4 and Figure 6): At the end of 4 weeks reduction in pain disability index score was 4.56 and 6.20 which was not significant ( $p > 0.05$ ). But at the end of 8 weeks better response was seen with group B i.e more reduction in pain disability index scoring value by (10.60) as compared to (7.84) group A which was statistically

significant reduction ( $p<0.05$ ). Also, at the end of 12 weeks again better response was seen in group B with more reduction in pain disability index scoring value (14.68) as compared to (10.64) group A which was statistically significant ( $p<0.05$ ).

**Table 4: Intergroup comparison of pain disability index (PDI) between both the groups at baseline, 4, 8 and 12 weeks.**

| Time interval         | Group 1 (n=25)    | Group II (n=25)   | P value |
|-----------------------|-------------------|-------------------|---------|
| Time point            | Mean $\pm$ SEM    | Mean $\pm$ SEM    |         |
| Baseline              | 48.08 $\pm$ 1.03  | 47.80 $\pm$ 0.53  | 0.82    |
| 4 <sup>th</sup> week  | 43.52 $\pm$ 1.07  | 41.60 $\pm$ 0.51  | 0.151   |
| 8 <sup>th</sup> week  | 40.24 $\pm$ 0.95# | 37.20 $\pm$ 0.52# | <0.05   |
| 12 <sup>th</sup> week | 37.44 $\pm$ 0.94# | 33.12 $\pm$ 0.60# | <0.05   |

All values are expressed as Mean $\pm$ SEM. #  $p<0.05$  at 8<sup>th</sup> and 12<sup>th</sup> week.

## DISCUSSION

The International Association for the Study of Pain (IASP) describes pain as an unpleasant sensory and emotional experience that may arise from actual or potential tissue injury.<sup>10</sup> Trigeminal neuralgia (TN) is a distinctive facial pain syndrome characterized by recurrent, abrupt-onset episodes of intense, brief, electric shock-like or stabbing pain affecting one or more divisions of the trigeminal nerve. It is recognized as a classical neuropathic pain disorder with a long-established clinical history. In a randomized, placebo-controlled clinical trial, Gilron I et al demonstrated that pregabalin effectively alleviated pain in patients with diabetic neuropathy and postherpetic neuralgia.

In addition to pain reduction, pregabalin improved affective symptoms, sleep quality, and overall quality of life.<sup>11</sup> An important strength of the present study is its randomized design with an active comparator, allowing a direct comparison between carbamazepine monotherapy and pregabalin as an add-on treatment. Pérez C et al investigated the effectiveness of pregabalin (PGB) in improving patient-reported health outcomes among 65 pregabalin-naïve patients with trigeminal neuralgia who had not responded adequately to previous analgesic treatment.<sup>12</sup>

This prospective, multicentre observational study was conducted in primary care settings over 12 weeks. Patients received pregabalin either as monotherapy ( $n=36$ ) or as an add-on treatment ( $n=29$ ). The study demonstrated a mean reduction in baseline pain intensity of 40.0 $\pm$ 22.1 mm, corresponding to a 55.4% reduction (effect size [ES] = 2.32;  $P<0.0001$ ). A clinically meaningful pain reduction of at least 50% was achieved in 59.4% of participants, while the number of days with absent or mild pain increased by 34.6  $\pm$  29.3 days.

Significant improvements were also observed in anxiety and depression scores, sleep quality, functional disability, and overall health status. The EQ-5D score improved by 31.6 $\pm$ 22.2 mm (ES=1.67;  $P<0.0001$ ), with a corresponding gain of 0.0388 $\pm$ 0.0374 quality-adjusted life-years (QALYs). The findings of the present study are consistent with these observations, as pregabalin treatment was associated with improved quality of life. However, unlike Pérez C et al., who used the EQ-5D instrument, quality of life in our study was evaluated using the Pain Disability Index (PDI). Campbell et al. demonstrated the superiority of carbamazepine over placebo in the management of trigeminal neuralgia, with treatment also resulting in improved quality of life.<sup>13</sup>

Similarly, Franken JH et al conducted a randomized, double-blind, placebo-controlled trial to examine the effects of pregabalin on pain, tolerability, health status, and quality of life in patients with central neuropathic pain resulting from brain or spinal cord injuries.<sup>14</sup> Forty patients received either pregabalin or matching placebo capsules for four weeks. Pregabalin was administered at escalating doses of 150, 300 or 600 mg/day, depending on the patient's response to treatment. Pain intensity was assessed using the visual analogue scale (VAS), while health status and functional disability were evaluated using the EQ-5D and Pain Disability Index, respectively. Quality of life was assessed using the SF-36 questionnaire. Pregabalin treatment resulted in a statistically significant reduction in mean pain intensity compared with placebo ( $P=0.016$ ). Significant improvements were also observed in the bodily pain domain of the SF-36 and the Pain Disability Index.

These findings suggest that flexible-dose pregabalin therapy can provide clinically meaningful pain relief and improve functional health in patients with neuropathic pain. Other randomized controlled trials have similarly reported significant reductions in neuropathic pain following pregabalin treatment ( $P<0.05$ ). In the present study, quality of life was assessed using PDI scores. In group I, the mean baseline PDI score was 48.08 $\pm$ 1.03, which decreased to 37.44 $\pm$ 0.94 at the end of 12 weeks, representing an overall reduction of 10.64 $\pm$ 0.09. In group II, the corresponding baseline score was 47.80 $\pm$ 0.53, which declined to 33.12 $\pm$ 0.60 after 12 weeks, with an overall reduction of 14.68 $\pm$ 0.07. The greater reduction in PDI scores in group II indicates a comparatively greater improvement in pain-related disability among patients receiving pregabalin as an adjunct to carbamazepine than among those receiving carbamazepine alone. Crawford M et al assessed the effectiveness of pregabalin in routine clinical practice for the management of peripheral neuropathic pain in Denmark.<sup>15</sup> Their prospective, observational, noninterventional study included 128 patients, of whom 86 completed three months of pregabalin treatment and were included in the efficacy evaluation. A clinically relevant and statistically significant reduction of 2.2 points in average pain intensity was observed after three months of therapy. Improvements

were also reported in pain-related sleep disturbances, patients' global impression of change, quality of life, and work-related productivity.

The findings suggested that the addition of pregabalin to the treatment regimen provided meaningful pain relief and improved quality of life in a substantial proportion of patients with peripheral neuropathic pain. These observations are in agreement with the findings of our study, in which the addition of pregabalin to carbamazepine was associated with a greater reduction in pain-related disability. The present study has certain limitations.

A placebo control group was not included because of ethical considerations; therefore, the comparison was restricted to an active control group. The open-label design and absence of blinding may have introduced participant and observer bias. Furthermore, the relatively small sample size and reliance on subjective outcome measures may restrict the generalizability and interpretation of the findings. The follow-up period of 12 weeks was also insufficient to evaluate the long-term efficacy and safety of the treatment regimens.

Larger, multicentre randomized controlled trials with longer follow-up periods are therefore warranted to confirm the present findings. To the best of our knowledge, relatively few studies have specifically evaluated quality of life in patients with trigeminal neuralgia using the Pain Disability Index. The present study contributes to the existing literature by assessing changes in pain-related disability following carbamazepine monotherapy and pregabalin add-on therapy. The greater improvement observed in the pregabalin add-on group suggests that combination therapy may offer additional benefits in improving functional status and quality of life in patients with trigeminal neuralgia.

## CONCLUSION

There was statistically significant improvement in quality of life at 4th, 8th and 12 weeks, in both the groups when compared to the baseline i.e. both carbamazepine and pregabalin as an add on therapy to carbamazepine were effective in improving the quality of life in patients of trigeminal neuralgia. However, on intergroup comparison, pregabalin as an add on drug to carbamazepine (Group II) produced statistically significant improvement in quality of life at 8th and 12th weeks when compared to carbamazepine alone (Group I). The present study suggested that pregabalin as an add on therapy to carbamazepine was found to cause significant improvement in quality of life and reduction in pain scoring. So, pregabalin as an add on could be a promising drug in patients of trigeminal neuralgia when therapeutic options are limited.

## ACKNOWLEDGEMENTS

The authors would like to thank Dr. Vivek Sharma and Dr. Kiran Bala for their support during the study.

*Funding: No funding sources*

*Conflict of interest: None declared*

*Ethical approval: The study was approved by the Institutional Ethics Committee*

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**Cite this article as:** Narwat A, Sindhu A. Assessment of quality of life in patients with trigeminal neuralgia receiving pregabalin as add-on therapy to carbamazepine. *Int J Res Med Sci* 2026;14:5188-94.