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Fluoroscopy-Guided Platelet-Rich Plasma Infiltration of the Gasser Ganglion for Refractory Trigeminal Neuralgia: A Retrospective Longitudinal Cohort Study

Saucedo-Padilla Juan Manuel[1], Pérez-Limón Susana Gabriela[2], Rosales-Esquivel Paola Ivonne[3], Acosta-Fierro Ana Gabriela[4], Morales-Zapien Jesús Roberto[5]

[1] Specialist in Anesthesiology, Fellowship-trained in Pain Medicine and Palliative Care, FIPP, WIRM Advisory Board, Pain Medical Group Pain and Palliative Care Clinic, Ciudad Juárez, Chihuahua, Mexico

[2] Anesthesiology Resident, Hospital General de Zona No. 35, Instituto Mexicano del Seguro Social (IMSS), Ciudad Juárez, Chihuahua, Mexico.

[3] Specialist in Anesthesiology, Fellowship-trained in Pain Medicine and Palliative Care, Pain Medical Group Pain and Palliative Care Clinic, Ciudad Juárez, Chihuahua, Mexico.

[4] Anesthesiology Resident, Hospital General de Zona No. 35, Instituto Mexicano del Seguro Social (IMSS), Ciudad Juárez, Chihuahua, Mexico.

[5] Specialist in Anesthesiology, Fellowship-trained in Pain Medicine and Palliative Care, Pain Medical Group Pain and Palliative Care Clinic, Ciudad Juárez, Chihuahua, Mexico.

Academic affiliation: Universidad Autónoma de Ciudad Juárez, Ciudad Juárez, Chihuahua, Mexico.

Abstract: ***Background and objective: Refractory trigeminal neuralgia (TN) affects 12-35% of patients, with no response to conventional strategies. Platelet-rich plasma (PRP) has emerged as a promising regenerative therapy owing to its neurotrophic and anti-inflammatory properties. Objective: To evaluate the clinical outcomes of percutaneous PRP infiltration targeting the Gasser ganglion in patients with refractory TN. Materials and methods: Observational, retrospective and longitudinal study. We included 17 patients with TN refractory to conventional treatment (ICHD-3, V2/V3 distribution) who received 1 mL of autologous PRP by percutaneous fluoroscopic approach. The primary endpoints were the Visual Analog Scale (VAS, 0-10) and the neuropathic pain scale (NPS, 0-100), assessed at baseline, 7 days, 1, 3 and 6 months. Pharmacological reduction and adverse events were recorded as secondary outcomes. Comparisons across follow-up were performed using the Wilcoxon signed-rank test. Results: Mean age was 63.5 ± 7.0 years; 88.2% women. VAS decreased from 8.6 ± 1.1 to 1.0 ± 0.9 at six months (88.6%; p = 0.0003), and NPS from 82.4 ± 8.3 to 9.4 ± 9.0 (88.0%; p = 0.0003). Mean analgesic medication requirements decreased by 89.4% ± 9.0 at six months. Conclusions: This retrospective observational cohort demonstrated that fluoroscopy-guided percutaneous platelet-rich plasma infiltration of the Gasser ganglion was associated with progressive and sustained reductions in pain intensity, neuropathic pain, and analgesic requirements over six months without major procedure-related complications. Although limited by its retrospective design and relatively small sample size, these findings support prospective controlled trials to confirm long-term efficacy, safety, and the role of PRP within multimodal treatment strategies for refractory trigeminal neuralgia.***

Keywords: trigeminal neuralgia; platelet-rich plasma; Gasser ganglion; neuropathic pain; regenerative medicine

1. Introduction

Among craniofacial neuropathic pain disorders, trigeminal neuralgia (TN) is one of the most debilitating conditions because of its extreme pain intensity and the challenges associated with long-term management. Historically regarded as one of the most severe pain syndromes affecting humans, TN is characterized by recurrent episodes of unilateral, electric shock-like facial pain with abrupt onset and termination, confined to one or more divisions of the trigeminal nerve and typically triggered by innocuous stimuli involving cutaneous or mucosal trigger zones.[1] According to the third edition of the *International Classification of Headache Disorders* (ICHD-3), TN is classified into three distinct entities with important therapeutic implications: classical TN, caused by neurovascular compression associated with morphological changes of the trigeminal root; idiopathic TN, in which no structural cause can be identified; and secondary TN, resulting from an underlying neurological disorder such as multiple sclerosis or cerebellopontine angle tumors.[2,3]

The prevailing pathophysiological model attributes TN to chronic neurovascular compression at the Obersteiner–Redlich transition zone, a 2–2.5 mm segment where central myelin produced by oligodendrocytes transitions to peripheral myelin formed by Schwann cells. Persistent compression induces focal demyelination, ectopic impulse generation, and ephaptic transmission between low-threshold mechanoreceptive fibers and nociceptive fibers, thereby explaining the hallmark clinical feature of TN: severe paroxysmal pain elicited by otherwise innocuous tactile stimulation.[4] Diffusion tensor imaging studies have demonstrated microstructural abnormalities within the affected trigeminal root that partially normalize following successful microvascular decompression, supporting a

predominantly peripheral mechanism with secondary central sensitization.[4] Sustained ectopic activity subsequently promotes hyperexcitability of trigeminal brainstem nuclei and maladaptive cortical reorganization, allowing pain to persist beyond the initial neurovascular insult.[3]

The reported annual incidence of TN ranges from 4 to 29 cases per 100,000 population, with a female predominance and peak incidence during the fifth and sixth decades of life.[1,5] The true prevalence is likely underestimated because of delayed diagnosis and frequent misclassification as odontogenic or other forms of orofacial pain.[3,5] Beyond pain itself, TN has a profound impact on quality of life, with substantially higher rates of anxiety, depression, functional disability, and social isolation compared with the general population, underscoring the need for comprehensive multidisciplinary management.[5]

Although several therapeutic options are available, important limitations remain. Carbamazepine continues to represent the first-line treatment supported by class I evidence, achieving an initial response in approximately 60–70% of patients; however, its long-term effectiveness is frequently limited by dose-dependent adverse effects and declining efficacy. Oxcarbazepine provides improved tolerability but retains the risk of hyponatremia.[5,6] When pharmacological therapy fails or is poorly tolerated, percutaneous interventions targeting the Gasser ganglion—including radiofrequency thermocoagulation, balloon compression, and glycerol rhizotomy- can achieve pain relief in 70–97% of patients but are associated with recurrence and varying degrees of sensory dysfunction, including hypoesthesia, dysesthesia, and, less commonly, anesthesia dolorosa.[1,4] Microvascular decompression remains the treatment of choice for classical TN associated with high-grade neurovascular conflict, whereas stereotactic radiosurgery offers a less invasive alternative but with delayed onset of analgesia and higher long-term recurrence rates.[1] Consequently, approximately 12–35% of patients continue to experience refractory disease, highlighting the need for regenerative therapeutic strategies with favorable safety profiles.[5]

Conventional therapies primarily aim to interrupt nociceptive transmission rather than restore neural integrity. In contrast, regenerative medicine seeks to modify the biological environment of injured neural tissue, thereby promoting repair and functional recovery. In TN, persistent neurovascular compression triggers a chronic inflammatory response characterized by increased expression of tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6), which contributes to ongoing peripheral and central sensitization. Orthobiologic therapies target these mechanisms by modulating the perineural inflammatory milieu instead of producing neural destruction.[7,8]

Platelet-rich plasma (PRP) is one of the most extensively investigated orthobiologic therapies for peripheral nerve disorders.[9] Following platelet activation, PRP releases multiple bioactive mediators, including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF), nerve growth factor (NGF), and brain-derived neurotrophic factor (BDNF).[9,10] These molecules promote Schwann cell activation, remyelination, axonal regeneration, and attenuation of local inflammatory signaling, thereby creating a biological environment conducive to neural repair.[11,12] Experimental models consistently demonstrate enhanced peripheral nerve regeneration and improved functional recovery following local PRP administration.[13,14]

Fluoroscopy-guided percutaneous delivery of PRP to the Gasser ganglion represents a biologically targeted, non-ablative intervention designed to modulate neuroinflammation while preserving neural integrity. Unlike conventional ablative procedures, this approach aims to promote tissue repair rather than intentionally injure trigeminal fibers. Furthermore, trigeminal sensory neurons exhibit marked responsiveness to neurotrophic stimulation because of their unique embryological origin, providing additional biological rationale for this therapeutic strategy.[1,15] The aim of the present study was to evaluate the effects of fluoroscopy-guided percutaneous PRP infiltration of the Gasser ganglion on pain intensity, neuropathic pain, medication requirements, and procedural safety in patients with refractory trigeminal neuralgia.

2. Materials and methods

2.1 Study design

This retrospective longitudinal observational cohort study was conducted at the Pain Medical Group – Pain and Palliative Care Clinic (Ciudad Juárez, Chihuahua, Mexico) between January and December 2025. Medical records of consecutive patients with refractory trigeminal neuralgia (TN) who underwent fluoroscopy-guided percutaneous platelet-rich plasma (PRP) infiltration as part of routine interventional pain management were reviewed. Only patients meeting all eligibility criteria and with complete clinical records and scheduled follow-up evaluations were included. Seventeen patients (15 women and 2 men), aged 51–73 years, fulfilled the inclusion criteria. The diagnosis of TN was established according to the International Classification of Headache Disorders, 3rd edition (ICHD-3). The manuscript was prepared in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement.

2.2 Selection criteria

Inclusion criteria:

- Diagnosis of classical, idiopathic, or secondary trigeminal neuralgia according to the *International Classification of Headache Disorders, 3rd edition (ICHD-3)*, with a disease duration of 3–10 years.
- Documented refractoriness to first-line pharmacological therapy, defined as persistent pain with a Visual Analog Scale (VAS) score ≥6 despite treatment with carbamazepine ≥800 mg/day or oxcarbazepine ≥1,200 mg/day for at least 8 weeks, or documented intolerance to either medication.
- Pain restricted to the maxillary (V2) and/or mandibular (V3) divisions of the trigeminal nerve, without ophthalmic (V1) involvement.

- Brain magnetic resonance imaging (MRI), including FIESTA sequences, without findings suggestive of alternative structural pathology.
- Complete medical records with documented follow-up at all scheduled evaluation time points.

Exclusion criteria:
- Incomplete medical records or loss to follow-up before the 6-month evaluation.
- Coagulation disorders, thrombocytopenia (<150,000/µL), or current anticoagulant or antiplatelet therapy.
- Active local or systemic infection.
- Active malignancy involving the trigeminal territory without definitive oncological treatment.
- Concomitant interventional procedures involving the trigeminal nerve during the study period.
- Previous invasive procedures for trigeminal neuralgia, including radiofrequency thermocoagulation, balloon compression, glycerol rhizotomy, or microvascular decompression.
- Skull base abnormalities compromising safe percutaneous access to the foramen ovale.

2.3 Obtaining and preparing PRP

PRP was prepared using a standardized protocol and a commercially available centrifugation system. Five days before the procedure, patients received standardized instructions intended to optimize PRP quality, including temporary avoidance of dairy products, caffeine, omeprazole, antibiotics, turmeric, arnica, tobacco, cannabis, and non-steroidal anti-inflammatory drugs whenever clinically appropriate, together with adequate hydration (at least 2 L of water on the day of the procedure).

Five milliliters of peripheral venous blood were collected into tubes containing 3.8% sodium citrate and centrifuged at 1,800 rpm for 8 minutes. The plasma fraction immediately above the erythrocyte layer was carefully harvested while avoiding the leukocyte layer to obtain leukocyte-poor PRP. Platelet concentration was verified by automated platelet count in every patient, confirming a final concentration approximately three to five times higher than baseline values. No exogenous activators were used, allowing physiological and gradual release of platelet-derived growth factors following administration.

2.4 Application technique

All procedures were performed by the authors, board-certified anesthesiologists with fellowship training in Pain Medicine and Interventional Pain Management, using a standardized fluoroscopy-guided technique.

Procedures were carried out in the interventional pain suite under strict aseptic conditions with standard non-invasive monitoring and conscious sedation using fentanyl (50–100 µg) and propofol (100–200 mg), titrated according to body weight, clinical characteristics, and individual patient response.

Under fluoroscopic guidance, a 22-gauge, 117-mm Quincke needle was advanced through the foramen ovale using a percutaneous approach targeting the Gasser ganglion according to the affected trigeminal divisions (V2 or V3). Needle position was confirmed in anteroposterior and lateral fluoroscopic projections before administration of 1 mL of autologous PRP into the perineural space.

Following a four-hour observation period, patients received standardized postoperative recommendations, including temporary nutritional supplementation (omega-3 fatty acids, alpha-lipoic acid, vitamins C and D) for seven days and temporomandibular rehabilitation exercises. Clinical follow-up was scheduled at 7 days and at 1, 3, and 6 months.

2.5 Outcome variables

The primary outcome was pain intensity, assessed using the Visual Analog Scale (VAS; 0–10) and the Neuropathic Pain Scale (NPS; 0–100) at baseline and at 7 days, 1 month, 3 months, and 6 months. Secondary outcomes included the percentage reduction in the total daily analgesic medication dose compared with baseline, the weekly frequency of pain paroxysms, adverse events, and treatment satisfaction, assessed using a five-point Likert scale.

Medication tapering followed a standardized clinical protocol. Analgesic medications were progressively reduced during scheduled follow-up visits only after sustained pain improvement and in the absence of pain recurrence. Dose adjustments were individualized according to each patient's clinical response and were performed by the same pain specialists who conducted the interventional procedures, following a standardized follow-up protocol.

2.6 Statistical analysis

Quantitative variables were described by mean ± standard deviation or median and interquartile range, according to their distribution, while qualitative variables were expressed as absolute frequencies and percentages. The normality of continuous variables was assessed using the Shapiro-Wilk test. Because the primary endpoints did not follow a normal distribution, comparisons between baseline and each of the follow-up time points (7 days, 1 month, 3 months, and 6 months) were performed using the Wilcoxon signed-rank test. All contrasts were bilateral and a value of $p < 0.05$ was considered statistically significant.

2.7 Ethical aspects

The study was in accordance with the Declaration of Helsinki and the provisions of the Regulations of the General Health Law on Health Research in force in Mexico, and was approved by the Ethics and Research Committee of Pain Medical Group Pain Clinic and Palliative Care on May 6, 2026. Because of its retrospective and observational nature, the committee classified the study as risk-free research and waived the requirement for individual informed consent owing to the retrospective observational design and the use of fully anonymized data.

3. Results

A total of 17 patients with refractory trigeminal neuralgia were included in the study, comprising 15 women (88.2%) and 2 men (11.8%), with a mean age of 63.5 ± 7.0 years (range, 51–73 years). At baseline, nine patients (52.9%) were receiving carbamazepine and eight (47.1%) oxcarbazepine. The most frequently prescribed adjuvant medications were duloxetine (14 patients, 82.4%), gabapentin (9 patients, 52.9%), and pregabalin (4 patients, 23.5%). All patients completed the scheduled 6-month follow-up, and no losses to follow-up were recorded.

The mean baseline Visual Analog Scale (VAS) score was 8.6 ± 1.1. No statistically significant reduction was observed at 7 days (8.3 ± 1.2; 3.8% reduction; p = 0.109). Significant improvements became evident at 1 month, with the mean VAS score decreasing to 3.8 ± 0.9 (55.6% reduction; p = 0.0002), followed by further reductions to 1.1 ± 1.0 at 3 months (86.6%; p = 0.0003) and 1.0 ± 0.9 at 6 months (88.6%; p = 0.0003) (Figure 2 and Table 1).

The mean baseline Neuropathic Pain Scale (NPS) score was 82.4 ± 8.3. Minimal changes were observed at 7 days (80.0 ± 11.2; 2.6% reduction; p = 0.317). Significant reductions were observed at 1 month (32.4 ± 13.5; 60.7% reduction; p = 0.0003), 3 months (8.8 ± 9.3; 88.7%; p = 0.0002), and 6 months (9.4 ± 9.0; 88.0%; p = 0.0003), with no evidence of clinically relevant rebound during follow-up (Figure 3 and Table 1).

Analgesic medication requirements remained unchanged during the first week after treatment. Thereafter, medication use progressively declined, with mean reductions of 51.8% ± 16.7 at 1 month (p = 0.0002), 81.8% ± 15.1 at 3 months (p = 0.0003), and 89.4% ± 9.0 at 6 months (p = 0.0002). In several patients, carbamazepine, oxcarbazepine, pregabalin, and duloxetine were substantially reduced or discontinued without pain recurrence (Figure 4).

Regarding clinically meaningful pain relief, 13 of 17 patients (76.5%) achieved at least a 50% reduction in VAS score by the first month. From the third month onward, all patients (17/17, 100%) maintained this level of improvement throughout the 6-month follow-up period (Figure 5).

No major neurological complications, permanent sensory deficits, infectious complications, or systemic adverse events related to the procedure were observed. Reported adverse events were limited to mild discomfort at the puncture site and self-limiting headache during the first 24 hours after the procedure.

4. Discussion

This study demonstrates that fluoroscopy-guided percutaneous platelet-rich plasma (PRP) infiltration of the Gasser ganglion was associated with substantial and sustained improvements in pain intensity, neuropathic pain, and analgesic requirements in patients with refractory trigeminal neuralgia (TN). Clinically meaningful pain relief was achieved by all patients after three months and was maintained throughout the six-month follow-up period, without major procedure-related complications. These findings support the potential role of PRP as a regenerative, non-ablative therapeutic strategy for patients with limited treatment options after failure of conventional pharmacological therapy.

The temporal pattern of clinical improvement provides important insight into the biological mechanisms underlying PRP therapy. The absence of a significant analgesic effect during the first week is consistent with the expected mechanism of action of PRP, which relies on progressive biological modulation rather than immediate neural conduction blockade. Following platelet activation, the gradual release of growth factors—including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF), nerve growth factor (NGF), and brain-derived neurotrophic factor (BDNF)-promotes Schwann cell activation, remyelination, modulation of neuroinflammation, and neural repair.[9,10,12] The marked improvement observed after the first month and its persistence throughout follow-up are consistent with these regenerative mechanisms.

Targeting the Gasser ganglion provides a strong anatomical and pathophysiological rationale. As the primary sensory ganglion of the trigeminal nerve, it represents a critical site where persistent neuroinflammation contributes to neuronal hyperexcitability through increased expression of pro-inflammatory mediators such as TNF-α, IL-1β, and IL-6.[8,17] Delivering PRP directly into the perineural space surrounding the ganglion may therefore modify the local inflammatory microenvironment while preserving neural integrity, in contrast to conventional ablative interventions.[1]

Although direct comparisons were beyond the scope of the present study, the observed clinical outcomes compare favorably with those reported for established percutaneous interventions. Radiofrequency thermocoagulation, balloon compression, and glycerol rhizotomy provide effective analgesia but achieve pain control through controlled neural injury and are associated with varying degrees of sensory dysfunction, including hypoesthesia, dysesthesia, and, less frequently, anesthesia dolorosa.[1,4] In contrast, PRP aims to promote biological repair without intentional neural destruction. Previous experimental and early clinical evidence supports the regenerative potential of PRP in peripheral nerve disorders, providing biological plausibility for the sustained clinical improvements observed in the present cohort.[18]

One of the most clinically relevant findings was the progressive reduction in analgesic medication requirements. By six months, the mean reduction approached 90%, allowing substantial dose reduction or discontinuation of anticonvulsants and adjuvant analgesics in several patients without pain recurrence. This finding is particularly important in older adults, in whom long-term treatment with carbamazepine and related medications is frequently limited by adverse effects such as hyponatremia, dizziness, somnolence, gait instability, and drug interactions.[5,6]

Equally important was the favorable safety profile observed throughout the study. No major neurological complications, permanent sensory deficits, infectious complications, or systemic adverse events were recorded. Reported adverse

events were mild, transient, and self-limiting. Although the sample size was limited, these findings suggest that fluoroscopy-guided PRP infiltration may offer a safer alternative to conventional ablative procedures by preserving sensory function while providing clinically meaningful pain relief.

The present study has several limitations. Its retrospective design and the absence of a control group preclude establishing a causal relationship between PRP administration and the observed clinical outcomes and do not allow the placebo effect to be excluded. The relatively small sample size, derived from a highly selected patient population treated at a single specialized center, limits the generalizability of the findings. Furthermore, patient-reported quality of life and functional outcomes were not assessed using validated instruments. Finally, the six-month follow-up period provides only medium-term outcome data and does not allow conclusions regarding long-term durability or recurrence. Future prospective randomized controlled trials with larger sample sizes and longer follow-up are warranted to confirm these preliminary findings.

5. Conclusion

Fluoroscopy-guided percutaneous platelet-rich plasma (PRP) infiltration of the Gasser ganglion was associated with substantial and sustained reductions in pain intensity, neuropathic pain, and analgesic medication requirements over a six-month follow-up period in patients with refractory trigeminal neuralgia, without major procedure-related complications. Although limited by its retrospective design, single-center setting, and relatively small sample size, this study provides preliminary clinical evidence supporting the potential role of PRP as a regenerative, non-ablative treatment strategy for refractory trigeminal neuralgia. Further prospective randomized controlled trials are warranted to confirm its long-term efficacy, safety, and place within multimodal treatment algorithms.

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Tables

Table 1: Clinical outcomes throughout follow-up. Values are presented as mean ± standard deviation unless otherwise indicated. VAS, Visual Analog Scale; NPS, Neuropathic Pain Scale; SD, standard deviation. Progressive clinical improvement became evident after the first month and remained stable through the six-month follow-up period without evidence of symptom rebound.

Variable	Baseline	7 days	1 month	3 months	6 months
VAS (mean ± SD)	8.6 ± 1.1	8.3 ± 1.2	3.8 ± 0.9	1.1 ± 1.0	1.0 ± 0.9
NPS (mean ± SD)	82.4 ± 8.3	80.0 ± 11.2	32.4 ± 13.5	8.8 ± 9.3	9.4 ± 9.0
Pharmacological reduction (% ± SD)	—	No change	51.8 ± 16.7	81.8 ± 15.1	89.4 ± 9.0
VAS vs. baseline reduction (%)	—	3.8	55.6	86.6	88.6
NPS vs baseline reduction (%)	—	2.6	60.7	88.7	88
Patients ≥50% reduction VAS	—	0/17 (0%)	13/17 (76.5%)	17/17 (100%)	17/17 (100%)

Figures

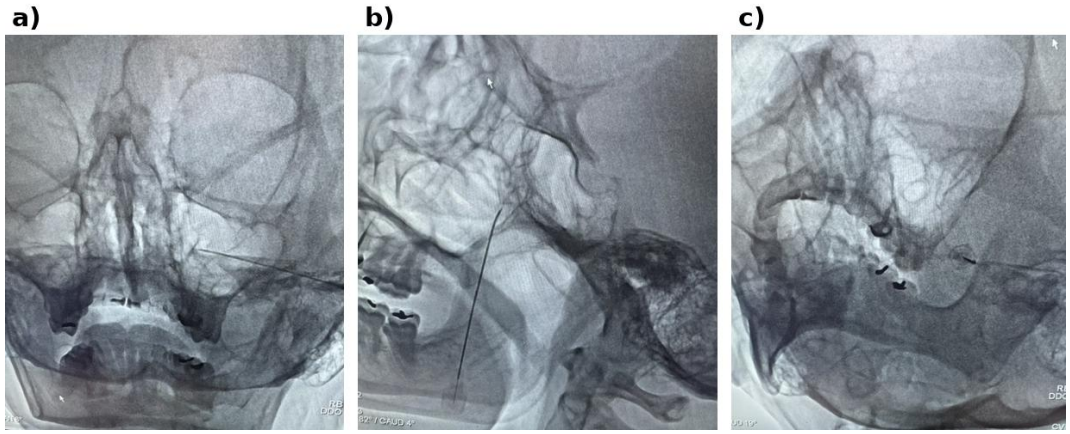

Figure 1: Fluoroscopy-guided percutaneous infiltration of the Gasser ganglion. (A) Anteroposterior fluoroscopic view showing the Quincke needle directed toward the foramen ovale. (B) Lateral fluoroscopic view demonstrating the anatomical relationship between the clivus and the petrous ridge. (C) Final fluoroscopic confirmation of needle placement within the perineural space adjacent to the Gasser ganglion.

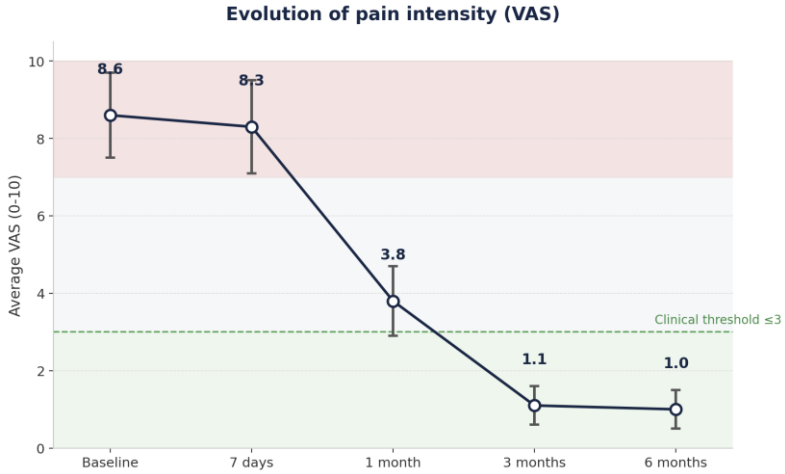

Figure 2: Changes in pain intensity measured using the Visual Analog Scale (VAS) during the six-month follow-up period. The greatest reduction occurred between the first week and the first month, with sustained improvement thereafter. Error bars represent standard deviations. The dashed line indicates the threshold for clinically acceptable pain control (VAS ≤3).

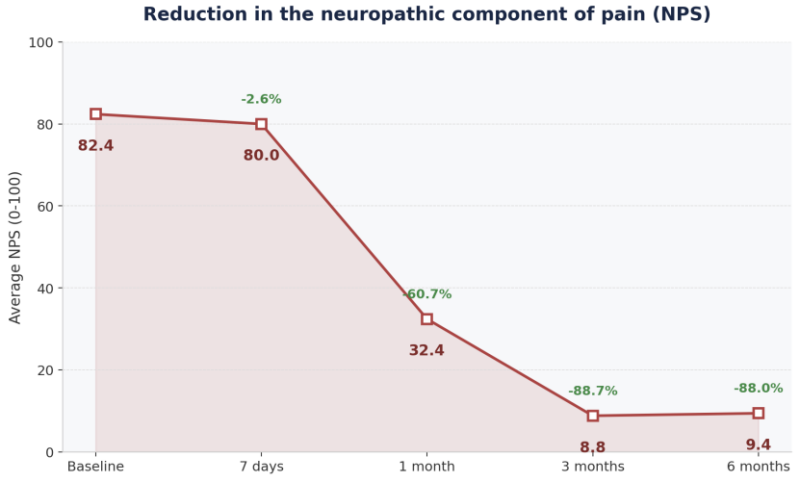

Figure 3: Changes in Neuropathic Pain Scale (NPS) scores during follow-up. Progressive reductions were observed from the first month onward and remained stable through the six-month evaluation. Error bars represent standard deviations.

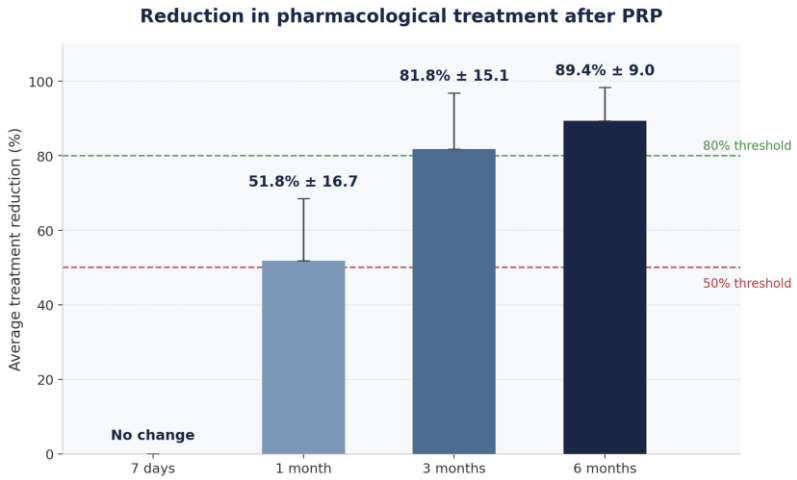

Figure 4: Mean percentage reduction in analgesic medication requirements compared with baseline. Medication use progressively declined throughout follow-up, reaching an average reduction of 89.4% at six months. Dashed lines indicate the 50% and 80% reduction thresholds.

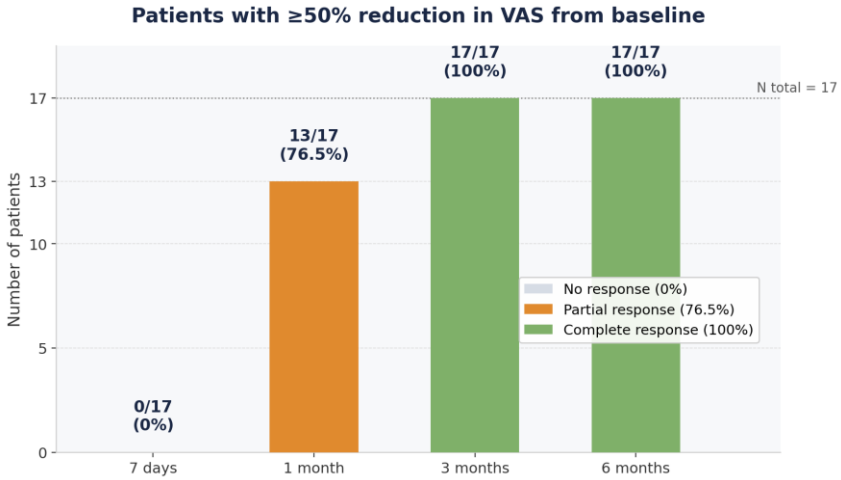

Figure 5: Proportion of patients achieving at least a 50% reduction in Visual Analog Scale (VAS) score. Clinically meaningful pain relief was achieved by 76.5% of patients at one month and by all patients from the third month onward, with sustained improvement through six months.