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Editorial

Dear Colleagues,

We are pleased to announce the publication of the first issue of the third year of The Eurasian Journal of Anesthesiology and Intensive Care (EAJAIC), under the auspices of Medihealth Academy, in February 2026. Entering our third year represents an important milestone for our journal and reflects the growing interest, trust, and scientific engagement of our academic community.

We would like to express our sincere appreciation to all authors, reviewers, and members of the editorial board whose dedicated efforts continue to strengthen the scientific quality and credibility of EAJAIC. We also thank our production and publishing team for their continued professionalism and commitment throughout the publication process.

In this issue, we are proud to present a diverse selection of high-quality manuscripts, including original research articles and case-based contributions that address current challenges and evolving practices in anesthesiology, algology, and intensive care medicine. Our ongoing objective is to provide a reliable platform for the dissemination of innovative research, evidence-based clinical practice, and interdisciplinary collaboration.

As we move forward into our third year, our primary focus remains on enhancing editorial standards, strengthening peer-review quality, increasing international visibility, and achieving inclusion in prominent national and international scientific indexing databases. With your continued support and contributions, we are confident that EAJAIC will further expand its impact within the global anesthesiology and critical care community.

We sincerely thank you for your trust, support, and valuable scientific contributions.

Best regards.

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Original Article

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Ultrasound-guided treatment of cervical facet joint pain: a retrospective comparison of repeated medial branch blocks and pulsed radiofrequency

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ABSTRACT

Aims: Ultrasound-guided cervical medial branch interventions are increasingly used for chronic facet-mediated neck pain, yet comparative data between serial cervical medial branch blocks (CMBB) and pulsed radiofrequency (pRF) under ultrasound guidance are limited. This study compared the short- and medium-term clinical outcomes of repeated ultrasound-guided CMBB with those of single-session ultrasound-guided pRF in patients with chronic cervical facet joint pain.

Methods: In this retrospective cohort, 104 patients with clinically and radiologically confirmed cervical facetogenic pain who exhibited ≥50% temporary pain relief after diagnostic medial branch blocks were analyzed. Patients underwent either four weekly ultrasound-guided CMBBs with 0.25% bupivacaine (n=45) or a single ultrasound-guided pRF procedure targeting the cervical medial branches (n=59). Pain intensity (Numeric Rating Scale, NRS), neck-related disability (Neck Disability Index, NDI), and average daily analgesic tablet intake were assessed at baseline and at 1, 3, and 6 months. Responder status was defined as ≥50% reduction in NRS and/or ≥40% improvement in NDI.

Results: Both CMBB and pRF produced significant within-group improvements in NRS, NDI, and analgesic consumption over 6 months (all $p<0.001$). CMBB yielded faster symptomatic relief, with lower NRS scores at 1 and 3 months (4.16±1.55 vs. 4.69±1.59, $p=0.045$; and 4.44±1.39 vs. 4.36±2.31, $p=0.043$) and higher early NRS responder rates at 1 month (77.8% vs. 50.8%, $p=0.009$). At 6 months, pain, disability, and analgesic use were comparable between groups, and NDI responder rates converged (62.2% vs. 66.1%, $p=0.839$). Hedges' g values indicated small-to-moderate between-group effect sizes favoring CMBB in the early phase (up to −0.65). No significant complications occurred; minor adverse events were infrequent and self-limiting in both groups.

Conclusion: Both repeated ultrasound-guided CMBB and single-session ultrasound-guided pRF are effective, safe, and feasible options for chronic cervical facet joint pain. CMBB provides more rapid, clinically meaningful improvement, whereas pRF offers comparable medium-term outcomes and may be preferable in patients unable to attend repeated procedures. These findings support individualized treatment selection based on clinical profile, comorbidities, and logistical constraints, and highlight the need for prospective randomized trials to confirm and extend these results.

Keywords: Facet joint pain, ultrasonography, interventional, medial branch block, pulsed radiofrequency treatment, neck pain

INTRODUCTION

Chronic pain originating from the cervical facet joints represents a major contributor to persistent neck discomfort, accounting for a significant proportion of chronic neck pain cases and exerting a considerable socioeconomic burden worldwide.[1] The facet joints receive sensory input from the medial branches of the dorsal rami, which contribute

critically to segmental spinal stability; therefore, they have become important targets in interventional pain treatment strategies.

Among available modalities, conventional radiofrequency (RF) neurotomy is recognized for producing prolonged pain relief by denervating nociceptive medial branches. However, this method can be technically demanding, often necessitating multilevel treatment and carrying a risk of post-procedural neuritis or dysesthesia.[2,3]

In contrast, pulsed radiofrequency (pRF) has been introduced as a minimally invasive alternative that delivers intermittent bursts of high-voltage current at subdestructive temperatures. Rather than ablating neural tissue, pRF modulates nociceptive transmission through controlled electric fields, resulting in reversible changes in neuronal signaling and dorsal horn activity. Previous investigations have confirmed its favorable safety profile and therapeutic efficacy in treating lumbar, thoracic, and cervical facet-related pain syndromes.[4-8] Although the duration of symptom control is often shorter than that achieved with conventional RF, its lower complication rate and improved procedural tolerance make it a valuable clinical option.

Historically, cervical medial branch procedures have been performed under fluoroscopic guidance, which ensures accurate needle localization but exposes both patient and operator to ionizing radiation. Fluoroscopy also has limitations in soft-tissue visualization and typically requires more time and equipment. The advent of ultrasound (US) guidance has addressed many of these drawbacks. US allows real-time imaging of both vascular and neural structures, eliminates radiation exposure, and facilitates bedside applications. Several comparative studies have shown that US-guided cervical medial branch blocks (CMBB) yield analgesic outcomes comparable to those achieved with fluoroscopy, confirming US as a safe and reliable alternative.[9-13]

Despite the growing use of US guidance in cervical medial branch interventions, direct comparative data evaluating US-guided CMBB and US-guided pRF remain absent from the literature.

Although both modalities have been widely investigated as individual treatment options for facet-mediated neck pain, existing studies have predominantly assessed them separately or under fluoroscopic guidance, leaving a significant gap regarding their relative clinical value when performed with US. To address this unmet need, the present study aimed to compare the short- and medium-term therapeutic outcomes of repeated US-guided CMBB and single-session US-guided pRF in patients with cervical facetogenic pain.

We hypothesized that repeated US-guided CMBB would yield more rapid symptom reduction due to their immediate anesthetic effect, whereas US-guided pRF would provide comparable mid-term improvement through neuromodulatory mechanisms. By delineating the temporal patterns of analgesic and functional recovery associated with these two techniques, this study seeks to generate clinically relevant evidence to inform individualized treatment planning in interventional pain practice.

The primary outcome of the study was pain intensity, assessed using the Numeric Rating Scale (NRS). Secondary outcomes included neck-related disability measured by the Neck Disability Index (NDI), changes in daily analgesic consumption, and responder rates based on predefined clinically meaningful thresholds.

METHODS

Ethics

The study protocol was approved by the Clinical Researches Ethics Committee of Antalya Training and Research Hospital (Date: 10.10.2024, Decision No: 15/13), and all procedures adhered to the principles of the Declaration of Helsinki. A formal sample size calculation was not performed, and all eligible patients within the study period were included.

Study Design and Setting

This retrospective, observational study was conducted at the Department of Pain Medicine, Antalya Training and Research Hospital. All interventions performed between June 2023 and June 2025 were retrospectively reviewed from medical records and had been carried out by a single experienced pain specialist with more than seven years of procedural experience, ensuring procedural consistency and minimizing operator-related variability.

Patient Enrollment

A total of 126 patients were screened for eligibility. Twenty-two individuals were excluded due to incomplete treatment sessions or loss to follow-up. The remaining 104 patients constituted the final study cohort, of whom 59 underwent US-guided pRF, and 45 received repeated US-guided CMBB procedures (Figure 1).

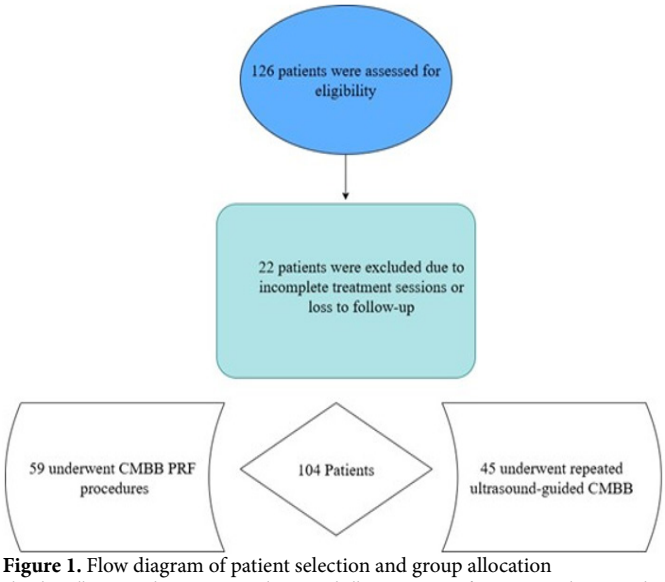

Figure 1. Flow diagram of patient selection and group allocation
Flowchart illustrating the screening, exclusion, and allocation process for patients undergoing either repeated ultrasound-guided CMBB or pRF treatment

Eligibility criteria included adults aged 18 to 80 years with at least 3 months of localized neck pain at the C3 to C6 levels, a baseline NRS of 4 or higher, and a positive response to diagnostic medial branch blocks, defined as at least 50% temporary pain relief at two or more levels. Exclusion criteria were pregnancy, psychiatric disorders, neurologic deficits,

infection, prior cervical spine surgery, and any interventional treatment within the preceding six months. Patients who did not attend follow-up visits or did not complete the planned intervention were also excluded (Figure 2).

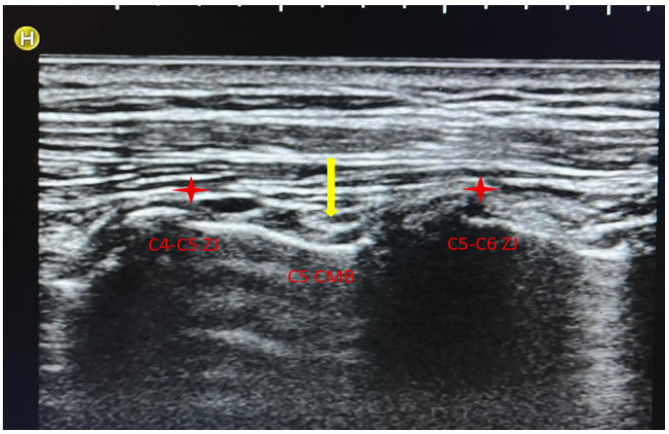

Figure 2. Sonographic localization of the C5 cervical medial branch in the sagittal plane
Sagittal ultrasonographic image showing the anatomical location of the cervical medial branch (yellow arrow) at the C5 level between two adjacent facet joints (asterisks)

Treatment allocation was based on real-world clinical considerations rather than randomization. Patients who were unable to attend repeated weekly sessions, had relative contraindications to repeated injections (e.g., continuous anticoagulant therapy), or had significant mobility or access limitations were preferentially treated with single-session pRF. Patients without these constraints were offered repeated CMBB.

Clinical Workflow

Diagnosis and follow-up were jointly carried out by an orthopedic spine surgeon and a pain physician to ensure diagnostic consistency. All interventions were performed in an outpatient setting. Standard pre-procedural evaluation was completed for each patient, including peripheral intravenous access. No sedation was administered.

Ultrasound Technique

A standardized sagittal out-of-plane US approach was used for both CMBB and pRF procedures. Patients were positioned prone, and a linear transducer operating at 10 to 15 MHz was aligned longitudinally over the paraspinal region to visualize the articular pillar. Under real-time imaging, the needle was advanced toward the medial branch without repositioning the probe, which was particularly advantageous in patients with restricted cervical mobility (Figure 3).

Diagnostic blocks were performed at a minimum of two cervical levels using 1 ml of 2% lidocaine per level. A reduction of at least 50 percent in pain within one hour confirmed facetogenic pain.

Treatment Protocols

CMBB group: Therapeutic injections were administered once weekly for four consecutive sessions using a 22 gauge, 10 cm insulated needle. Correct needle placement was confirmed using sensory stimulation at 50 Hz below 0.5 V and motor stimulation at 2 Hz. After confirmation, 1 ml of 0.25 percent bupivacaine was delivered at each targeted medial branch level. Corticosteroids were not used.

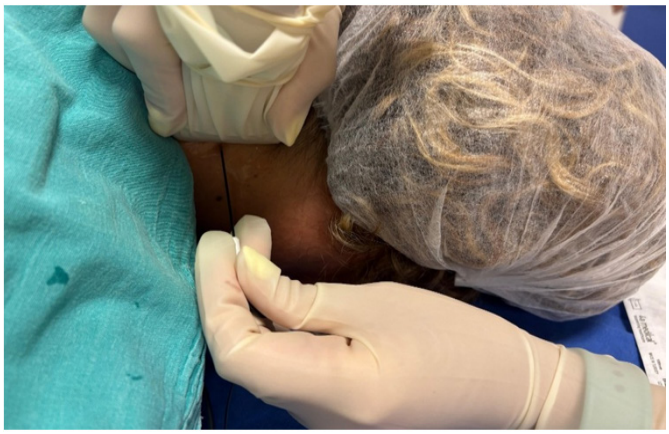

Figure 3. Patient positioning and ultrasound probe placement during cervical medial branch block or pulsed radiofrequency procedure
Patient in prone position with slight cervical flexion. Ultrasound probe positioned longitudinally over the posterolateral neck to identify the articular pillars. Needle insertion performed via an out-of-plane, anterior-to-posterior approach

pRF group: Patients who could not attend repeated sessions or had contraindications to serial injections, such as continuous anticoagulant therapy, severe mobility limitations, or limited access to follow-up visits, underwent single-session pRF one week after diagnostic blocks. Using the same US-guided approach, a 22-gauge, 10 cm RF cannula with a 5 mm active tip was positioned at the medial branch. Positioning was verified by sensory and motor stimulation, and pRF was applied at 65 V and 42 °C for two 120-second cycles. All pRF procedures were performed using identical settings.

Post-Procedure Care

Minor complications occurring within 24 hours were documented through standardized nursing forms. All patients received structured education on daily isometric cervical exercises by a dedicated pain nurse. Exercise adherence was encouraged but not objectively monitored.

Statistical Analysis

Data were analyzed using SPSS version 25 (IBM, Armonk, NY, USA). Quantitative variables were expressed as mean±standard deviation, and categorical variables as frequencies and percentages. Normality of distribution was assessed using the Shapiro–Wilk test. As most continuous variables were non-normally distributed, nonparametric tests were primarily used.

Baseline comparisons between groups used Independent samples t-tests or Mann–Whitney U-tests, as appropriate. Categorical variables were evaluated using Chi-square or Fisher's exact tests. Longitudinal changes in NRS, NDI, and analgesic consumption were assessed using Friedman tests with Bonferroni-adjusted Dunn post hoc analyses. Between-group comparisons at each follow-up were performed using Mann–Whitney U tests. Responder rates were compared using Chi-square or Fisher's exact tests.

Correlations between pain and disability were evaluated using Spearman's rank correlation. Between-group effect sizes were quantified using Hedges' g with 95 percent confidence intervals. Statistical significance was defined as a two-tailed p-value less than 0.05. No imputation for missing data was required because all included patients completed the follow-up assessments.

RESULTS

A total of 104 patients were included in the analysis, comprising 59 individuals in the pRF group and 45 in the CMBB group. Baseline demographic and clinical variables were comparable between groups, with no significant differences in age, sex distribution, or baseline NRS and NDI scores (Table 1).

Pain scores (NRS) improved significantly over time in both treatment arms (p<0.001). In the CMBB group, NRS decreased from 7.89±0.71 at baseline to 4.16±1.55, 4.44±1.39, and 4.78±1.72 at one, three, and six months. Similar reductions were observed in the pRF group, with scores declining from 7.66±0.86 to 4.69±1.59, 4.36±2.31, and 4.64±2.13. Between-group comparisons revealed lower NRS values in the CMBB group at one and three months (p=0.045 and p=0.043), although no significant difference remained at six months. Early NRS responder rates were higher with CMBB at one month (77.8 percent versus 50.8 percent, p=0.009), while responder proportions converged at three and six months (Table 1).

Neck-related disability, measured by the NDI, also improved significantly within both groups (p<0.001). CMBB reduced NDI from 23.73±5.01 to 12.44±5.09 at one month, 12.09±6.09 at three months, and 13.36±5.84 at six months. The pRF group showed comparable improvement from 24.78±4.53 to 15.78±5.16, 12.42±5.45, and 12.83±5.60 across the same time points. Between-group analysis demonstrated better NDI scores with CMBB at one month (p=0.001), but no differences at later follow-up. Responder rates based on a 40 percent NDI reduction were similar between groups throughout follow-up (Table 1).

Pain and disability were positively correlated at baseline and throughout follow-up in both groups. In the CMBB group, Spearman's rho ranged from 0.50 to 0.70 (all p<0.001). Correlations were stronger in the pRF group, with coefficients ranging from 0.61 to 0.77 (all p<0.001) (Table 1).

Analgesic consumption declined significantly in both groups over six months (p<0.001). CMBB reduced daily tablet use from 19.24±4.02 to 10.44±5.51, 9.87±5.94, and 10.53±5.66. The pRF group demonstrated a similar trend, decreasing from 19.80±3.31 to 11.59±5.60, 9.32±6.43, and 9.78±6.21. No significant between-group differences were detected at any follow-up interval. CMBB showed a higher proportion of early analgesic-use responders at one month (p=0.034), but responder rates were comparable thereafter (Figure 4, Table 2).

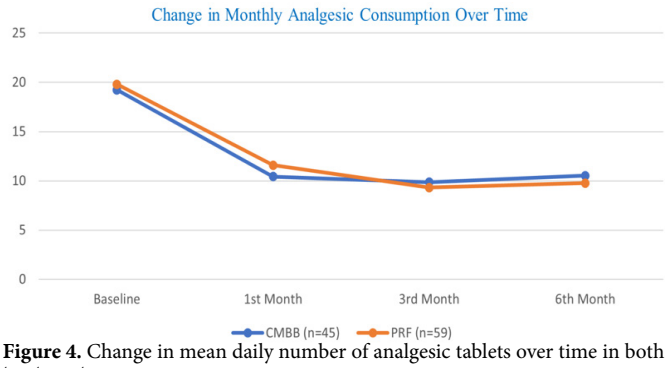

Figure 4. Change in mean daily number of analgesic tablets over time in both treatment groups

Both CMBB and pRF groups showed significant reductions in analgesic consumption from baseline to 6 months (p<0.001, Friedman test). The CMBB group demonstrated an earlier reduction at 1 month (p=0.034, Chi-square test).

No major procedure-related adverse events occurred. Minor events were reported in 14.4 percent of patients, consisting of vascular puncture in 5.8 percent and transient post-procedural discomfort in 8.7 percent. Complication rates did not differ significantly between groups, and all events resolved within 24 to 48 hours (Table 2).

DISCUSSION

To the best of our knowledge, direct head-to-head comparisons between repeated US-guided CMBB and US-guided pRF for the management of chronic facetogenic neck pain remain limited in the existing literature, particularly with respect to medium-term clinical outcomes and real-

Table 1. Pain and disability outcomes following CMBB and pRF treatments

Outcome measure	CMBB (n=45)	pRF (n=59)	p-value	Hedges g (95% CI)
NRS score (baseline)	7.89±0.71	7.66±0.86	0.237	NA
NRS score (1 month)	4.16±1.55	4.69±1.59	0.045	-0.33 (-0.68 to-0.02)
NRS score (3 months)	4.44±1.39	4.36±2.31	0.043	-0.21 (-0.52 to 0.11)
NRS score (6 months)	4.78±1.72	4.64±2.13	0.070	-0.15 (-0.45 to 0.16)
≥50% NRS reduction (1 month)	77.8% (35/45)	50.8% (30/59)	0.009	NA
≥50% NRS reduction (3 months)	75.6% (34/45)	72.9% (43/59)	0.934	NA
≥50% NRS reduction (6 months)	60.0% (27/45)	64.4% (38/59)	0.798	NA
NDI score (baseline)	23.73±5.01	24.78±4.53	0.224	NA
NDI score (1 month)	12.44±5.09	15.78±5.16	0.001	-0.65 (-1.05 to -0.25)
NDI score (3 months)	12.09±6.09	12.42±5.45	0.560	-0.12 (-0.42 to 0.17)
NDI score (6 months)	13.36±5.84	12.83±5.60	0.567	-0.09 (-0.38 to 0.20)
≥40% NDI reduction (1 month)	66.7% (30/45)	55.9% (33/59)	0.364	NA
≥40% NDI reduction (3 months)	68.9% (31/45)	69.5% (41/59)	1.000	NA
≥40% NDI reduction (6 months)	62.2% (28/45)	66.1% (39/59)	0.839	NA

Values are presented as mean±SD or % (n/N). NRS: Numeric Rating Scale, NDI: Neck Disability Index, CMBB: Cervical medial branch block, pRF: Pulsed radiofrequency

Table 2. Analgesic consumption and adverse events following CMBB and pRF treatments

Outcome measure	CMBB (n=45)	pRF (n=59)	p-value
Mean daily analgesic tablet use (baseline)	19.24±4.02	19.80±3.31	0.574
Mean daily analgesic tablet use (1 month)	10.44±5.51	11.59±5.60	0.304
Mean daily analgesic tablet use (3 months)	9.87±5.94	9.32±6.43	0.249
Mean daily analgesic tablet use (6 months)	10.53±5.66	9.78±6.21	0.274
≥50% reduction in analgesic use (1 month)	73.3% (33/45)	50.8% (30/59)	0.034
≥50% reduction in analgesic use (3 months)	77.8% (35/45)	71.2% (42/59)	0.472
≥50% reduction in analgesic use (6 months)	66.7% (30/45)	66.1% (39/59)	0.955
Any minor adverse event	15.6% (7/45)	13.6% (8/59)	0.785
Vascular puncture	6.7% (3/45)	5.1% (3/59)	0.739
Transient post-procedural discomfort	8.9% (4/45)	8.5% (5/59)	0.953

Data are presented as mean±SD or % (n/N). CMBB: Cervical medial branch block; pRF: Pulsed radiofrequency.

world outpatient practice settings. The findings of this study demonstrate that both interventions substantially alleviate pain, improve functional capacity, and reduce analgesic requirements over a six-month follow-up period.

Although both techniques were clinically effective, the CMBB group experienced faster symptomatic improvement, particularly during the first month. This early benefit likely reflects the immediate interruption of nociceptive input by local anesthetic administration. In contrast, pRF exerts its effect through delayed neuromodulatory mechanisms that gradually stabilize dorsal horn excitability. Such temporal divergence between rapid but transient block effects and slower, sustained pRF outcomes is consistent with previous neurophysiological observations in spinal pain management.[15-19] This distinction underscores the fundamentally different mechanisms of action underlying these two interventions and provides a physiological explanation for the observed temporal differences in clinical response.

Effect size analyses indicated that between-group differences were small to moderate, suggesting that while CMBB accelerated early recovery, the overall magnitude of difference was modest and mainly relevant in the short term. The convergence of outcomes at medium-term follow-up supports the possibility that these US-guided interventions may play complementary or interchangeable roles in individualized treatment planning. From an evidence-based perspective, this finding suggests that treatment selection may reasonably be guided by patient-specific priorities such as the need for rapid symptom relief versus procedural convenience.

In addition to statistical significance, improvements should be interpreted relative to established minimal clinically important difference thresholds. An NRS reduction of approximately 2 points and an NDI improvement of 7 to 10 points are generally considered meaningful in cervical spine pain. Both CMBB and pRF exceeded these benchmarks across all follow-up intervals, confirming the clinical relevance of the observed improvements. The early advantage of CMBB likewise surpassed MCID criteria, reinforcing the mechanistic distinction between immediate anesthetic effects and delayed neuromodulation. This observation strengthens the argument that the early superiority of CMBB is not merely statistically detectable but also clinically meaningful.

The stronger correlation between pain intensity and disability observed in the pRF group suggests that neuromodulatory interventions may exert broader central effects on pain processing and functional restoration than peripheral block techniques alone. This observation aligns with the hypothesis that pRF influences both peripheral nociceptors and central sensitization, thereby contributing to more integrated improvements in pain–function. Such an effect may be particularly relevant in patients with features of central sensitization or long-standing chronic pain.

The earlier functional gains in the CMBB group during the first three months are consistent with evidence indicating that local anesthetic-based medial branch blocks can improve cervical mobility and normalize muscle activation patterns.[20] The greater early reduction in analgesic consumption observed in this group further supports its clinical utility, particularly in older patients or those with polypharmacy concerns, in whom minimizing systemic medication exposure is desirable. In this context, CMBB may serve as an effective strategy for short-term functional restoration and medication de-escalation.

Although direct head-to-head US-guided comparisons are lacking, Manchikanti et al.[21] previously evaluated these modalities under fluoroscopic guidance. However, their study employed conventional RF at 80 °C with a tissue-destructive intent, which differs fundamentally from the neuromodulatory mechanism of pRF. Additionally, whereas their protocol distributed repeated blocks over a one-year period, the present study implemented a condensed four-week CMBB schedule. This pragmatic design more closely reflects real-world outpatient practice and may enhance treatment adherence while reducing attrition and procedural burden.

Importantly, no corticosteroids or adjunct anesthetics were used in the pRF arm of the present study, allowing a more accurate assessment of the intrinsic neuromodulatory effects of pRF. This methodological choice strengthens causal interpretation and aligns with emerging evidence demonstrating that pRF alone can provide sustained analgesia without pharmacologic confounding.[22,23] This feature represents a methodological strength of the current study compared with prior mixed-intervention protocols.

From a pragmatic standpoint, treatment allocation reflected common clinical constraints, including anticoagulant therapy, mobility limitations, and difficulty attending repeated appointments. In such scenarios, single-session pRF represented a feasible alternative, whereas CMBB remained appropriate for patients without contraindications

to repeated procedures. These findings support a patient-centered, individualized approach rather than a one-size-fits-all treatment algorithm.

Beyond clinical efficacy, US guidance offers several procedural advantages. The elimination of ionizing radiation enhances long-term safety for both patients and clinicians, while real-time visualization of vascular and neural structures may reduce procedural risk. The out-of-plane sagittal technique used in this study proved time-efficient and allowed multilevel access without frequent probe repositioning, particularly benefiting patients with short necks or limited cervical mobility.

These technical efficiencies carry important economic implications. Reducing the number of visits, avoiding fluoroscopy, and shortening procedural duration may substantially lower healthcare costs, making US-guided interventions especially suitable for outpatient pain practices.[24,25] Furthermore, recent studies have confirmed the accuracy and clinical utility of US-guided pRF, demonstrating outcomes comparable to fluoroscopic approaches.[26-28] The present study extends this growing body of evidence by providing the first medium-term, US-guided comparison of CMBB and pRF within the same clinical framework.

Limitations

This study has several limitations. First, its retrospective and non-randomized design introduces inherent selection bias, as treatment allocation reflected clinical and logistical considerations rather than random assignment. Second, although baseline characteristics were comparable between groups, unmeasured confounders, including chronicity of pain, psychosocial status, and adherence to exercise recommendations, may have influenced treatment response. Third, the six-month follow-up period limits the ability to evaluate long-term durability. Finally, outcomes beyond pain, disability, and analgesic use, such as quality-of-life indices or objective functional assessments, were not examined. These limitations should be considered when interpreting the findings, and prospective randomized studies with longer follow-up are needed to validate and extend these results.

All data were reviewed by an independent spine surgeon blinded to treatment allocation, which reduced the potential for observer bias. While patients were advised to perform isometric cervical exercises, adherence was not objectively verified. Future prospective, multicenter, randomized studies incorporating exercise monitoring or wearable sensors may help elucidate the interaction between neuromodulation, muscular reactivation, and functional recovery.

In addition, existing studies on US-guided medial branch pRF have several limitations that should be acknowledged. Most reports involve small sample sizes, heterogeneous patient populations, or short follow-up periods, and many lack direct comparisons with alternative interventional techniques. Variability in pRF parameters and target localization further limits cross-study comparability. These gaps in the literature highlight the need for standardized protocols and comparative designs, which the present study partially addresses by evaluating two US-guided modalities within the same clinical framework.

CONCLUSION

As a result, both repeated US-guided CMBB and single-session US-guided pRF demonstrated significant and clinically meaningful improvements in pain, disability, and analgesic consumption over a six-month follow-up period. CMBB provided faster early-phase symptom relief, likely reflecting the immediate interruption of nociceptive input by local anesthetics, whereas pRF achieved comparable medium-term outcomes through sustained neuromodulatory effects. The favorable safety profile and absence of major complications in both groups support the feasibility of US-guided approaches as practical and well-tolerated interventions for chronic cervical facetogenic pain. Given the non-randomized treatment allocation and potential for unmeasured confounding, these results should be interpreted cautiously. Prospective randomized trials with longer follow-up and broader functional outcome measures are needed to establish comparative effectiveness and inform optimal patient selection strategies more definitively.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study protocol was approved by the Clinical Researches Ethics Committee of Antalya Training and Research Hospital (Date: 10.10.2024, Decision No: 15/13).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: Ç.A., G.Ç.; Methodology: Ç.A., G.Ç.; Data Collection and Investigation: G.Ç.; Formal Analysis: C.A., Ç.A.; Writing–Original Draft: Ç.A.; Writing–Review and Editing: Ç.A., G.Ç., C.A.; Supervision: C.A.; Critical Review: All authors.

REFERENCES

1. Manchikanti L, Singh V, Rivera J, Pampati V. Prevalence of cervical facet joint pain in chronic neck pain. *Pain Physician*. 2002;5(3):243-249. Erratum in: *Pain Physician*. 2002;5(4):445.
2. Groen GJ, Baljet B, Drukker J. The innervation of the spinal dura mater: anatomy and clinical implications. *Acta Neurochir (Wien).* 1988;92(1-4):39-46. doi:10.1007/BF01401971. PMID: 3407473
3. Tekin I, Mirzai H, Ok G, Erbuyun K, Vatansever D. A comparison of conventional and pulsed radiofrequency denervation in the treatment of chronic facet joint pain. *Clin J Pain*. 2007;23(6):524-529. doi:10.1097/AJP.0b013e318074c99d

4. Kroll HR, Kim D, Danic MJ, Sankey SS, Gariwala M, Brown M. A randomized, double-blind, prospective study comparing the efficacy of continuous versus pulsed radiofrequency in the treatment of lumbar facet syndrome. *J Clin Anesth*. 2008;20(7):534-537. doi:10.1016/j.jclinane.2008.05.013
5. Chang MC. Effect of pulsed radiofrequency treatment on the thoracic medial branch for managing chronic thoracic facet joint pain refractory to medial branch block with local anesthetics. *World Neurosurg.* 2018;111:e644-8. doi:10.1016/j.wneu.2017.12.141
6. Mikeladze G, Espinal R, Finnegan R, Routon J, Martin D. Pulsed radiofrequency application in treatment of chronic zygapophyseal joint pain. *Spine J.* 2003;3(5):360-362. doi:10.1016/s1529-9430(03)00065-2
7. Liliang PC, Lu K, Hsieh CH, Kao CY, Wang KW, Chen HJ. Pulsed radiofrequency of cervical medial branches for treatment of whiplash-related cervical zygapophysial joint pain. *Surg Neurol.* 2008;70(Suppl 1):S1:50-55. doi:10.1016/j.surneu.2008.07.006
8. Chang MC, Yang S. Effectiveness of pulsed radiofrequency on the medial cervical branches for cervical facet joint pain. *World J Clin Cases.* 2022;10(22):7720-7727. doi:10.12998/wjcc.v10.i22.7720
9. Chen CK, Wong CC, Teo YY, Phui VE. Single entry posterior parasagittal approach radiofrequency neurotomy of cervical medial branch: a feasible alternative to conventional approaches in the treatment of cervical facet pain. *Int J Spine Surg.* 2021;15(6):1192-200. doi:10.14444/8151
10. Han SH, Park KD, Cho KR, Park Y. Ultrasound versus fluoroscopy-guided medial branch block for the treatment of lower lumbar facet joint pain: a retrospective comparative study. *Medicine (Baltimore).* 2017;96(16):e6655. doi:10.1097/MD.0000000000006655
11. Park KD, Lim DJ, Lee WY, Ahn J, Park Y. Ultrasound versus fluoroscopy guided cervical medial branch block for the treatment of chronic cervical facet joint pain: a retrospective comparative study. *Skeletal Radiol*. 2017;46(1):81-91. doi:10.1007/s00256-016-2516-2
12. Finlayson RJ, Etheridge JP, Tiyaprasertkul W, Nelems B, Tran DQ. A randomized comparison between ultrasound- and fluoroscopy-guided C7 medial branch block. *Reg Anesth Pain Med.* 2015;40(1):52-57. doi:10.1097/AAP.0000000000000186
13. Siegenthaler A, Mlekusch S, Trelle S, Schliessbach J, Curatolo M, Eichenberger U. Sonographically guided cervical facet nerve and joint injections: why sonography? *J Ultrasound Med*. 2013;32(11):1885-1892. doi:10.7863/ultra.32.11.1885
14. Bogduk N, Dreyfuss P, Govind J. A narrative review of lumbar medial branch neurotomy for the treatment of back pain. *Pain Med*. 2009;10(6):1035-1045. doi:10.1111/j.1526-4637.2009.00692.x
15. Van Zundert J, Patijn J, Kessels A, Lamé I, van Suijlekom H, van Kleef M. Pulsed radiofrequency adjacent to the cervical dorsal root ganglion in chronic cervical radicular pain: a double blind sham controlled randomized clinical trial. *Pain*. 2007;127(1-2):173-182. doi:10.1016/j.pain.2006.09.002
16. Bogduk N. Pulsed radiofrequency. *Pain Med.* 2006;7(5):396-407. doi:10.1111/j.1526-4637.2006.00210.x
17. Byrd D, Mackey S. Pulsed radiofrequency for chronic pain. *Curr Pain Headache Rep.* 2008;12(1):37-41. doi:10.1007/s11916-008-0010-3
18. Choi GS, Ahn SH, Cho YW, Lee DG. Long-term effect of pulsed radiofrequency on chronic cervical radicular pain refractory to repeated transforaminal epidural steroid injections. *Pain Med*. 2012;13(3):368-375. doi:10.1111/j.1526-4637.2012.01333.x
19. Malec-Milewska M, Buczek M, Sęk M. Pulsed radiofrequency for the treatment of cervical and lumbar facet joint pain. *Neurol Neurochir Pol.* 2021;55(1):70-77. doi:10.5603/PJNNS.a2020.0080
20. Manchikanti L, Hirsch JA, Falco FJ, et al. Management of lumbar facet joint pain: a systematic review of the evidence. *Pain Physician*. 2015;18(6):E535-82.
21. Manchikanti L, Kosanovic R, Pampati V, Sanapati MR, Hirsch JA. Outcomes of cervical therapeutic medial branch blocks and radiofrequency neurotomy: clinical outcomes and cost utility are equivalent. *Pain Physician*. 2022;25(1):35-47.
22. Galiano K, Obwegeser AA, Walch C, et al. Ultrasound-guided facet nerve block in the cervical spine: a computed tomography-controlled feasibility study. *Reg Anesth Pain Med.* 2005;30(6):552-556. doi:10.1016/j.rapm.2005.07.005
23. Bodor M, Murthy N, Uribe Y. Ultrasound-guided cervical facet joint injections. *Spine J.* 2022;22(6):983-992. doi:10.1016/j.spinee.2022.01.011
24. Kietaibl S, Ferrandis R, Godier A, et al. Regional anaesthesia in patients on antithrombotic drugs: joint ESAIC/ESRA guidelines. *Eur J Anaesthesiol.* 2022;39(2):100-132. doi:10.1097/EJA.0000000000001600
25. Moster M, Bolliger D. Perioperative guidelines on antiplatelet and anticoagulant agents: 2022 update. *Curr Anesthesiol Rep.* 2022;12(3):286-296. doi:10.1007/s40140-021-00511-3
26. Alsaeid MA, Algyar MF, Mahmoud AM, Farghaly OS, Salah AG, Raouf MM. Ultrasound-guided high-voltage pulsed radiofrequency versus standard-voltage pulsed radiofrequency in refractory chronic cervical radicular pain randomized clinical trial. *J Anesth.* 2025. doi:10.1007/s00540-025-03535-5
27. Lai J, Wu Y, Xiang Y, Wang Y. A novel transverse ultrasound-guided approach for puncturing the cervical dorsal root ganglion at the level of articular pillar for radiofrequency treatment. *J Pain Res.* 2024;17:3473-3485. doi:10.2147/JPR.S479986
28. Chalermkitpanit P, Pannangpetch P, Kositworakitkun Y, et al. Ultrasound-guided pulsed radiofrequency of cervical nerve root for cervical radicular pain: a prospective randomized controlled trial. *Spine J.* 2023;23(5):651-655. doi:10.1016/j.spinee.2023.01.004

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Effects of dexmedetomidine on bupivacaine-induced myotoxicity and inflammation in rats

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ABSTRACT

Aims: The increasing use of regional anesthesia techniques has heightened concern regarding local anesthesia-related tissue damage. Bupivacaine is widely used because of its long duration of action and high potency, however it is associated with a high myotoxic potential. This study aims to evaluate whether the addition of dexmedetomidine as an adjuvant to bupivacaine during peripheral block may influence bupivacaine-induced myotoxicity and inflammation.

Methods: Twenty-eight Wistar albino rats were allocated into four equal groups. Group C received 0.2 ml of 0.9% normal saline, group B received 0.2 ml of 0.25% bupivacaine, group D received 0.2 ml of dexmedetomidine at a concentration of 0.005%, and group BD received a combination of 0.25% bupivacaine and 0.005% dexmedetomidine. All injections were administered intramuscularly into the right gastrocnemius muscle. After 48 hours, muscle tissues were harvested for histopathological evaluation using the Modified Benoit Score (MBS). Serum creatine phosphokinase (CPK) and C-reactive protein (CRP) levels were measured.

Results: No rats were lost during the study, and all animals completed the experimental protocol. Histopathological evaluation revealed that group B exhibited the highest degree of muscle damage, as assessed by the MBS, with scores significantly higher than those of all other groups ($p<0.05$). MBS values in group BD were significantly lower than those in group B but remained higher than those observed in groups C and D ($p<0.05$). CPK levels were significantly elevated in group B compared with groups C and D ($p<0.05$), whereas no significant differences were observed among the remaining groups. In contrast, CRP levels did not differ significantly between the groups ($p>0.05$).

Conclusion: Dexmedetomidine was associated with a reduction in bupivacaine-induced myotoxicity in rats, as supported by histopathological and biochemical findings. These results suggest that dexmedetomidine may have the potential to reduce muscle injury when used as an adjuvant to local anesthetics, although further experimental and clinical studies are required.

Keywords: Bupivacaine, dexmedetomidine, myotoxicity

INTRODUCTION

Peripheric nerve blocks, peripheric nerve catheters, plane blocks are applied for intraoperative anesthesia and postoperative pain control. These techniques significantly reduce the use of additional analgesics and opioids, as well as shortening the postoperative hospital stay and recovery time. The increasing popularity of them has also increased interest in tissue damage induced by local anesthetics.[1]

Bupivacaine is the most preferred agent due to its long duration of action and high potency, but it is the agent with the highest cardiotoxicity and myotoxicity. Adjuvant agents can be added to local anesthetics for the purposes of both prolonging duration of action and reducing toxic effects.[2,3]

Dexmedetomidine is a sympatholytic used as a selective alpha-2 adrenergic agonist agent for sedation. There are

studies showing that adding dexmedetomidine as an adjuvant to local anesthetics reduces local inflammation while prolonging the duration of nerve block.[2,4]

Myotoxicity might be a complication peripheral nerve blocks and bupivacaine is known to have a high myotoxic pontential.[5-7]

We hypothesized that dexmedetomidine, when used as an adjuvant to bupivacaine, might reduce bupivacaine-induced myotoxicity in a rat model, as assessed by histopathological muscle damage and serum CPK levels.

METHODS

Ethics committee approval was obtained from the Animal Experiments Local Ethics Committee of University of Health Sciences Ankara Training and Research Hospital (Date: 31.01.2023, Decision No: 0719). All procedures were conducted in accordance with the ARRIVE guidelines and the national/international guidelines for the care and use of laboratory animals. Blood samples were evaluated in Private ENA Laboratory and tissue samples were evaluated in Ankara Training and Research Hospital Pathology Department Laboratory.

The study was carried out using 28 male Wistar-albino type rats with an average weight of 250 grams, in accordance with the standards recommended by the Council of Europe (European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes) (ETS123). The room temperature was 22-25^{0}C and the environment including 12 hours light/dark cycles was provided before the experiment. Experimental animals were fed ad libitum (23% protein, 5% fat, 15% fiber, 50% carbohydrate) standard rat chow and drank tap water. Experimental animals were randomly divided into four groups of 7 rats each. The right legs of the rats were marked and the gastrocnemius muscles were injected.

- **Group C:** 0.2 ml 0.9% normal saline
- **Group B:** Bupivacaine 0.25% concentration; 0.1 ml 0.5% bupivacaine diluted with 0.1 ml 0.9% saline
- **Group D:** 0.1 ml of 0.01% dexmedetomidine, diluted with 0.1 ml of 0.9% saline, at a concentration of 0.005%
- **Group BD:** 0.1 ml of 0.5% bupivacaine was diluted with 0.1 ml of 0.01% dexmedetomidine and injected intramuscularly at a concentration of 0.25% bupivacaine and 0.005% of dexmedetomidine (Figure 1).

The right gastrocnemius muscle of the rats was used for injection. Injection of medications was administered with a 30 G tuberculin needle after negative aspiration. For the B and BD groups, as a commercial preparation, Buvicaine^{Ò} 0.5% in 20 ml vial, Polifarma, was diluted 1:1 with a concentration of 0.25%. Dekstomid^{Ò} 200mcg/2ml, Polifarma was used as a commercial preparation for dexmedetomidine injected into groups D and BD by diluting it 1:1 with a concentration of 0.005%.

After injections, the rats were placed in separate cages for each group. At the 48th hour after the injection, the injected

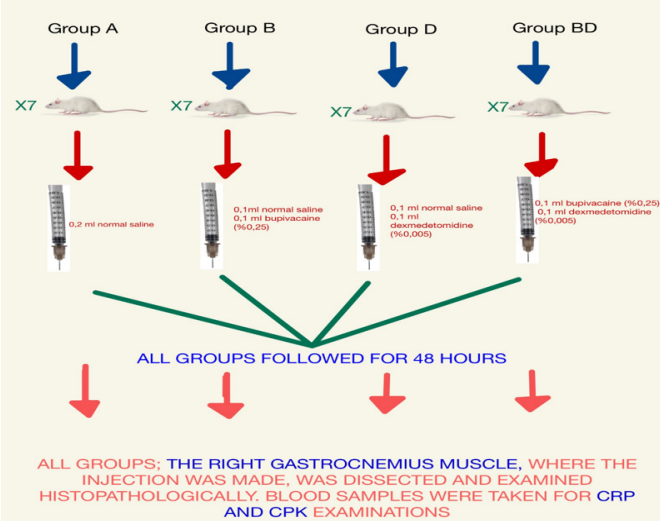

Figure 1. Schematic illustration of the experimental design and study groups

extremities of the rats were removed after skin tissue dissection under ketamine anesthesia and fixed in 10% formalin solution that was previously labeled. After this procedure, the gastrocnemius muscle was separated from the extremities that were fixed as a whole and placed in 10% formaline solution again. Blood samples were taken from the inferior vena cava from the rats for biochemical analysis. At the end of the procedure, the rats were sacrificed by intracardiac blood collection method.

After the tissues were fixed in 10% formalin for 24 hours in the Pathology Department of Ankara Training and Research Hospital, they were dehydrated by passing through a series of 50%, 70%, 80%, 96% and 100% alcohol. Afterwards, the tissues, which were made transparent by passing through xylene, were placed in paraffin blocks by keeping them in paraffin overnight. 5 micron thick sections were taken from paraffin blocks with a microtome (Leica RM 2155), stained with hematoxylin-eosin and examined under a light microscope (Olimpus BX51). Microphotographs of tissue sections (Zeiss Axio Imager M2) were taken. Damage-based scoring was performed and evaluated using the "Benoit, Yagiela, and Ferrell" scoring system[8] (Table 1).

Table 1. Histopathological scoring of muscle damage among study group

Score	Damage status
0	No fiber damage
1	Localized and/or sparse fiber destruction and localized inflammatory cell accumulation
2	Necrosis of several muscle fibers
3	Destruction and necrosis of most of the muscle mass (more than 5 fibers)

Legend: Histopathological muscle injury was graded using the Modified Benoit Score. Higher scores indicate more severe muscle damage.

The primary outcome of the study was histopathological muscle damage, assessed using the Modified Benoit Score (MBS) based on the degree of muscle fiber degeneration, necrosis, and inflammatory cell infiltration in the gastrocnemius muscle.

The secondary outcomes included biochemical markers of muscle injury and inflammation, namely CPK and CRP levels, measured from blood samples obtained at 48 hours after injection.

Statistical Analysis

Data analysis was performed using the IBM SPSS 25.0 (Armonk, NY: IBM Corp.) statistical package program. Descriptive statistical methods (frequency, percentage, mean, standard deviation, median, min-max) were used when evaluating study data. The conformity of the data to the normal distribution was evaluated using the Shapiro-Wilk test, skewness-kurtosis, and graphical methods (histogram, Q-Q Plot, Stem and Leaf, Boxplot). In the study, Kruskal-Wallis test was used for the comparison of quantitative that did not show normal distribution between groups. In cases where there was a difference, a post-hoc Bofferroni correction was applied to find the source of the difference. Relationships between variables were evaluated with Spearman's Rho Correlation test. Statistical significance level was accepted as α=0.05.

RESULTS

This study was performed during a two-day period in March 2023. A total of 28 rats were included in the study, and all animals completed the experimental protocol; no rats were lost during the study. Macroscopic examination performed during dissection revealed mild edema at the injection site in all groups, without evidence of gross necrosis or abscess formation. Histopathological evaluation of muscle samples obtained from the injection sites was performed using light microscopy. Representative photographs and histopathological scores are presented in Figure 2-6.

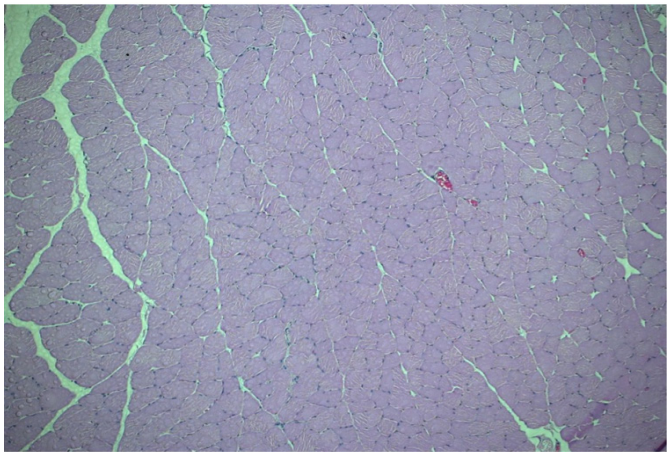

Figure 2. Histopathological appearance of the gastrocnemius muscle in the control group

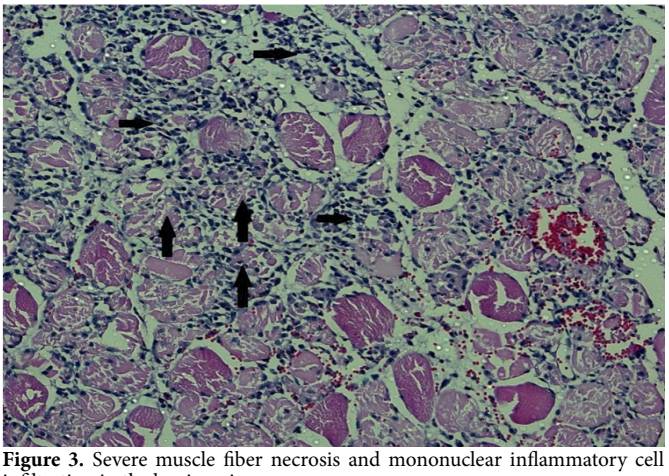

Figure 3. Severe muscle fiber necrosis and mononuclear inflammatory cell infiltration in the bupivacaine group

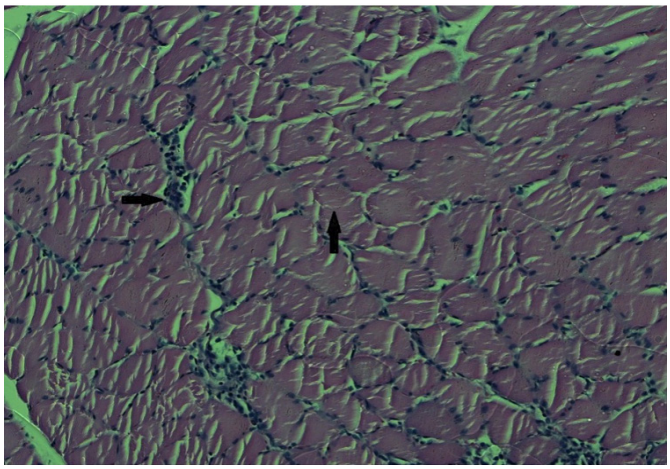

Figure 4. Minimal inflammatory changes in the dexmedetomidine-only group

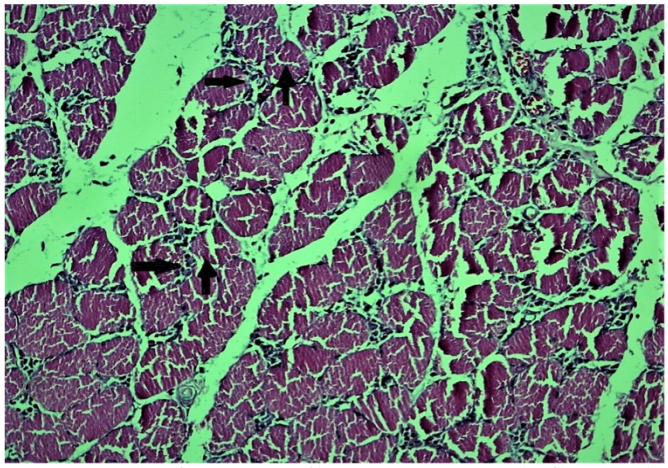

Figure 5. Mild inflammatory infiltration between muscle fibers in the bupivacaine–dexmedetomidine group

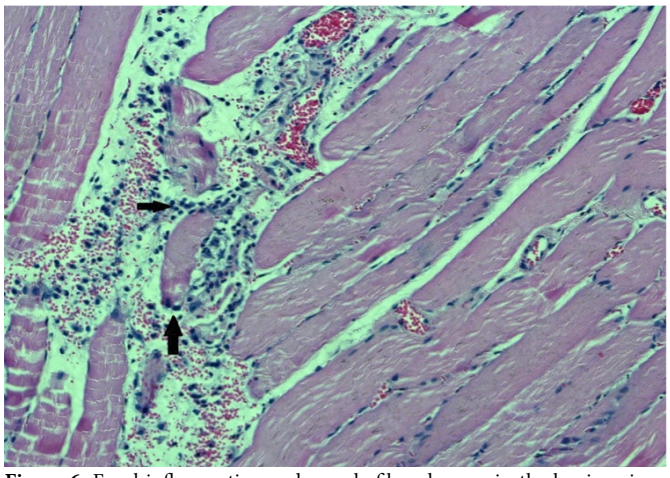

Figure 6. Focal inflammation and muscle fiber damage in the bupivacaine–dexmedetomidine group

In the microscopic evaluation, no damage to the muscle fibers and inflammatory cell infiltration were observed in any of the samples in the 0.9% saline injection group (group C) (Figure 2). In between myofibrillary areas diffuse mononuclear leukocyte infiltration, diffuse necrobiotic change and degeneration were observed in most of the samples in group B (Figure 3). In group group D, the appearance was generally similar to the group C. In some samples, only focal and light density mononuclear cell accumulation was observed between muscle fibers (Figure 4). In the group BD, mild inflammation was observed between muscle fibers (score 1) in only three

samples (Figure 5). Focal inflammation and damage to some muscle fibers (score 2) were observed in some samples in the group BD. Inflammatory cells were predominantly composed of lymphocytes. In some cases, eosinophils were also seen in the infiltrate (Figure 6).

There was a statistically significant difference ($p<0.05$) between the groups in MBS values. Multiple comparison (post-hoc) tests were applied to find out from which groups the difference originated. There was a difference between group B and other groups, group B MBS values were higher than the other groups. In addition, there was a difference between group BD and other groups, group BD MBS values were found to be higher than group C and group D MBS values, and lower than group B values (Table 2).

There was a statistically significant difference between the groups in CPK values ($p<0.05$). When multiple comparison (post-hoc) tests were applied to it, there was a difference between group B and group C and D. group B values were found to be the highest (Table 3).

In the comparisons between the groups; It was found that there was no statistically significant difference between the groups in terms of CRP values ($p>0.05$) (Table 4).

Relationships between the MBS–CPK–CRP are:

- A positive correlation was found between the MBS and CPK, at the level of r=0.827 (it can explain approximately 68% of the variation in MBS (R^2=0.684)),
- A positive correlation was found between the MBS and CRP, at the level of r=0.471. (which can explain approximately 22% of the variation in MBS (R^2=0.222)),
- A positive correlation was found between CPK and CRP at the level of r=0.669. (explains approximately 45% of the variation in CPK (R^2=0.448)),
- These relationships were found to be statistically significant ($p<0.05$) (Table 5).

Table 5. Relationships between Modified Benoit Score–CPK–CRP

	Modified Benoit Score		CPK		CRP	
	r	p*	r	p*	r	p*
Modified Benoit Score	1.000	--	0.827	<0.001	0.471	0.011
CPK	0.827	<0.001	1.000	--	0.669	<0.001
CRP	0.471	0.011	0.669	<0.001	1.000	--

Legend: Correlations were assessed using Spearman's rho correlation test. CPK: Creatine phosphokinase, CRP: C-reactive protein

DISCUSSION

In this experimental study, we investigated the histopathological and biochemical effects of dexmedetomidine on bupivacaine-induced myotoxicity in a rat model. The main findings of the study demonstrated that bupivacaine caused significant myotoxic muscle damage, as evidenced by high MBSs and elevated serum CPK levels. The addition of dexmedetomidine to bupivacaine resulted in a significant reduction in histopathological muscle damage and lower CPK levels compared with bupivacaine alone, while dexmedetomidine administered alone did not induce myotoxic changes. No significant differences were observed among the groups with respect to CRP levels. These findings suggest that dexmedetomidine may attenuate bupivacaine-

Table 2. Comparison of Modified Benoit Score between groups

		Group A (n=7)	Group B (n=7)	Group D (n=7)	Group BD (n=7)	p*	Difference
Modified Benoit Score*		0.00±0.00 0 (0.0-0.0)	2.57±0.79 3.0 (1.0-3.0)	0.43±0.53 0.0 (0.0-1.0)	1.57±0.53 2.0 (1.0-2.0)	<0.001[a]	B with A-D-BD BD with A-B-D
	0**	7 (100.0%)	--	4 (57.1%)	--	--	--
	1**	--	1 (14.3%)	3 (42.9%)	3 (42.9%)		
	2**	--	1 (14.3%)	--	4 (57.1%)		
	3**	--	5 (71.4%)	--	--		

Legend: Data are presented as mean±SD and median (min–max). Comparisons were performed using the Kruskal–Wallis test. Abbreviations: C: Control, B: Bupivacaine, D: Dexmedetomidine, BD: Bupivacaine+ Dexmedetomidine

Table 3. Comparison of CPK between groups

	Group A (n=7)	Group B (n=7)	Group D (n=7)	Group BD (n=7)	p	Difference
CPK*	894.7±270.6 844.0 (514.0–1.196,0)	1.784,9±535,5 1.731,0 (1.110,0–2.562,0)	885.9±258.3 753.0 (687.0–1.308,0)	1.207,0±428.2 1.462,0 (522.0–1.591,0)	0.007[a]	B with A-D

Legend: Serum CPK levels were compared using the Kruskal–Wallis test. Abbreviations: CPK: Creatine phosphokinase

Table 4. Comparison of CRP between groups

	Group A (n=7)	Group B (n=7)	Group D (n=7)	Group BD (n=7)	p
CRP*	0.16±0.10 0.10 (0.10–0.30)	0.21±0.09 0.20 (0.10–0.30)	0.14±0.05 0.10 (0.10–0.20)	0.17±0.05 0.20 (0.10–0.20)	0.343[a]
0.1**	5 (71.4%)	2 (28.6%)	4 (57.1%)	2 (28.6%)	
0.2**	--	2 (28.6%)	3 (42.9%)	5 (71.4%)	--
0.3**	2 (28.6%)	3 (42.9%)	--	--	

Legend: Serum CRP levels were compared using the Kruskal–Wallis test. Abbreviations: CRP: C-reactive protein

induced myotoxicity without causing additional muscle injury.

Bupivacaine is the local anesthetic that is frequently preferred in peripheral blocks due to its duration of action and high potency.[9] Bupivacaine has the highest known myotoxic potential.[3,10] Due to the advantages and comfort of peripheral blocks, the toxic effect profile of local anesthetics has started to be seen more frequently as a result of the increased frequency of use. Myotoxicity due to local anesthetics has been argued to be clinically insignificant due to its rarity and reversible nature, but myonecrosis and severe muscle dysfunction have been reported with increasing frequency of use.[11] In our study, we investigated how dexmedetomidine affects bupivacaine-induced myotoxicity histopathologically and biochemically.

Bupivacaine causes damage by causing intracellular calcium release via ryanodine receptors. It's also known that bupivacaine inhibits calcium absorption into the sarcoplasmic reticulum. As a result, the intracellular calcium concentration reaches toxic levels.[12,13] Bupivacaine-induced myotoxicity has been closely associated with mitochondrial dysfunction.[5] It affects energy metabolism by disrupting mitochondrial functions and increases its effects on calcium.[13] Nouette-Gaulain et al.[14,15] showed that the preservation of mitochondrial functions with the effect of human recombinant erythropoietin (rhEPO) in rats treated with rhEPO can reduce the myotoxic effects of bupivacaine.

Oz Gergin et al.[16] investigate the myotoxic effects of bupivacaine, ropivacaine and levobupivacaine on rats, showed that bupivacaine caused more myotoxic damage than other agents. Histopathology detected myonecrotic debris and inflammatory cells. In electron microscopy, they observed vacuolization and mitochondrial swelling in the sarcoplasmic reticulum. These findings were consistent with studies describing pathophysiology in the literature. In histopathology, the highest level of myocnecrosis and all indicators of necrobiotic changes are best observed 48 hours after bupivacaine injection.[17,18] Foster et al.[19] they administered local anesthetics in 200 µl volume by i.m. injection in rats and reported that this volume did not cause pressure damage. Nouette-Gaulain et al.[15] showed that toxic damage increased in direct proportion with exposure time. In addition, a dose-related relationship between bupivacaine-induced myotoxicity has been shown in the literature. It has been shown that as the concentration increases, it can cause longer lasting myotoxicity and even permanent damage.[20]

In our study, we examined the histopathological changes on the myocytes at 48th hour by injecting 200 µl of 0.25% concentration of bupivacaine into the gastrocnemius muscle of rats in group B, based on the information in the literature. We observed statistically significant high ($p<0.001$) myonecrotic changes in the bupivacaine group only when compared to all other groups histopathologically. We observed these changes in 71.4% of the rats in the bupivacaine group, the highest score of 3 when scored on myonecrotic muscle fibrils and mononuclear inflammatory cell deposition. Histopathological score of 3 was not observed in rats in other groups.

Adjuvant agents are added to reduce the toxic effects of local anesthetics and to prolong their duration of action. It has been reported that steroid and epinephrine added to local anesthetics increase myotoxicity.[21,22] Guttu et al.[22] showed that adding steroids to bupivacaine significantly increased myotoxicity. Padera et al.[23] In their study on rats, they showed that tetradotoxin added to bupivacaine as a Na channel blocker significantly prolonged the blocking time, but had no reducing effect on myotoxicity. Plank et al.[7] conducted in mouse muscle cell culture, found that dantrolene reduced the damage due to its effect on calcium metabolism, while caffeine increased it. Although clonidine, found to not have an effect on the duration of action of long-acting local anesthetics such as bupivacaine in peripheral blocks.[24–26] Erdivanli et al.[27] showed that dexmedetomidine added to intrathecal bupivacaine increased the quality and duration of analgesia in a dose-dependent manner, and dexmedetomidine didn't cause any neurotoxicity and inflammation. Brummet et al.[2] showed that perineural administration of dexmedetomidine combined with bupivacaine increased the duration of sensory and motor blockade without causing neurotoxicity, while dexmedetomidine alone did not cause significant motor and sensory blockade. They concluded that dexmedetomidine exerts these effects presynaptically by inhibiting nociceptive transmission and by hyperpolarizing postsynaptic a-2a receptors.[2] Addition of dexmedetomidine to bupivacaine has been shown to reduce postoperative pain scores and significantly prolong the duration of the block in supraclavicular block, abdominal facial plane blocks, and thoracic wall facial plane blocks.[28–30] Jin et al.[31] reported that dexmedetomidine added to bupivacaine reduced cardiac toxicity by reducing bupivacaine-induced vasopermeability-related protein production. The effects of dexmedetomidine and dexamethasone added to liposomal bupivacaine were compared and the block times were prolonged, but there was less inflammation in the samples added dexmedetomidine.[4] In a recent review of the myotoxic effects of local anesthetics, it was mentioned that there is no study on the effects of dexmedetomidine or dexamethasone added to local anesthetics on myotoxicity, and it was stated that there is a need for experimental studies on this subject. There are no experimental studies on the myotoxicity of dexmedetomidine.[11]

We aimed to show whether dexmedetomidine, which has been shown to prolong the block time and reduce tissue inflammation in previous studies, has an effect on myotoxic damage. The dose of dexmedetomidine was calculated for rats according to the publications and injected at a concentration of 0.005% and in a volume of 200 µl.[32] In group D, we found histopathologically similar scores to the control group. While 57.1% of the muscle samples of the rats in group D had the same findings as the control group, localized myofibril damage and localized inflammatory cell accumulation were observed in 42.9% of them. In the BD group, the scores were higher than groups A and D, but lower than group B. As a result of our histopathological evaluations, it was observed that dexmedetomidine alone did not cause myotoxicity and it statistically significantly reduced bupivacaine-induced myotoxicity ($p<0.001$).

We think that dexmedetomidine can reduce the required concentrations of bupivacaine with its analgesic feature alone, and it will also reduce bupivacaine-induced myotoxicity.

In a randomized controlled study, it was shown that premedication of low-dose intramuscular dexmedetomidine (1 μg.kg-1) caused preoperative sedation without clinically significant bradycardia or hypotension.[33] Similarly, in our study, we observed the sedation effect in D and BD group rats to which we applied dexmedetomidine.

Nosaka et al.[34] showed that plasma creatine phosphokinase (CPK) levels increased after bupivacaine injection into the human biceps muscle, and this increase was directly proportional to the dose of bupivacaine administered and thus the amount of muscle damage. In our study, a statistically significant difference ($p<0.05$) was found between the groups in terms of CPK values, parallel to similar studies. There was a difference between group B and group C and D, and group B values were the highest. In the BD group to which we added dexmedetomidine, the CPK value was not as high as in the B group.

He et al.[35] observed that oxidative stress and apoptosis were less in the group supplemented with dexmedetomidine from doxorubucin-induced myocardial cell cultures. We examined CRP values biochemically to evaluate the inflammatory response. In the comparisons between the groups, there was no statistically significant difference between the groups in terms of CRP values ($p>0.05$). This result is consistent with studies in the literature showing that the myotoxic effect of bupivacaine is local and does not cause systemic inflammation. In the literature, the inflammatory response-reducing effects of dexmedetomidine have been observed in cases with a stress factor such as surgery or cancer. Since there was no stress factor in our study, a statistically significant difference may not have been observed.

When the relations between MBS–CPK–CRP are examined, with MBS; While there was a strong correlation between CPK, a weak correlation was found between CRP. This made us think that high CPK values are closely related to high histopathological damage and that we can use it to predict myocyte damage.

If we talk about the limitations of our study, we do not have any factors such as incision or surgical stress exposure that will cause systemic inflammation when evaluating CRP. Therefore, we think that we did not find a significant change in CRP values. In addition, we could not compare because the rats did not have CRP and CPK values before intramuscular drug injection.

Although myotoxicity due to local anesthetics is seen as a rare and reversible side effect of these drugs, it can cause serious clinical problems especially in low-volume muscles such as ocular muscles, previously damaged muscles and in those with existing muscle disease. Continuous infusions and repeated injections of local anesthetics can also cause serious clinical damage.

Regional blocks are preferred in the geriatric age group to avoid the risks of general anesthesia due to comorbidities. Peribulbar and retrobulbar blocks are preferred especially in ophthalmic surgeries. Myotoxic damage to the extraocular muscles due to local anesthetics used in these blocks presents as diplopia and strabismus.[36,37] Parris et al.[38] reported that atrophy of the trapezius muscle, characterized by pain in the pericapsular region, developed as a result of trigger point injections using bupivacaine for the treatment of myofascial pain syndrome.

Therefore, clinicians should not forget the fact of myotoxicity and aim for longer duration of anesthesia and analgesia with single-dose injections. For this purpose, adjuvant agents thought to reduce myotoxicity should be used.

Limitations

This study has certain limitations. First, the experimental design was based on a rat model, and therefore the findings have limited direct applicability to clinical practice. In addition, only a single dose and administration protocol were evaluated, and the effects of different dosing regimens or repeated applications were not examined.

Furthermore, the assessment of myotoxicity was limited to histopathological findings at a single time point, without evaluation of functional outcomes or underlying molecular mechanisms. Accordingly, further experimental and clinical studies are needed to better clarify the potential protective effects of dexmedetomidine against bupivacaine-induced myotoxicity.

CONCLUSION

The growing use of plane blocks and peripheral nerve blocks for intraoperative anesthesia and postoperative analgesia has increased the clinical relevance of potential toxic effects associated with local anesthetics. Bupivacaine, which is widely preferred in clinical practice due to its long duration of action and high potency, has a pronounced myotoxic potential related to its high lipophilicity.

In this study, we aimed to test the hypothesis that dexmedetomidine, which is known to reduce local inflammation, may attenuate bupivacaine-induced myotoxicity when used as an adjuvant to bupivacaine in a rat model. Our findings suggest that dexmedetomidine does not exhibit myotoxic effects when administered alone and may reduce bupivacaine-induced myotoxicity when used in combination with bupivacaine. Nevertheless, further experimental and clinical studies involving different doses and administration protocols are required to clarify the clinical implications of these findings.

ETHICAL DECLARATIONS

Ethics Committee Approval

Ethics committee approval was obtained from the Animal Experiments Local Ethics Committee of University of Health Sciences Ankara Training and Research Hospital (Date: 31.01.2023, Decision No: 0719).

Informed Consent

Informed consent is not required because it is an animal experiment.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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Author Contributions

Concept or Design of the Manuscript: G.Y.Y., Z.N.A.; Acquisition, Analysis and Interpretation of Data: M.S.D., M.C.Y.; Writing–Drafting the Manuscript: All authors; Critical Revision for Important Intellectual Content: E.Y.A., G.Y.Y.; Final Approval of the Version to be Published: All authors

REFERENCES

1. Shams D, Sachse K, Statzer N, Gupta RK. Regional anesthesia complications and contraindications. *Clin Sports Med*. 2022;41(2):329-343. doi:10.1016/j.csm.2021.11.006
2. Brummett CM, Norat MA, Palmisano JM, Lydic R. Perineural administration of dexmedetomidine in combination with bupivacaine enhances sensory and motor blockade in sciatic nerve block without inducing neurotoxicity in rat. *Anesthesiology*. 2008;109(3):502-511. doi:10.1097/ALN.0b013e318182c26b
3. Zink W, Graf BM. Local anesthetic myotoxicity. *Reg Anesth Pain Med*. 2004;29(4):333-340. doi:10.1016/j.rapm.2004.04.002
4. Rwei AY, Sherburne RT, Zurakowski D, Wang B, Kohane DS. Prolonged duration local anesthesia using liposomal bupivacaine combined with liposomal dexamethasone and dexmedetomidine. *Anesth Analg*. 2018;126(4):1170-1175. doi:10.1213/ANE.0000000000002771
5. Irwin W, Fontaine E, Agnolucci L, et al. Bupivacaine myotoxicity is mediated by mitochondria. *J Biol Chem*. 2002;277(14):12221-12227. doi:10.1074/jbc.M111242200
6. Mao Q, Yu W, Liu S, et al. Downregulation of HSPA12A underlies myotoxicity of local anesthetic agent bupivacaine through inhibiting PGC1α-mediated mitochondrial integrity. *Toxicol Appl Pharmacol*. 2022;434:115798. doi:10.1016/j.taap.2021.115798
7. Plank C, Hofmann P, Gruber M, et al. Modification of bupivacaine-induced myotoxicity with dantrolene and caffeine in vitro. *Anesth Analg*. 2016;122(2):418-423. doi:10.1213/ANE.0000000000000988
8. Benoit PW, Yagiela JA, Ferrell Fort N, Fort JA. Pharmacologic correlation between local anesthetic-induced myotoxicity and disturbances of intracellular calcium distribution. *Toxicol Appl Pharmacol*. 1980;52(2):187-198. doi:10.1016/0041-008X(80)90105-2
9. Finneran JJ, Ilfeld BM. Peripheral nerve blocks. In: Butterworth JF, Mackey DC, Wasnick JD, eds. Morgan & Mikhail's Clinical Anesthesiology. 7th ed. New York, NY: McGraw-Hill; 2022:975-1107.
10. Zink W, Seif C, Bohl JRE, et al. The acute myotoxic effects of bupivacaine and ropivacaine after continuous peripheral nerve blockades. *Anesth Analg*. 2003;97(4):1173-1179. doi:10.1213/01.ANE.0000080610.14265.C8
11. Hussain N, McCartney CJL, Neal JM, Chippor J, Banfield L, Abdallah FW. Local anaesthetic-induced myotoxicity in regional anaesthesia: a systematic review and empirical analysis. *Br J Anaesth*. 2018;121(4):822-841. doi:10.1016/j.bja.2018.06.013
12. Shoshan-Barmatz V, Zchut S. The interaction of local anesthetics with the ryanodine receptor of the sarcoplasmic reticulum. *J Membr Biol*. 1993;133(2):171-181. doi:10.1007/BF00233049
13. Zink W, Graf BM, Sinner B, Martin E, Fink RHA, Kunst G. Differential effects of bupivacaine on intracellular Ca^2 regulation: potential mechanisms of its myotoxicity. *Anesthesiology*. 2002;97(3):710-716. doi:10.1097/00000542-200209000-00027
14. Nouette-Gaulain K, Bellance N, Pré B, et al. Erythropoietin protects against local anesthetic myotoxicity during continuous regional analgesia. *Anesthesiology*. 2009;110(4):821-831. doi:10.1097/ALN.0b013e31819c4a4f
15. Nouette-Gaulain K, Bringuier S, Canal-Raffin M, et al. Time course of mitochondrial metabolism alterations to repeated injections of bupivacaine in rat muscle. *Can J Anaesth*. 2010;57(9):836-842. doi:10.1007/s12630-010-9337-2
16. Öz Gergin Ö, Yildiz K, Bayram A, et al. Comparison of the myotoxic effects of levobupivacaine, bupivacaine, and ropivacaine: an electron microscopic study. *Ultrastruct Pathol*. 2015;39(3):169-176. doi:10.3109/01913123.2015.1028429
17. Horiguchi T, Shibata MA, Ito Y, Eid NAS, Abe M, Otsuki Y. Macrophage apoptosis in rat skeletal muscle treated with bupivacaine hydrochloride: possible role of MCP-1. *Muscle Nerve*. 2002;26(1):79-86. doi:10.1002/mus.10166
18. Yildiz K, Efesoy SN, Ozdamar S, et al. Myotoxic effects of levobupivacaine, bupivacaine and ropivacaine in a rat model. *Clin Invest Med*. 2011;34(5):E273-E281. doi:10.25011/cim.v34i5.15365
19. Foster AH, Carlson BM. Myotoxicity of local anesthetics and regeneration of the damaged muscle fibers. *Muscle Nerve*. 1980;3(4):286-293. doi:10.1002/mus.880030405
20. Zhang C, Phamonvaechavan P, Rajan A, Poon DY, Topcu-Yilmaz P, Guyton DL. Concentration-dependent bupivacaine myotoxicity in rabbit extraocular muscle. *J AAPOS*. 2010;14(4):323-327. doi:10.1016/j.jaapos.2010.05.004
21. Benoit PW. Reversible skeletal muscle damage after administration of local anesthetics with and without epinephrine. *J Oral Surg*. 1978;36(3):198-201. doi:10.1016/S0030-4220(78)80005-1
22. Guttu RL, Page DG, Laskin DM. Delayed healing of muscle after injection of bupivacaine and steroid. *Ann Dent*. 1990;49(1):5-8.
23. Padera RF, Tse JY, Bellas E, Kohane DS. Tetrodotoxin for prolonged local anesthesia with minimal myotoxicity. *Muscle Nerve*. 2006;34(6):747-753. doi:10.1002/mus.20633
24. Culebras X, Van Gessel E, Hoffmeyer P, Gamulin Z. Clonidine combined with a long acting local anesthetic does not prolong postoperative analgesia after brachial plexus block but does induce hemodynamic changes. *Anesth Analg*. 2001;92(1):199-204. doi:10.1097/00000539-200101000-00039
25. Duma A, Urbanek B, Sitzwohl C, Kreiger A, Zimpfer M, Kapral S. Clonidine as an adjuvant to local anaesthetic axillary brachial plexus block: a randomized, controlled study. *Br J Anaesth*. 2005;94(1):112-116. doi:10.1093/bja/aei012
26. Kanazi GE, Aouad MT, Jabbour-Khoury SI, et al. Effect of low-dose dexmedetomidine or clonidine on the characteristics of bupivacaine spinal block. *Acta Anaesthesiol Scand*. 2006;50(2):222-227. doi:10.1111/j.1399-6576.2006.00919.x
27. Erdivanli B, Altun M, Sezen ÖK, Çolakoğlu SA. Anti-nociceptive, analgesic and pathohistological effects of intrathecal dexmedetomidine and bupivacaine in rats. *Rev Bras Anestesiol*. 2013;63(4):373-379. doi:10.1016/j.bjane.2012.08.004
28. Aksu R, Patmano G, Biçer C, Emek E, Çoruh AE. Efficiency of bupivacaine and association with dexmedetomidine in transversus abdominis plane block ultrasound guided in postoperative pain of abdominal surgery. *Braz J Anesthesiol (Engl Ed)*. 2018;68(1):49-56. doi:10.1016/j.bjane.2017.09.004
29. Campoy L, Martin-Flores M, Boesch JM, et al. Transverse abdominis plane injection of bupivacaine with dexmedetomidine or a bupivacaine liposomal suspension yielded lower pain scores and requirement for rescue analgesia in a controlled, randomized trial in dogs undergoing elective ovariohysterectomy. *Am J Vet Res*. 2022;83(9):729-738. doi:10.2460/ajvr.22.03.0045
30. Manzoor S, Taneja R, Sood N, Puri A, Kadayaprath G. Comparative study to assess the quality of analgesia of bupivacaine and bupivacaine with dexmedetomidine in ultrasound-guided pectoral nerve block type I and II in breast surgeries. *J Anaesthesiol Clin Pharmacol*. 2018;34(2):227-231. doi:10.4103/joacp.JOACP_310_16
31. Jin Z, Xia F, Lin T, Cai Y, Chen H, Wang Y. Dexmedetomidine may decrease the bupivacaine toxicity to heart. *Open Med (Poland)*. 2021;16(1):1070-1075. doi:10.1515/med-2021-0300
32. Miller DL, Dou C, Dong Z, Raghavendran K. The influence of dexmedetomidine on ultrasound-induced pulmonary capillary hemorrhage in rats. *Ultrasound Med Biol*. 2016;42(4):964-970. doi:10.1016/j.ultrasmedbio.2015.12.004
33. Sun Y, Liu C, Zhang Y, et al. Low-dose intramuscular dexmedetomidine as premedication: a randomized controlled trial. *Med Sci Monit*. 2014;20:2714-2719. doi:10.12659/MSM.891099
34. Nosaka K, Sakamoto K. Changes in plasma enzyme activity after intramuscular injection of bupivacaine into the human biceps brachii. *Acta Physiol Scand*. 1999;167(3):259-265. doi:10.1046/j.1365-201X.1999.00502.x

35. He Y, Yang Z, Li J, Li E. Dexmedetomidine reduces the inflammation and apoptosis of doxorubicin-induced myocardial cells. *Exp Mol Pathol*. 2020;113:104380. doi:10.1016/j.yexmp.2020.104380
36. Wong MB, Wong DH. Regional anaesthesia for intraocular surgery. *Can J Anaesth*. 1993;40(7):635-642. doi:10.1007/BF03009716
37. Gómez-Arnau JI, Yangüela J, González A, et al. Anaesthesia-related diplopia after cataract surgery. *Br J Anaesth*. 2003;90(2):189-193. doi:10.1093/bja/aeg046
38. Parris WC, Dettbarn WD. Muscle atrophy following nerve block therapy. *Anesthesiology*. 1988;69(2):289-294. doi:10.1097/00000542-198808000-00021

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Length of stay in intensive care unit and factors associated with clinical outcomes in patients with postoperative intracranial masses

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ABSTRACT

Aims: This study aimed to evaluate factors associated with intensive care unit (ICU) length of stay (LOS) and mortality in patients who underwent surgical intervention for postoperative intracranial masses and required ICU monitoring.

Methods: This retrospective, single-center observational study included adult patients who underwent intracranial mass surgery and were followed in the ICU for at least 48 hours between January 2017 and December 2025. Demographic characteristics, comorbidities, tumor-related variables, clinical scores (Glasgow Coma Scale and APACHE II), need for and duration of mechanical ventilation, ICU complications, LOS, and mortality were recorded. Prolonged ICU stay was defined as >3 days. Univariate and multivariate logistic regression analyses were performed to identify independent predictors of prolonged ICU stay.

Results: A total of 128 patients were included. The median ICU LOS was 3 days, and ICU mortality was 25.8%. Mechanical ventilation requirement, ventilation duration, respiratory complications, and higher APACHE II scores were significantly associated with both ICU LOS and mortality. In multivariate analysis, the APACHE II score was the only independent predictor of prolonged ICU stay (OR: 0.924, 95% CI: 0.862–0.991; p=0.027). Tumor type showed an association with ICU LOS in univariate analyses, whereas tumor location was not independently associated with outcomes.

Conclusion: In patients with postoperative intracranial masses, ICU LOS and mortality are primarily influenced by acute clinical severity and systemic complications rather than tumor characteristics. The APACHE II score is an independent predictor of prolonged ICU stay, highlighting the importance of early risk stratification to optimize ICU resource utilization.

Keywords: Intensive care unit, intracranial mass, length of stay, APACHE II, mortality

INTRODUCTION

Intracranial masses constitute a heterogeneous group of conditions, including primary brain tumors, metastatic lesions, vascular pathologies, and other space-occupying intracranial disorders.[1] These lesions may cause serious clinical manifestations, such as increased intracranial pressure, focal neurological deficits, and altered consciousness, frequently necessitating surgical intervention.[2] After surgery, a subset of patients requires admission to the intensive care unit (ICU) for close hemodynamic and neurological monitoring.[3]

Early identification of postoperative complications—including intracranial hemorrhage, cerebral edema, ischemic events, seizures, and hydrocephalus—is critical in this patient population.[4] Factors such as neurological deterioration, the need for mechanical ventilation, hemodynamic instability, infectious complications, and metabolic disturbances pose major challenges that may prolong the ICU course.[5] In this context, ICU length of stay (LOS) is considered a key indicator of both patient prognosis and efficient use of healthcare resources.[5,6]

The duration of hospitalization after craniotomy and tumor resection has been shown to be influenced by multiple factors, including patient-related characteristics (age, comorbidities,

frailty, and preoperative neurological deficits), surgical and anesthetic variables (operative duration, intraoperative blood loss and transfusion requirements), and postoperative complications such as prolonged mechanical ventilation, infections, and thromboembolic events.[7,8]

Extended stays in intensive care are associated with increased risk of complications, higher mortality rates, and a greater cost burden on the healthcare system.[9] Therefore, identifying the factors that determine the LOS in intensive care and clinical outcomes in patients with postoperative intracranial masses is important for optimizing patient management.[10] Although studies exist on this patient group, those that focus on intensive care and examine clinical outcome-related factors in detail are limited.[11]

This study aimed to retrospectively evaluate factors associated with intensive care LOS and mortality in patients who underwent surgical intervention for an intracranial mass and were monitored in intensive care during the postoperative period.

METHODS

Ethics

After obtaining ethical approval from the Fırat University Non-interventional Researches Ethics Committee (Date: 05.01.2026, Decision No: E-50716828-020-755356), patient data were analyzed retrospectively. Our study was conducted in accordance with the Helsinki Declarations and the Strengthening the Reporting of Observational studies in Epidemiology (STROBE) guidelines.

Study Design and Setting

This retrospective, observational, single-center study included patients with postoperative intracranial tumors who were followed in the third-level intensive care unit of Fırat University Hospital between January 2017 and December 31, 2025.

Owing to the study's retrospective nature, informed consent was waived.

Patient Selection and Inclusion Criteria

All patients who underwent intracranial tumor surgery within the specified time period and were monitored in the ICU for at least 48 hours postoperatively were included in the study. The inclusion criteria were as follows:

- Surgical intervention due to an intracranial mass
- Postoperative admission to the ICU

Patients with an ICU stay of less than 48 hours were excluded because early discharge or brief observation was deemed inadequate to capture the clinical course. Additionally, pregnant patients and those with incomplete clinical data that would not permit evaluation of the primary endpoints were excluded from the study. The study included only the initial postoperative ICU admission.

Patients were divided into two groups according to the median ICU LOS of the study population. Patients with an ICU stay of ≤3 days were classified as the short ICU stay group, whereas those with an ICU stay of >3 days were classified as the prolonged ICU stay group. All comparative and regression analyses were performed based on this grouping.

Data Collection

The data was obtained retrospectively utilizing the hospital information management system (HIMS), the intensive care patient monitoring system, and patient medical records. Demographic characteristics (age, sex, and comorbidities), diagnosis, type and location of the intracranial mass, tumor size, Glasgow Coma Scale (GCS) score, APACHE II score, requirement for and duration of mechanical ventilation, LOS in the ICU, complications (neurological, hemodynamic, respiratory, hemorrhagic, and metabolic), ICU mortality, and patient outcomes at ICU discharge (transfer to the ward, discharge, or death) were retrospectively collected from patient medical records and the hospital information system.

Study Endpoints

The primary endpoint of this study was prolonged ICU stay, defined as an ICU LOS exceeding 3 days.

Secondary endpoints included ICU mortality, need for mechanical ventilation, duration of mechanical ventilation, and complications developing during the ICU stay.

Statistical Analysis

The collected data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Variables with normal distribution were presented as mean±standard deviation. For non-normally distributed continuous variables, data are presented as median and interquartile range (IQR), defined as the 25^{th}–75^{th} percentiles. Categorical variables were expressed as counts and percentages. Prolonged ICU stay was defined as an ICU LOS exceeding the median value of the study population (>3 days). Multivariate logistic regression was performed to identify independent risk factors for a prolonged ICU stay (>3 days). Variables with $p < 0.10$ in univariate analyses were included in the multivariate model. Multicollinearity among variables included in the multivariable model was assessed using the variance inflation factor (VIF), and variables with a VIF>5 were considered indicative of problematic multicollinearity. Results were reported as odds ratios (OR) and 95% confidence intervals (CI). $p<0.05$ was accepted as statistically significant.

RESULTS

Patient Characteristics

A total of 128 postoperative patients with intracranial masses were included in the study. Patients were divided into two groups according to the median ICU LOS: ≤3 days (n=59) and >3 days (n=69). The overall mean age was 50.78±17.50 years. Patients with prolonged ICU stay were significantly older than those with shorter ICU stay (53.72±16.60 vs. 47.35±18.04 years, p=0.040). Sex distribution did not differ significantly between the two groups (p=0.302).

The mean GCS score was lower in patients with prolonged ICU stays than in those with shorter ICU stays (6.91±4.50 vs. 8.57±4.56), although this difference did not reach

statistical significance (p=0.087). In contrast, the APACHE II score was significantly higher in the prolonged ICU stay group (18.81±8.87 vs. 12.77±6.27, p<0.001). Tumor type was significantly associated with ICU LOS (p=0.004). Prolonged ICU stay was more frequent among patients with glioblastoma/oligodendroglioma and metastatic tumors compared with other tumor types. Tumor location was not significantly associated with ICU LOS (p=0.744). Preoperative neurological deficits were significantly more common in patients with prolonged ICU stay (71.0% vs. 44.1%, p=0.002). The Charlson Comorbidity Index, expressed as median [IQR], did not differ significantly between groups (p=0.062). Intraoperative vasopressor use was significantly more frequent in patients with prolonged ICU stay (37.7% vs. 6.8%, p<0.001). Detailed demographic and clinical characteristics are presented in Table 1.

Complications Observed During ICU Follow-up

Complications observed during ICU follow-up were significantly more frequent in patients with prolonged ICU stay. Neurological deficits were observed in 56.5% of patients with ICU LOS >3 days compared to 15.3% in those with ICU LOS ≤3 days (p<0.001). Similarly, seizures (39.1% vs. 1.7%), arrhythmias (60.9% vs. 18.6%), respiratory complications (63.8% vs. 8.5%), metabolic complications (47.8% vs. 6.8%), and hemorrhagic complications (27.5% vs. 5.1%) were all significantly more common in the prolonged ICU stay group (all p<0.001). These findings are summarized in Table 2.

Clinical Outcomes

Mechanical ventilation was required in 72 patients (56.3%) overall and was significantly more common among patients with prolonged ICU stay (71.0% vs. 39.0%, p<0.001). The duration of mechanical ventilation was also significantly longer in patients with prolonged ICU stay (median 7 [1–110] days vs. 1 [1–2] days, p<0.001). ICU mortality occurred in 33 patients (25.8%) and was markedly higher in the prolonged ICU stay group compared to the short stay group (42.0% vs. 6.8%, p<0.001). Correspondingly, survival at ICU discharge was significantly lower among patients with ICU LOS >3 days (58.0% vs. 93.2%, p<0.001). Clinical outcomes according to ICU LOS are presented in Table 3.

Table 3. Clinical outcomes according to ICU length of stay

Outcome	Total (n=128)	ICU LOS ≤3 days	ICU LOS >3 days	p value
Mechanical ventilation (yes)	72 (56.3%)	23 (39.0%)	49 (71.0%)	<0.001
Duration of mechanical ventilation (days)	1 (1–110)	1 (1–2)	7 (1–110)	<0.001
ICU length of stay (days)	3 (1–110)	2 (1–2)	9 (3–110)	<0.001
ICU mortality	33 (25.8%)	4 (6.8%)	29 (42.0%)	<0.001
Discharge status (alive)	95 (74.2%)	55 (93.2%)	40 (58.0%)	<0.001

ICU: Intensive care unit, LOS: Length of stay

Table 1. Demographic and clinical characteristics according to ICU length of stay

Variable	Total (n=128)	ICU LOS ≤3 days (n=59)	ICU LOS >3 days (n=69)	p value
Age (years)	50.78±17.50	47.35±18.04	53.72±16.60	0.040
Sex (female/male)	63/65	31/28	32/37	0.302
Glasgow Coma Scale	7.67±4.58	8.57±4.56	6.91±4.50	0.087
Tumour type:				
Metastasis	8 (6.2%)	3 (4.0%)	5 (9.2%)	0.004
Glioblastoma/ oligodendroglioma	41 (32.0%)	17 (22.9%)	24 (44.4%)	
Meningioma	45 (35.2%)	30 (40.5%)	15 (27.7%)	
Schwannoma/ acoustic neuroma	4 (3.1%)	1 (1.3%)	4 (7.4%)	
Other	30 (23.4%)	24 (32.4%)	6 (11.1%)	
Tumour location:				
Supratentorial	97 (75.8%)	58 (77.3%)	39 (73.6%)	0.744
Posterior fossa	17 (13.3%)	8 (10.7%)	9 (17.0%)	
Ventricular	9 (7.0%)	6 (8.0%)	3 (5.7%)	
Brainstem	5 (3.9%)	3 (4.0%)	2 (3.8%)	
Preoperative neurological deficit (yes)	75 (58.6%)	26 (44.1%)	49 (71.0%)	0.002
Charlson Comorbidity Index	2 (0–6)	1 (0–5)	2 (0–6)	0.062
APACHE II score	16.03±8.32	12.77±6.27	18.81±8.87	<0.001

ICU: Intensive care unit, LOS: Length of stay, APACHE II: Acute Physiology and Chronic Health Evaluation II

Table 2. ICU-related complications according to ICU length of stay

Complication	Total (n=128)	ICU LOS ≤3 days	ICU LOS >3 days	p value
Ventriculoperitoneal shunt	31 (24.2%)	4 (6.8%)	26 (37.7%)	<0.001
Neurological deficit	48 (37.5%)	9 (15.3%)	39 (56.5%)	<0.001
Seizures	28 (21.9%)	1 (1.7%)	27 (39.1%)	<0.001
Arrhythmia	53 (41.4%)	11 (18.6%)	42 (60.9%)	<0.001
Respiratory complications	49 (38.3%)	5 (8.5%)	44 (63.8%)	<0.001
Metabolic complications	37 (28.9%)	4 (6.8%)	33 (47.8%)	<0.001
Hemorrhage	22 (17.2%)	3 (5.1%)	19 (27.5%)	<0.001

ICU: Intensive care unit, LOS: Length of stay

Factors Associated with Prolonged ICU Length of Stay

In univariate analyses, APACHE II score, respiratory complications, mechanical ventilation requirement, and duration of mechanical ventilation were significantly associated with prolonged ICU stay. In the multivariable logistic regression analysis, the APACHE II score was the independent predictor of prolonged ICU stay (OR 0.924, 95% CI 0.862–0.991; p=0.027). In addition, respiratory complications (OR 0.109, 95% CI 0.029–0.408; p=0.001) and duration of mechanical ventilation (OR 0.307, 95% CI 0.151–0.625; p=0.001) remained significantly associated with ICU LOS in the multivariable model. Other variables, including age, sex, tumor characteristics, Charlson Comorbidity Index, and intraoperative vasopressor use, were not independently associated with prolonged ICU stay. Detailed univariate and multivariable regression results are shown in Table 4.

DISCUSSION

This retrospective, single-center study evaluated factors associated with ICU LOS and mortality in postoperative patients with intracranial tumors. The main findings indicate that the APACHE II score is an independent predictor of prolonged ICU stay. The need for mechanical ventilation, its duration, and respiratory complications also significantly affected both mortality and the LOS. In contrast, age, gender, and the tumor's anatomical characteristics (type and location) were not significantly associated with these outcomes.

Prolonged ICU stays are associated not only with increased mortality and complication risks but also with a significant cost burden on the healthcare system. This underscores that prolonged hospital stays are a critical issue, both clinically and economically, in centers with limited intensive care resources. Moitra et al.[9] demonstrated that hospital stays exceeding seven days in intensive care account for a significant proportion of total bed days and that this patient group also incurs higher costs for long-term care, rehabilitation, and readmissions. When these data are considered alongside our finding that prolonged ICU stays are associated with mortality, it suggests that the LOS in intensive care should be regarded as both a clinical and economic quality indicator.

In our study, the finding that the APACHE II score was independently associated with prolonged ICU stay suggests that ICU duration is influenced not only by surgical or tumor-related factors but also by indicators of acute physiologic deterioration and overall systemic disease severity. In line with this, Decavèle et al.[14] reported that mortality among patients admitted to intensive care for primary malignant brain tumors was primarily determined by indicators of acute clinical severity, including the need for mechanical ventilation, respiratory parameters, and the GCS. Moreover, the strong association observed between mechanical ventilation requirements and the duration of ventilation with mortality in our cohort is consistent with previous neurointensive care studies, which have identified mechanical ventilation as one of the most powerful predictors of poor prognosis. Collectively, these findings suggest that mechanical ventilation represents not merely a supportive intervention but also a surrogate marker of a high-risk clinical phenotype in postoperative intracranial tumor patients.

Our study found that respiratory complications were associated with both prolonged ICU stays and mortality, highlighting the importance of systemic complications in patients with postoperative intracranial tumors. de Almeida et al.[11] reported that respiratory failure and seizures were among the most common reasons for requiring transfer to intensive care in patients following elective intracranial surgery. These complications in the early postoperative period have been identified as key factors prolonging intensive care and worsening prognosis. Our findings are consistent with these reports in the literature.

Table 4. Univariate and multivariate logistic regression analysis of factors associated with length of stay

Variable	Univariate p	Multivariate B	Multivariate OR	95% CI for OR	Multivariate p
Age (years)	0.318	-0.012	0.988	0.962–1.015	0.375
Sex (male/female)	0.269	-0.467	0.627	0.272–1.448	0.275
Glasgow Coma Scale	0.929	-0.003	0.997	0.886–1.121	0.958
Tumor type	0.066	0.115	1.122	0.989–1.271	0.073
Tumor localization	0.332	-0.033	0.967	0.910–1.029	0.290
Preoperative neurological deficit	0.158	-0.647	0.524	0.226–1.213	0.131
Charlson Comorbidity Index	0.144	-0.248	0.781	0.570–1.069	0.123
APACHE score	0.024	-0.079	0.924	0.862–0.991	0.027
Ventriculoperitoneal shunt	0.677	0.083	1.086	0.190–6.198	0.926
Neurological deficit	0.583	-0.248	0.780	0.230–2.640	0.690
Seizure	0.116	-2.209	0.110	0.010–1.146	0.065
Arrhythmia	0.229	-0.676	0.509	0.143–1.805	0.296
Respiratory complication	<0.001	-2.212	0.109	0.029–0.408	0.001
Metabolic complication	0.421	0.528	1.696	0.269–10.674	0.574
Hemorrhage	0.595	-0.622	0.537	0.096–2.996	0.478
Mechanical ventilation	0.015	0.005	1.005	0.382–2.644	0.992
Duration of mechanical ventilation	<0.001	-1.180	0.307	0.151–0.625	0.001

OR: Odds ratio, CI: Confidence interval, APACHE: Acute Physiology and Chronic Health Evaluation

The association between prolonged ICU stay and poor clinical outcomes is not unique to neurosurgical patients. Although our study did not evaluate long-term mortality, the finding that prolonged ICU stay is associated with mortality is consistent with the general intensive care literature and suggests that ICU LOS may be a strong prognostic indicator.

An increasing number of studies suggest that it may not always be necessary to routinely admit all patients to intensive care after elective intracranial tumor surgery. Phillips et al.[6] reported that prolonged hospital stays after craniotomy are largely attributable to modifiable processes (delays in postoperative imaging, rehabilitation, and discharge planning) and that the LOS can be reduced through appropriate patient selection. This approach, when considered alongside our finding that prolonged ICU stays are associated with specific clinical risk profiles, suggests that intensive care resources could be used more rationally in selected patient groups.

On the other hand, the importance of preoperative physiological reserve is increasingly emphasized. Thommen et al.[7] demonstrated in their large multicenter analysis that frailty is a strong independent predictor of mortality, complications, and prolonged hospital stay after craniotomy, independent of age. Although we did not use the frailty score directly in our study, the association of scores reflecting acute illness severity, such as APACHE II, with prolonged ICU stay supports the notion that the concept of physiological reserve plays a decisive role in intensive care outcomes.

Tumor type was associated with ICU LOS in the univariate analysis; however, this association did not persist after adjustment for clinical severity parameters, indicating that physiological derangement and postoperative course may outweigh tumor-related characteristics in determining ICU outcomes. Consistent with this, Decavèle et al.[14] reported that intensive care mortality is more closely associated with the reason for ICU admission and the life-support therapies administered than with tumor type.

Limitations

This study has certain limitations. The retrospective, single-center design is subject to selection bias and limited in its ability to establish causal relationships. Furthermore, the inability to include tumor resection grade, intraoperative hemodynamic variables, and detailed histopathologic data in the analysis limited the evaluation of certain factors that could influence intensive care duration and mortality. However, the patient population spanning a long time period and the detailed intensive care data are among the study's strengths.

CONCLUSION

As a result, the APACHE II score is an independent predictor of prolonged ICU stay in patients with postoperative intracranial tumors. The need for mechanical ventilation, ventilation duration, and respiratory complications significantly affect both LOS and mortality. When considered alongside the literature, these findings provide clinical clues that could help identify high-risk patients early and use intensive care resources more cost-effectively and selectively.

ETHICAL DECLARATIONS

Ethics Committee Approval

Ethical approval for this study was obtained from the Non-interventional Researches Ethics Committee of Fırat University (Date: 05.01.2026, Decision No: E-50716828-020-755356).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

The author confirms sole responsibility for the study conception, design, data collection, analysis, interpretation, and manuscript preparation. All aspects of the work were carried out by the author.

REFERENCES

1. Bonneville F, Jäger HR, Smirniotopoulos JG. Differential diagnosis of intracranial masses. In: Hodler J, Kubik-Huch RA, Roos JE, eds. Diseases of the Brain, Head and Neck, Spine. 2024-2027: Diagnostic Imaging. Cham, Switzerland: Springer; 2024: Chapter 8.
2. Faraj CA, Snyder RI, McCutcheon IE. Intracranial emergencies in neurosurgical oncology: pathophysiology and clinical management. *Emerg Cancer Care*. 2022;1:13. doi:10.1186/s44201-022-00013-6
3. Narvaez-Rojas AR, Mo-Carrascal J, Maraby J, et al. Neurocritical care of intracranial brain tumor surgery: an overview. *MAMC J Med Sci*. 2018;4(1):4-11. doi:10.4103/mamcjms.mamcjms_79_17
4. Lonjaret L, Guyonnet M, Berard E, et al. Postoperative complications after craniotomy for brain tumor surgery. *Anaesth Crit Care Pain Med*. 2017;36(4):213-218. doi:10.1016/j.accpm.2016.06.012
5. Agboola OO, Idowu OO, Balogun JA. Postoperative intensive care unit admission for elective brain tumour surgeries: a Nigerian neurosurgical unit experience. *Ann Ib Postgrad Med*. 2023;21(2):44-52.
6. Phillips KR, Enriquez-Marulanda A, Mackel C, et al. Predictors of extended length of stay related to craniotomy for tumor resection. *World Neurosurg*. 2023;19:100176. doi:10.1016/j.wnsx.2023.100176
7. Thommen R, Kazim SF, Rumalla K, et al. Preoperative frailty measured by risk analysis index predicts complications and poor discharge outcomes after brain tumor resection in a large multicenter analysis. *J Neurooncol*. 2022;160:763-772. doi:10.1007/s11060-022-04212-3
8. Azumi M, Mizobuchi Y, Nakanishi N, et al. Value of the Acute Physiology and Chronic Health Evaluation II score in predicting hospital mortality for postoperative brain tumor patients in intensive care units in Japan: a retrospective case-control study. *Clin Neurol Neurosurg*. 2024;244:108435. doi:10.1016/j.clineuro.2024.108435
9. Moitra VK, Guerra C, Linde-Zwirble WT, Wunsch H. Relationship between ICU length of stay and long-term mortality for elderly ICU survivors. *Crit Care Med*. 2016;44(4):655-662. doi:10.1097/CCM.0000000000001480

10. Neumann JO, Schmidt S, Nohman A, Jakobs M, Unterberg A. Routine ICU admission after brain tumor surgery: retrospective validation and critical appraisal of two prediction scores. *Acta Neurochir (Wien).* 2023; 165(6):1655-1664. doi:10.1007/s00701-023-05592-9
11. de Almeida CC, Boone MD, Laviv Y, et al. The utility of routine intensive care admission for patients undergoing intracranial neurosurgical procedures: a systematic review. *Neurocrit Care.* 2018;28:35-42. doi:10.1007/s12028-017-0433-4
12. De la Garza-Ramos R, Kerezoudis P, Tamargo RJ, Brem H, Huang J, Bydon M. Surgical complications following malignant brain tumor surgery: an analysis of 2002–2011 data. *Clin Neurol Neurosurg.* 2016; 140:6-10. doi:10.1016/j.clineuro.2015.11.005
13. Nuño M, Carico C, Mukherjee D, et al. Association between in-hospital adverse events and mortality for patients with brain tumors. *J Neurosurg.* 2015;123:1247-1255. doi:10.3171/2014.10.JNS141516
14. Decavèle M, Weiss N, Rivals I, et al. Prognosis of patients with primary malignant brain tumors admitted to the intensive care unit: a two-decade experience. *J Neurol.* 2017;264:2303-2312. doi:10.1007/s00415-017-8624-7
15. Tabouret E, Boucard C, Devillier R, et al. Neuro-oncological patients admitted in intensive-care unit: predictive factors and functional outcome. *J Neurooncol.* 2016;127:111-117. doi:10.1007/s11060-015-2015-7
16. Cho DY, Wang YC. Comparison of the APACHE III, APACHE II, and Glasgow Coma Scale in acute head injury for prediction of mortality and functional outcome. *Intensive Care Med.* 1997;23:77-84. doi:10.1007/s001340050294
17. Schuss P, Lehmann F, Schäfer N, et al. Postoperative prolonged mechanical ventilation in patients with newly diagnosed glioblastoma—an unrecognized prognostic factor. *Front Oncol.* 2020;10:607557. doi:10.3389/fonc.2020.607557
18. Schuss P, Schäfer N, Bode C, et al. The impact of prolonged mechanical ventilation on overall survival in patients with surgically treated brain metastases. *Front Oncol.* 2021;11:658949. doi:10.3389/fonc.2021.658949
19. Cote DJ, Smith TR. Venous thromboembolism in brain tumor patients. *J Clin Neurosci.* 2016;25:13-18. doi:10.1016/j.jocn.2015.05.053
20. Gordon HS, Rosenthal GE. The relationship of gender and in-hospital death: increased risk of death in men. *Med Care.* 1999;37:318-324. doi:10.1097/00005650-199903000-00011

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Case Report

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Our experience with serratus posterior superior intercostal plane block in postoperative analgesia of shoulder arthroscopy: a case report

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ABSTRACT

Postoperative pain management after shoulder arthroscopy is crucial for early mobilization and patient satisfaction. Although the interscalene brachial plexus block remains the gold standard for postoperative analgesia, it may be unsuitable in patients with pulmonary comorbidities due to its high incidence of hemidiaphragmatic paralysis. The serratus posterior superior intercostal plane (SPSIP) block is a newly described interfascial plane block that provides cervicothoracic analgesia while avoiding motor blockade. We present a case in which SPSIP block provided effective postoperative analgesia for shoulder arthroscopy in a patient with asthma and coronary artery disease.

Keywords: Shoulder arthroscopy, serratus posterior superior intercostal plane block, postoperative analgesia, regional anesthesia

INTRODUCTION

Shoulder arthroscopy is associated with significant postoperative pain, particularly within the first 24 hours. Effective postoperative analgesia facilitates early rehabilitation and reduces opioid requirements. Although the interscalene brachial plexus block is considered the gold standard for shoulder surgery, its use may be limited in patients with reduced pulmonary reserve due to hemidiaphragmatic paralysis secondary to phrenic nerve involvement.[1-2]

In recent years, fascial plane blocks have gained attention due to their favorable safety profile and minimal motor involvement. The serratus posterior superior intercostal plane (SPSIP) block, first described by Tulgar et al.,[3] is an interfascial block targeting the cervicothoracic junction. Cadaver studies have shown that dye spread extends from C4–T7, involving dorsal rami of upper thoracic nerves, correlating with C4–T6 dermatomal sensory distribution.

Beyond shoulder surgery, SPSIP block has demonstrated effective analgesia in various procedures including breast surgery, thoracic surgery (VATS), minimally invasive cardiac surgery, clavicle surgery, posterior cervical/thoracic spinal procedures, and upper rib fractures.[4-6]

The purpose of this case report is to highlight the postoperative analgesic efficacy and clinical feasibility of SPSIP block as part of multimodal shoulder arthroscopy analgesia, particularly in a patient with pulmonary comorbidities where conventional regional techniques carry increased risk.

CASE

A 52-year-old female patient with a history of hypertension, coronary artery disease, and asthma was scheduled for elective shoulder arthroscopy. After cardiology and pulmonology evaluations, the patient was classified as ASA III.

Anesthesia Management

Standard ASA monitoring was applied. General anesthesia was induced using propofol 2 mg/kg, fentanyl 2 µg/kg, and rocuronium 0.6 mg/kg, followed by endotracheal intubation.

Maintenance consisted of sevoflurane in an oxygen–air mixture. Hemodynamics remained stable without additional intraoperative opioids. The patient was positioned in the beach-chair position for arthroscopy. Total surgical duration was 60 minutes.

SPSIP Block Technique

Approximately 30 minutes prior to induction, an ultrasound-guided SPSIP block was performed in the sitting position. A linear probe (6–13 MHz) was placed parasagittally over the second–third ribs. The trapezius, rhomboid major, and serratus posterior superior muscles were identified (Figure 1).

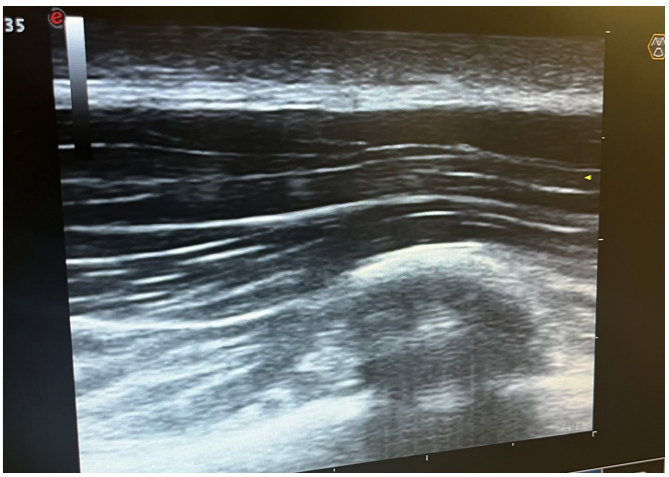

Figure 1. Ultrasound image of the serratus posterior superior intercostal plane (SPSIP) block–needle trajectory and target plane

A 22G, 100-mm needle was advanced in-plane into the fascial plane beneath the serratus posterior superior muscle. After hydrodissection, 20 ml of 0.25% bupivacaine was injected. Spread was visualized without complications (Figure 2).

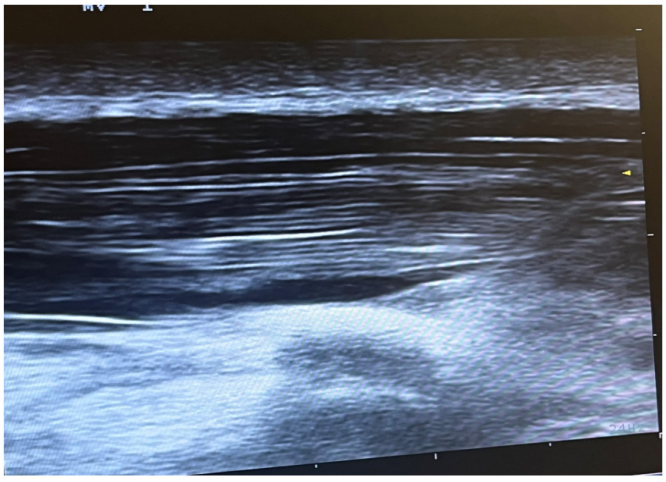

Figure 2. Distribution of local anesthetic in the SPSIP plane

Postoperative Course

At the 1st postoperative hour, the VAS score at rest was 3 with sensory involvement from C4–T6. At 8 hours, VAS remained 3. At 12 hours, the VAS score increased to 5, with regression to C5–T4. Only 1 g IV paracetamol was required every 8 hours; no rescue opioids were needed.

DISCUSSION

In this case, the interscalene block was avoided due to the patient's history of asthma, and the SPSIP block—a more superficial fascial plane block that does not cause motor blockade—was preferred. The SPSIP block is a newly described technique that aims to provide analgesia to the shoulder and upper thoracic regions by affecting the dorsal branches of the intercostal nerves and the cervicothoracic transition zone.[3] Previous studies have reported that the SPSIP block provides effective analgesia in thoracic, breast, and shoulder surgeries.[3,4]

Tulgar et al.[3] demonstrated that the SPSIP block can achieve wide dermatomal spread in thoracoscopic and shoulder surgeries and may reduce opioid consumption. In our case, the postoperative sensory block extending from C4 to T6 supports the contribution of the SPSIP block to analgesia after shoulder arthroscopy.

Cadaveric studies have shown that SPSIP achieves wide dye spread from C4–T7, supporting its use for cervicothoracic analgesia.[3] Clinical studies have demonstrated consistent dermatomal coverage from C3–T7, correlating with our patient's findings.

Interscalene block is the gold standard for shoulder surgery analgesia but results in hemidiaphragmatic paralysis in up to 70–100% of cases.[1,2] This presents a significant risk for patients with asthma, COPD, or other pulmonary limitations. Alternative techniques such as suprascapular or axillary nerve blocks provide incomplete analgesia and often require combination.[7]

Thoracic interfascial plane blocks such as erector spinae plane and paravertebral blocks are effective but carry risks such as pneumothorax, vascular puncture, or epidural spread.[8-10] The SPSIP block provides a more superficial and safer option, with minimal motor involvement and no impact on diaphragmatic function.

Recent prospective and randomized trials have confirmed the efficacy of SPSIP in diverse surgeries. A randomized controlled trial comparing SPSIP with thoracic paravertebral block in VATS demonstrated equivalent postoperative analgesia, similar opioid consumption, and comparable recovery outcomes.[5-11] Additionally, interfascial plane block literature supports SPSIP's expanding role in multimodal postoperative analgesia.[5]

In our case, SPSIP provided effective analgesia with opioid-sparing benefits and no respiratory compromise—important advantages in patients with cardiopulmonary comorbidities.

CONCLUSION

The SPSIP block is a safe and effective option for postoperative analgesia in shoulder arthroscopy, especially for patients with limited pulmonary reserve. Its wide dermatomal spread, low complication profile, and opioid-sparing potential make it a valuable alternative to traditional regional techniques.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Concept: E.B., S.F.K.; Desing: E.B.; Control: S.F.K.; Data Collection and/or Processing: E.B.; Literature Review: E.B., S.F.K.; Article Writing: E.B.; Critical Review: All authors.

REFERENCES

1. Urmey WF, Talts KH, Sharrock NE. One hundred percent incidence of hemidiaphragmatic paresis associated with interscalene brachial plexus anesthesia as diagnosed by ultrasonography. *Anesth Analg.* 1991;72(4):498-503. doi:10.1213/00000539-199104000-00014
2. Renes SH, Rettig HC, Gielen MJ, Wilder-Smith OH, van Geffen GJ. Ultrasound-guided low-dose interscalene brachial plexus block reduces the incidence of hemidiaphragmatic paresis. *Reg Anesth Pain Med*. 2009;34(5):498-502. doi:10.1097/AAP.0b013e3181b49256
3. Tulgar S, Ciftci B, Ahiskalioglu A, et al. Serratus posterior superior intercostal plane block: a technical report on the description of a novel periparavertebral block for thoracic pain. *Cureus.* 2023;15(2):e34582. doi:10.7759/cureus.34582
4. Kültüroğlu G, Altınsoy S, Özgüner Y, Çataroğlu CK. Novel serratus posterior superior intercostal plane block provided satisfactory analgesia after breast cancer surgery: two case reports. *Turk J Anaesthesiol Reanim*. 2024;52(1):33-35. doi:10.4274/TJAR.2024.231431
5. Avci O, Gundogdu O, Balci F, Tekcan MN, Ozbey M. Efficacy of serratus posterior superior intercostal plane block (SPSIPB) on post-operative pain and total analgesic consumption in patients undergoing video-assisted thoracoscopic surgery (VATS): a double-blinded randomised controlled trial. *Indian J Anaesth*. 2023;67(12):1116-1122. doi:10.4103/ija.ija_589_23
6. Dogan G, Küçük O, Kayır S, et al. Serratus posterior superior intercostal plane block versus thoracic paravertebral block for pain management after video-assisted thoracoscopic surgery: a randomized prospective study. *Brazilian J Anesthesiol*. 2025;75(5):844647.
7. Hussain N, Goldar G, Ragina N, Banfield L, Laffey JG, Abdallah FW. Suprascapular and interscalene nerve block for shoulder surgery: a systematic review and meta-analysis. *Anesthesiology.* 2017;127(6):998-1013. doi:10.1097/ALN.0000000000001894
8. Forero M, Adhikary SD, Lopez H, Tsui C, Chin KJ. The erector spinae plane block: a novel analgesic technique in thoracic neuropathic pain. *Reg Anesth Pain Med.* 2016;41(5):621-627. doi:10.1097/AAP.0000000000000451
9. Karmakar MK. Thoracic paravertebral block. *Anesthesiology.* 2001;95(3):771-780. doi:10.1097/00000542-200109000-00033
10. Arık E, Zengin M, Şeker G, et al. Is the serratus posterior superior intercostal plane block a viable alternative to the erector spinae plane block for postoperative analgesia in breast surgery? A prospective, randomized trial. *BMC Anesthesiol*. 2025;25:3531. doi:10.1186/s12871-025-03531-y
11. Celik E, Fulya L, Yazar CO, Zengin M, Ergil J. Serratus posterior superior intercostal plane block for rescue analgesia after clavicle fracture surgery: a case report. *A A Practice.* 2024;18(10):e01859. doi:10.1213/XAA.0000000000001859

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Letter to the Editor

Microbiological signals from a tertiary ICU: gram-negative dominance and the emergence of *Candida parapsilosis* in device-associated infections

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Keywords: Carbapenem-resistant, gram-negative bacteria, *Acinetobacter baumannii* complex

To the Editor,

We would like to share an observation from our tertiary anesthesia intensive care unit (ICU) surveillance records that may be relevant to clinicians facing evolving local pathogen ecology. We retrospectively reviewed healthcare-associated infection (HAI) episodes recorded in our unit between 2020 and 2024. This dataset includes only patients who developed an ICU-acquired infection; therefore, it should be interpreted as a descriptive case series of HAI episodes rather than incidence rates.

A total of 343 HAI episodes were documented among 259 patients. Device-associated infections constituted the majority of episodes in our cohort. After harmonizing terminology in the registry, the two most frequent infection groups were ventilator-associated pneumonia (VAP) (n=137) and central line–associated bloodstream infection (CLABSI) (n=130). VAP and CLABSI were identified using standardized surveillance definitions routinely applied in our institutional infection control registry. The median time from ICU admission to infection diagnosis was 18 days (IQR 10–31), suggesting that infections tended to occur in the later course of prolonged critical illness, which remains a significant challenge in global ICU settings.[1] A summary of major infection types and the most frequently isolated pathogens is presented in Table.

Table. Distribution of major infection types and frequently isolated pathogens

Category	n
Ventilator-associated pneumonia (VAP)	137
Central line–associated bloodstream infection (CLABSI)	130
Klebsiella pneumoniae	104
Acinetobacter baumannii	68
Candida parapsilosis	25

Most notably, we observed a striking predominance of Gram-negative pathogens. The most frequently reported microorganisms were *Klebsiella pneumoniae* (n=104) and *Acinetobacter baumannii* (n=68). Given the well-recognized association of these pathogens with multidrug resistance in ICU settings, our findings may have implications for local empiric therapy pathways and infection prevention strategies.[2] Furthermore, *Candida parapsilosis* (n=25) appeared as a recurrent isolate. This finding is noteworthy as *Candida parapsilosis* is frequently associated with catheter-related infections and hand-borne transmission, potentially warranting a targeted review of catheter care and parenteral nutrition processes.[3]

It should also be acknowledged that local antimicrobial policies, infection prevention practices, and temporal factors may influence pathogen distribution, and these findings should be interpreted within the context of unit-specific ICU ecology.

We acknowledge important limitations. First, due to the absence of denominators (patient-days or device-days), we cannot provide HAI incidence densities. Second, susceptibility profiles were not available in this dataset, preventing assessment of multidrug resistance patterns. Finally, the registry is episode-based; some patients

contributed multiple infection episodes. In addition, this was a single-center study, which may limit the generalizability of the findings.

Despite these limitations, we believe that sharing local microbiological "signals" from real-world ICU surveillance may help raise awareness of evolving pathogen distributions and prompt centers to periodically re-evaluate prevention bundles and empiric antimicrobial strategies based on their own ecology.

Our five-year surveillance highlights a predominance of gram-negative pathogens and the recurrent isolation of *Candida parapsilosis* in device-associated infections. These findings reinforce the value of ongoing, unit-specific microbiological surveillance to inform infection prevention efforts and empiric antimicrobial decision-making in the ICU.

ETHICAL DECLARATIONS

Peer Review Process

This letter was externally peer-reviewed.

Conflict of Interest

The author declare no conflicts of interest.

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Author Contributions

The author confirms sole responsibility for the study conception, design, data collection, analysis, interpretation, and manuscript preparation. All aspects of the work were carried out by the author.

REFERENCES

1. Vincent JL, Sakr Y, Singer M, et al. Prevalence and outcomes of infection among patients in intensive care units in 2017. *JAMA*. 2020; 323(15):1478. doi:10.1001/jama.2020.2717
2. Jean SS, Harnod D, Hsueh PR. Global threat of carbapenem-resistant gram-negative bacteria. *Front Cell Infect Microbiol*. 2022;12:823684. doi:10.3389/fcimb.2022.823684
3. Tóth R, Nosek J, Mora-Montes HM, et al. *Candida parapsilosis*: from genes to the bedside. *Clin Microbiol Rev*. 2019;32(2):e00111-18. doi:10. 1128/CMR.00111-18