



Effects of Dexmedetomidine on the Incidence of Agitation and Pain Severity after Traumatic Nasal Surgeries

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ABSTRACT

Introduction: Conventional agents such as opioids and benzodiazepines may control symptoms but can impair recovery and pose airway-related risks. Dexmedetomidine offers sedative, analgesic, and sympatholytic benefits with minimal respiratory depression. The aim of this trial was to evaluate intraoperative dexmedetomidine for reducing emergence agitation and postoperative pain after traumatic nasal surgery.

Material and methods: This prospective, double-blind randomized controlled trial at Imam Reza Hospital included 60 patients undergoing traumatic nasal surgery, randomized equally to receive either intraoperative dexmedetomidine or saline placebo. Dexmedetomidine was administered as a 1 µg/kg loading dose followed by a 0.5 µg/kg/h infusion. Primary outcomes were postoperative agitation and pain severity, while secondary outcomes included hemodynamic changes, recovery duration, rescue analgesic use, and adverse events.

Results: Dexmedetomidine significantly reduced postoperative emergence agitation and pain scores at all assessed time points compared with placebo. It also attenuated heart rate and mean arterial pressure during early recovery without affecting oxygen saturation. Although dexmedetomidine prolonged extubation time and PACU stay, it did not significantly increase major adverse events and was associated with a lower incidence of postoperative shivering.

Conclusion: In conclusion, intraoperative infusion of dexmedetomidine during traumatic nasal surgery significantly attenuates emergence agitation and mitigates postoperative pain severity, while ensuring superior hemodynamic stability in the early recovery phase.

Introduction

Traumatic nasal injuries represent one of the most prevalent presentations in maxillofacial emergency clinics, frequently requiring surgical intervention to restore both anatomical patency and facial aesthetics. Nasal fractures, whether presenting as isolated skeletal disruptions or as components of complex Naso-orbito-ethmoid injuries, are often managed through closed or open reduction techniques.

While these surgical interventions are highly effective at aligning displaced bone and cartilage, the immediate postoperative period presents substantial clinical challenges. The nasal cavity is highly vascularized and densely innervated by branches of the trigeminal nerve, making surgical manipulation in this region exceptionally painful.

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Despite advances in multimodal analgesic techniques, patients undergoing corrective nasal surgeries often experience severe postoperative pain and acute emotional distress. This clinical scenario is further complicated by the routine use of tight nasal packing, which is essential to control hemorrhage and stabilize the nasal septum but causes significant physical discomfort and forces patients to breathe exclusively through their mouths (1).

The combination of severe localized pain, nasal airway obstruction, and the disorientation associated with emergence from general anesthesia frequently culminates in emergence agitation. Emergence agitation is a well-documented post-anesthetic phenomenon characterized by confusion, irritability, combativeness, and disorientation during the immediate transition from consciousness to wakefulness. In the context of traumatic nasal surgeries, the incidence of emergence agitation is particularly high compared to other elective procedures. The sensation of suffocation induced by bilateral nasal packing triggers a state of panic, which is exacerbated by residual anesthetic agents and acute pain. When patients experience emergence agitation, they often exhibit hyperactive behaviors, including thrashing, attempts to remove intravenous lines, and pulling at nasal splints or packings. These behaviors not only threaten patient safety by increasing the risk of self-injury but also compromise the surgical outcome by causing mucosal trauma, wound dehiscence, and severe postoperative hemorrhage due to elevated systemic blood pressure (2).

Historically, the management of postoperative pain and emergence agitation in nasal surgery has relied heavily on opioid analgesics, benzodiazepines, and non-steroidal anti-inflammatory drugs. However, these pharmacological interventions carry significant limitations and potential adverse effects that can complicate patient recovery. Systemic opioids, while effective at mitigating severe pain, are associated with dose-dependent respiratory depression, which is particularly dangerous in patients whose nasal airways are completely occluded by packing. Furthermore, opioids frequently induce postoperative nausea, vomiting, and pruritus, which increase intraocular and intravenous pressure, potentially leading to postoperative bleeding at the surgical site. Benzodiazepines, on the other hand, can effectively reduce perioperative anxiety but often prolong recovery times, delay emergence, and, in some cases, paradoxically exacerbate agitation in elderly or highly stressed patient cohorts. Consequently, there is an ongoing clinical search for an adjuvant pharmacological agent that can provide effective analgesia and stable sedation without compromising respiratory drive or delaying emergence (3).

In recent years, dexmedetomidine has emerged as a promising adjuvant in perioperative medicine due to its unique pharmacological profile. As a highly selective alpha-2 adrenergic receptor agonist, dexmedetomidine exerts its effects primarily within the central nervous system, particularly within the locus coeruleus. By stimulating presynaptic alpha-2 receptors, it inhibits the release of norepinephrine, thereby reducing sympathetic tone and producing a state of conscious sedation. Unlike GABA-receptor agonists such as propofol or midazolam, dexmedetomidine-induced sedation closely mimics natural non-rapid eye movement sleep, allowing patients to be easily aroused to follow commands. Additionally, dexmedetomidine possesses intrinsic analgesic properties mediated by the stimulation of alpha-2 receptors in the dorsal horn of the spinal cord, which inhibits the transmission of nociceptive signals. Crucially, even at higher sedative doses, dexmedetomidine does not cause significant respiratory depression, making it an attractive candidate for patients undergoing procedures that compromise the airway, such as nasal surgery (4).

The clinical utility of dexmedetomidine in reducing postoperative emergence agitation has been demonstrated across various surgical specialties, yet its specific efficacy in traumatic nasal surgeries remains a subject of ongoing investigation. Traumatic nasal surgeries differ from elective rhinoplasties because the tissue undergoes acute inflammatory changes, pre-existing localized edema, and varying degrees of nerve injury prior to surgical incision. The baseline inflammatory state of traumatic tissue may alter the threshold for pain perception and heighten the patient's susceptibility to postoperative delirium and agitation. Although previous clinical trials have evaluated the effects of dexmedetomidine in elective nasal and sinus surgeries, the results cannot be directly extrapolated to the acute trauma population, where psychological distress, sudden airway changes, and pre-operative pain levels are significantly more pronounced (5).

Furthermore, the optimal dosing regimen and timing of dexmedetomidine administration to balance efficacy with cardiovascular stability remain topics of clinical debate. Dexmedetomidine is known to cause dose-dependent cardiovascular effects, primarily bradycardia and transient hypotension, resulting from its sympatholytic action. While these effects are generally mild and self-limiting in healthy individuals, they require careful monitoring, particularly in trauma patients who may have experienced hemodynamic fluctuations. Administering dexmedetomidine as a continuous intraoperative infusion versus a single bolus at the end of surgery represents two distinct clinical strategies. Determining which method provides superior control over emergence agitation and postoperative pain while minimizing the incidence of bradycardia is essential for refining clinical

protocols in maxillofacial and ENT surgical units (6).

Despite the theoretical advantages of dexmedetomidine, there is a lack of consensus regarding its impact on the longitudinal severity of postoperative pain and the consumption of rescue analgesics in the post-anesthesia care unit (PACU) and the early ward period. Some studies suggest that the analgesic sparing effect of dexmedetomidine is short-lived, while others report a sustained reduction in pain scores up to twenty-four hours postoperatively. Investigating the clinical course of pain severity and agitation incidence in patients undergoing reconstructive procedures for nasal trauma is vital for designing effective multimodal recovery pathways. Clarifying these dynamics can help clinicians reduce the reliance on postoperative opioids, facilitate earlier discharge from the PACU, and improve overall patient satisfaction scores (7). To address these clinical questions, systematic research comparing dexmedetomidine to standard anesthetic regimens in traumatic nasal surgeries is warranted. By focusing specifically on the trauma demographic, researchers can control for the confounding variables associated with chronic nasal disease and elective cosmetic alterations. The primary objective of this randomized clinical trial was to investigate the effects of intraoperative dexmedetomidine administration on the incidence of emergence agitation and the severity of postoperative pain in patients undergoing corrective surgery for traumatic nasal injuries.

Material and methods

Study Design and Setting:

This study was designed as a prospective, randomized, double-blind, placebo-controlled clinical trial. The study was conducted at Imam Reza Hospital, affiliated with Tabriz University of Medical Sciences, Tabriz, Iran. Eligible patients undergoing surgery for traumatic nasal injuries were randomly allocated into two equal groups: the dexmedetomidine group and the placebo/control group. The study was performed under double-blind conditions, meaning that the patients, surgeons, anesthesiologists involved in intraoperative management, postoperative evaluators, and data analysts were unaware of the group assignments. Randomization was performed before the intervention, and the study medications were prepared in identical syringes by an independent person who was not involved in patient management or outcome assessment.

Sample Size and Sampling Method

The sample size was estimated using the standard sample size formula for comparing two independent groups. Based on the expected difference in the incidence of postoperative agitation between the intervention and control groups, with a confidence

level of 95% and a study power of 80%, the required sample size was calculated as 30 patients in each group. Therefore, a total of 60 patients were included in the study, with 30 patients assigned to the dexmedetomidine group and 30 patients assigned to the control group. Participants were recruited using a convenience sampling method from patients referred to Imam Reza Hospital for surgical management of traumatic nasal injuries. After confirming eligibility and obtaining written informed consent, patients were randomly assigned to one of the two study groups.

Eligibility Criteria

Patients were included in the study if they were candidates for traumatic nasal surgery under general anesthesia, were within the eligible adult age range, had an American Society of Anesthesiologists physical status of I or II, were able to understand the study procedures and postoperative pain assessment scales, and provided written informed consent to participate in the trial. Patients were excluded if they had a known history of hypersensitivity or contraindication to dexmedetomidine, significant cardiovascular disease such as severe bradycardia, conduction abnormalities, uncontrolled hypertension, or heart failure, severe hepatic or renal dysfunction, neurological or psychiatric disorders that could interfere with postoperative agitation assessment, chronic use of sedatives, opioids, or psychoactive medications, substance abuse, pregnancy or lactation, obstructive sleep apnea or severe respiratory disease, intraoperative complications requiring major deviation from the anesthetic protocol, or refusal to continue participation at any stage of the study.

Intervention and Study Procedure

After arrival in the operating room, all patients underwent standard monitoring, including electrocardiography, non-invasive blood pressure measurement, pulse oximetry, and routine anesthetic monitoring. Baseline hemodynamic variables, including heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and oxygen saturation, were recorded before induction of anesthesia. General anesthesia was induced using a standardized anesthetic protocol for all patients to minimize confounding effects. After induction and airway management, patients in the dexmedetomidine group received an intravenous loading dose of dexmedetomidine at 1 microgram/kg administered slowly over 10 minutes, followed by a continuous infusion at a rate of 0.5 microgram/kg/hour until the end of surgery. In the control group, patients received an equal volume of normal saline as placebo, prepared in the same manner and administered using the same timing and infusion protocol as the dexmedetomidine group. The placebo syringes were identical in appearance

to the dexmedetomidine syringes to preserve blinding. The surgical procedure, anesthetic management, and postoperative care were kept as similar as possible between the two groups. At the end of surgery, anesthetic agents were discontinued, neuromuscular blockade was reversed according to routine practice, and patients were extubated after meeting standard clinical criteria for safe extubation. The variables assessed in this study included demographic characteristics, duration of surgery, duration of anesthesia, hemodynamic parameters, postoperative agitation, postoperative pain intensity, recovery characteristics, need for rescue analgesics, and postoperative complications. The primary outcome variables were the incidence and severity of postoperative agitation and the severity of postoperative pain after traumatic nasal surgery. Postoperative agitation was evaluated in the post-anesthesia care unit using standard agitation or sedation assessment scales, such as the Richmond Agitation-Sedation Scale or the Riker Sedation-Agitation Scale, at predetermined intervals after extubation. Postoperative pain intensity was assessed using the Visual Analog Scale, in which 0 indicates no pain and 10 indicates the worst imaginable pain. Pain scores were recorded during the recovery period and at selected postoperative time points. Secondary outcome variables included changes in heart rate and blood pressure during and after surgery, time to emergence from anesthesia, duration of stay in the post-anesthesia care unit, total requirement for rescue analgesic medication, and the frequency of adverse events such as bradycardia, hypotension, respiratory depression, nausea, vomiting, excessive sedation, or any other drug-related complications. Rescue analgesia was administered according to the hospital's routine postoperative pain management protocol when patients reported moderate to severe pain or when clinically indicated.

Statistical Analysis

Data were analyzed using SPSS software version 26. Quantitative variables were evaluated for normal distribution using the Kolmogorov-Smirnov test.

Since the data followed a normal distribution, continuous variables were reported as mean and standard deviation, and comparisons between the two groups were performed using the independent samples t-test. Repeated measurements, such as hemodynamic variables and pain scores assessed at different time points, were analyzed using repeated measures analysis of variance. Qualitative variables, including sex, incidence of postoperative agitation, and frequency of complications, were reported as frequency and percentage and compared using the Chi-square test or Fisher's exact test when appropriate. A p-value less than 0.05 was considered statistically significant.

Ethical Considerations

This study was approved by the Ethics Committee of Tabriz University of Medical Sciences (IR.TBZMED.REC.1402.971) and registered in the Iranian Clinical Trials Registry (IRCT20190325043107N37). The research objectives were explained in simple language to the participants, and they signed the informed consent form. No costs were charged to the participants for their involvement in the study. Participation was completely voluntary, and all participants were informed that they could withdraw from the study at any time without any effect on their treatment process. Patient information was kept confidential, and all collected data were used only for research purposes.

Results

Baseline demographic and perioperative characteristics were comparable between the dexmedetomidine and control groups, with no statistically significant between-group differences observed in age ($p=0.783$), sex distribution ($p=0.781$), body weight ($p=0.817$), BMI ($p=0.810$), ASA physical status ($p=0.781$), procedure type ($p=0.793$), surgical duration ($p=0.846$), anesthetic duration ($p=0.822$), baseline heart rate ($p=0.727$), or baseline mean arterial pressure ($p=0.735$) (table 1).

Table 1. Baseline demographic and clinical characteristics of patients in the dexmedetomidine and control groups

Variable	Dexmedetomidine (n=30)	Control (n=30)	p-value
Age (years)	32.40 ± 9.68	33.10 ± 10.02	0.783
Sex (male/female), n	21/9	20/10	0.781
Weight (kg)	74.20 ± 10.35	73.60 ± 9.90	0.817
BMI (kg/m ²)	24.58 ± 2.71	24.41 ± 2.65	0.810
ASA physical status (I/II), n	22/8	21/9	0.781
Type of procedure (closed/open reduction), n	19/11	18/12	0.793
Duration of surgery (min)	47.60 ± 12.14	48.20 ± 11.86	0.846
Duration of anesthesia (min)	63.90 ± 13.52	64.70 ± 14.06	0.822
Baseline heart rate (bpm)	78.30 ± 9.12	79.10 ± 8.85	0.727
Baseline mean arterial pressure (mmHg)	92.40 ± 8.06	93.10 ± 7.89	0.735

Data are presented as mean $\pm$ SD or number (n). p-values were calculated using the independent-samples t-test for continuous variables and the chi-square test for categorical variables.

Postoperative assessment of emergence agitation using the Riker Sedation-Agitation Scale revealed that patients in the dexmedetomidine group had significantly lower sedation-agitation scores at ten minutes ($p<0.001$), twenty minutes ($p<0.001$), thirty minutes ($p<0.001$), and sixty minutes ($p=0.038$) after tracheal extubation compared to those in the control group. Furthermore, the overall incidence of

emergence agitation was significantly reduced in the dexmedetomidine cohort ($p=0.005$), which was primarily driven by a lower rate of mild to moderate agitation ($p=0.038$), whereas the reduction in severe agitation did not reach statistical significance ($p=0.076$). Consequently, a significantly smaller proportion of patients in the dexmedetomidine group required the administration of postoperative rescue pharmacological sedation to manage hyperactive behaviors in the post-anesthesia care unit ($p=0.023$) (table 2).

Table 2. Comparison of postoperative emergence agitation scores between the study groups

Variable	Dexmedetomidine (n=30)	Control (n=30)	p-value
Riker Sedation-Agitation Scale (SAS)	-	-	-
SAS at 10 min after extubation	3.20 $\pm$ 0.61	4.80 $\pm$ 0.96	<0.001
SAS at 20 min after extubation	3.10 $\pm$ 0.48	4.27 $\pm$ 0.83	<0.001
SAS at 30 min after extubation	3.03 $\pm$ 0.18	3.63 $\pm$ 0.61	<0.001
SAS at 60 min after extubation	3.00 $\pm$ 0.00	3.13 $\pm$ 0.35	0.038
Emergence Agitation Status	-	-	-
Incidence of emergence agitation (SAS $\geq$ 5), n (%)	2 (6.67%)	11 (36.67%)	0.005
Mild to moderate agitation (SAS = 5), n (%)	2 (6.67%)	8 (26.67%)	0.038
Severe agitation (SAS $\geq$ 6), n (%)	0 (0.00%)	3 (10.00%)	0.076
Need for postoperative rescue sedation, n (%)	1 (3.33%)	7 (23.33%)	0.023

Data are presented as mean $\pm$ SD or number (n) followed by percentage (%). p-values were calculated using the independent-samples t-test for continuous scores and the chi-square test or Fisher's exact test for categorical incidence rates.

Postoperative pain intensity was consistently lower in the dexmedetomidine group compared with the control group throughout the assessment period. Patients receiving dexmedetomidine demonstrated significantly reduced VAS scores at ten minutes

($p<0.001$), twenty minutes ($p<0.001$), thirty minutes ($p<0.001$), and sixty minutes ($p<0.001$) after tracheal extubation. This analgesic benefit remained statistically significant at two hours ($p<0.001$), six hours ($p<0.001$), twelve hours ($p<0.001$), and twenty-four hours ($p<0.001$) postoperatively, indicating a sustained reduction in postoperative pain severity among patients treated with dexmedetomidine (table 3).

Table 3. Comparison of postoperative pain intensity between the study groups

Variable	Dexmedetomidine (n=30)	Control (n=30)	p-value
VAS score at 10 min after extubation	2.40 $\pm$ 0.77	4.13 $\pm$ 1.04	<0.001
VAS score at 20 min after extubation	2.27 $\pm$ 0.69	3.87 $\pm$ 0.97	<0.001
VAS score at 30 min after extubation	2.10 $\pm$ 0.61	3.43 $\pm$ 0.82	<0.001
VAS score at 60 min after extubation	1.93 $\pm$ 0.58	3.10 $\pm$ 0.76	<0.001
VAS score at 2 h postoperatively	2.17 $\pm$ 0.65	3.37 $\pm$ 0.81	<0.001
VAS score at 6 h postoperatively	2.43 $\pm$ 0.73	3.67 $\pm$ 0.88	<0.001
VAS score at 12 h postoperatively	2.70 $\pm$ 0.79	3.83 $\pm$ 0.91	<0.001
VAS score at 24 h postoperatively	2.13 $\pm$ 0.68	2.87 $\pm$ 0.78	<0.001

Data are presented as mean $\pm$ SD. Pain intensity was assessed using the Visual Analog Scale (VAS), ranging from 0 to 10. p-values were calculated using the independent-samples t-test.

Postoperative hemodynamic assessment demonstrated that patients in the dexmedetomidine

group had significantly lower heart rate and mean arterial pressure values during the early recovery period compared with the control group, indicating greater hemodynamic attenuation after surgery ($p<0.001$). This pattern remained consistent from admission to the post-anesthesia care unit through the subsequent monitoring intervals ($p<0.001$). In

contrast, peripheral oxygen saturation remained comparable between the two groups throughout the postoperative observation period, and no

statistically significant difference was identified at any measured time point ($p>0.050$) (table 4).

Table 4. Comparison of postoperative hemodynamic parameters between the study groups

Variable	Dexmedetomidine (n=30)	Control (n=30)	p-value
Heart rate (beats/min)	-	-	-
At PACU admission	72.43 ± 6.18	81.57 ± 7.24	<0.001
10 min after admission	70.87 ± 5.94	79.93 ± 6.88	<0.001
20 min after admission	69.90 ± 5.63	77.80 ± 6.41	<0.001
30 min after admission	70.23 ± 5.71	76.67 ± 6.20	<0.001
Mean arterial pressure (mmHg)	-	-	-
At PACU admission	84.17 ± 7.03	92.60 ± 8.11	<0.001
10 min after admission	82.73 ± 6.74	90.83 ± 7.52	<0.001
20 min after admission	81.96 ± 6.40	89.37 ± 7.08	<0.001
30 min after admission	82.50 ± 6.28	88.70 ± 6.95	<0.001
Oxygen saturation (%)	-	-	-
At PACU admission	98.13 ± 0.97	97.80 ± 1.03	0.201
10 min after admission	98.27 ± 0.87	97.93 ± 0.98	0.159
20 min after admission	98.33 ± 0.84	98.00 ± 0.95	0.158
30 min after admission	98.40 ± 0.77	98.07 ± 0.87	0.124

Data are presented as mean ± SD. p-values were calculated using the independent-samples t-test. PACU: post-anesthesia care unit.

Analysis of recovery profiles and adverse events revealed that patients receiving dexmedetomidine experienced a statistically significant increase in both the time to tracheal extubation ($p<0.001$) and the total duration of post-anesthesia care unit stay ($p=0.005$) compared to patients in the control group. Regarding postoperative adverse events, the

incidence of bradycardia ($p=0.237$), hypotension ($p=0.999$), postoperative nausea and vomiting ($p=0.201$), and respiratory depression ($p=0.999$) did not show statistically significant differences between the two study cohorts. However, the occurrence of postoperative shivering was significantly lower in the dexmedetomidine group than in the control group ($p=0.026$), indicating a protective clinical benefit against hypothermia-associated shaking (table 5).

Table 5. Comparison of postoperative recovery times and adverse events between the study groups

Variable	Dexmedetomidine (n=30)	Control (n=30)	p-value
Recovery Intervals (min)	-	-	-
Time to extubation	12.13 ± 2.48	9.87 ± 2.13	<0.001
PACU stay duration	48.67 ± 8.54	42.43 ± 7.91	0.005
Postoperative Adverse Events	-	-	-
Bradycardia (HR < 50 beats/min), n (%)	3 (10.00%)	0 (0.00%)	0.237
Hypotension (MAP decrease > 20%), n (%)	2 (6.67%)	1 (3.33%)	0.999
Postoperative nausea and vomiting, n (%)	4 (13.33%)	9 (30.00%)	0.201
Respiratory depression (RR < 10 breaths/min), n (%)	1 (3.33%)	0 (0.00%)	0.999
Shivering, n (%)	1 (3.33%)	8 (26.67%)	0.026

Data are presented as mean ± SD or number (n) followed by percentage (%). p-values were calculated using the independent-samples t-test for continuous recovery intervals, and the chi-square test or Fisher's exact test for categorical adverse event rates. PACU: post-anesthesia care unit; HR: heart rate; MAP: mean arterial pressure; RR: respiratory rate.

Discussion

The primary objective of this randomized clinical trial was to evaluate the clinical efficacy and safety of intraoperative dexmedetomidine infusion on the incidence of emergence agitation, postoperative pain severity, hemodynamic stability, and recovery characteristics in patients undergoing traumatic nasal surgeries. The clinical findings of this study demonstrate that the administration of dexmedetomidine leads to a significant attenuation of emergence agitation, reducing both the overall

incidence and the severity of hyperactive recovery behaviors. Furthermore, patients receiving dexmedetomidine experienced a substantial reduction in postoperative pain intensity across all measured intervals, alongside improved hemodynamic stability, as evidenced by lower heart rates and mean arterial pressure values without compromised oxygenation. Although the intervention slightly prolonged the time to extubation and the duration of the post-anesthesia care unit stay, it did not increase the rate of adverse clinical events and provided a notable protective effect against postoperative shivering (8).

To understand the significant reduction in emergence agitation observed in the dexmedetomidine group, it is necessary to examine the agent's receptor-specific pharmacology. Dexmedetomidine is a highly selective alpha-2 adrenergic receptor agonist with an affinity for these receptors that is significantly higher than that of clonidine. The sedative and anxiolytic effects of this drug are principally mediated by its action on pre- and postsynaptic alpha-2 receptors within the locus coeruleus of the brainstem, which is the primary noradrenergic nucleus in the central nervous system. By stimulating these receptors, dexmedetomidine inhibits the release of norepinephrine, thereby terminating the propagation of excitatory signals and producing a state of sedation that closely resembles natural non-rapid eye movement sleep. In the context of emergence from general anesthesia, this central sympatholytic action prevents the sudden, disorganized surge of catecholamines that typically triggers agitation, combative behaviors, and emotional distress as patients regain consciousness (9).

The sustained analgesic efficacy observed in the dexmedetomidine group throughout the twenty-four-hour postoperative period can be explained by both central and peripheral antinociceptive mechanisms. Centrally, dexmedetomidine acts on alpha-2 adrenergic receptors located within the locus coeruleus and the dorsal horn of the spinal cord. In the spinal cord, activation of these receptors, particularly the alpha-2A subtype, inhibits the presynaptic release of primary nociceptive neurotransmitters such as substance P and glutamate from C-fibers, while simultaneously hyperpolarizing postsynaptic projection neurons. This dual pre- and postsynaptic inhibition effectively blocks the transmission of ascending pain signals to the thalamus and cerebral cortex. Furthermore, by reducing systemic catecholamine levels and modulating descending inhibitory pain pathways, dexmedetomidine synergizes with volatile anesthetics and opioids, thereby lowering the cumulative requirement for rescue analgesia and providing a continuous, stable pain-relief profile (10).

The hemodynamics observed in the post-anesthesia care unit reflect the systemic sympatholytic properties of dexmedetomidine. Traumatic nasal surgeries are associated with intense noxious stimulation, airway manipulation, and mucosal irritation, all of which trigger reflex sympathetic activation characterized by tachyarrhythmias and systemic hypertension. Dexmedetomidine counteracts these hyperdynamic states by reducing central sympathetic outflow and decreasing the concentration of circulating epinephrine and norepinephrine. The reduction in heart rate is primarily driven by the suppression of sympathetic tone at the sinoatrial node combined with an increase in vagal activity, while the reduction in mean arterial pressure stems from a decrease in systemic vascular resistance. Importantly, these hemodynamic adjustments remained within safe physiological boundaries because the slow, titrated infusion rate avoided the transient systemic hypertension that can occur with rapid bolus administration due to the stimulation of peripheral vascular alpha-2B receptors (11).

Regarding the recovery intervals, the slight prolongation in the time to tracheal extubation and post-anesthesia care unit stay observed in the intervention group is a direct consequence of the pharmacokinetics and sedative profile of dexmedetomidine. Because dexmedetomidine produces a cooperative, easy-to-arouse sedation rather than deep respiratory depression, it does not compromise ventilatory drive, as confirmed by the equivalent oxygen saturation levels between the study groups. However, the residual concentration of the drug at the end of surgery, combined with its elimination half-life of approximately two hours, inevitably delays the return of full cognitive alert status. This mild prolongation of the immediate recovery phase is a clinically acceptable trade-off for the substantial reduction in the risk of emergence agitation and severe pain, both of which can cause nasal mucosal bleeding, surgical site disruption, and accidental self-extubation (12).

The safety profile evaluated in this trial indicates that the occurrence of adverse events like bradycardia, hypotension, and respiratory depression was not statistically different between the cohorts, highlighting the safety of the dosing regimen. Conversely, the significant reduction in postoperative shivering in the dexmedetomidine group represents a valuable clinical benefit. Postoperative shivering is a highly distressing thermoregulatory response triggered by core hypothermia and the vasodilatory effects of anesthetic agents. Dexmedetomidine prevents shivering through a central mechanism by lowering both the vasoconstriction and shivering thresholds in the hypothalamus. Additionally, its action on alpha-2 receptors in the spinal cord and neuromuscular junctions suppresses the descending pathways that

generate somatic motor shaking, thereby reducing oxygen consumption, myocardial workload, and surgical wound tension (13).

When comparing these findings with the existing literature, our results align closely with previous investigations evaluating alpha-2 agonists in nasal and maxillofacial surgeries. These types of surgical interventions are particularly prone to high rates of emergence agitation due to the sensation of nasal obstruction from packing and the presence of blood in the posterior pharynx. Previous clinical trials have similarly reported that perioperative dexmedetomidine administration smooths the transition from anesthesia to consciousness by preventing the hyperactive emergence state without causing respiratory depression. While some studies have reported higher rates of clinically significant bradycardia with dexmedetomidine, our study did not demonstrate a statistical difference, which can be attributed to our conservative dosing protocol and the exclusion of patients with pre-existing conduction abnormalities (14).

This study has several limitations that should be acknowledged. First, the sample size of sixty patients, while statistically powered to detect differences in primary outcomes like pain and agitation, may not be large enough to detect rare adverse events or subtle differences in secondary outcomes. Second, because the study was conducted at a single tertiary academic medical center, the generalizability of these findings to other clinical settings or diverse patient demographics may be limited. Third, we did not measure plasma catecholamine concentrations, which would have provided direct biochemical validation of the sympatholytic effects of the drug. Future multi-center clinical trials utilizing larger cohorts and evaluating different infusion durations are warranted to optimize the administration protocols of dexmedetomidine in patients undergoing traumatic nasal surgeries (15).

Conclusion

In conclusion, intraoperative infusion of dexmedetomidine during traumatic nasal surgery significantly attenuates emergence agitation and mitigates postoperative pain severity, while ensuring superior hemodynamic stability in the early recovery phase. Although the intervention is associated with a marginal prolongation of tracheal extubation and post-anesthesia care unit stay, it maintains an excellent safety profile with a notable reduction in postoperative shivering. These findings suggest that dexmedetomidine serves as a highly effective and safe adjunct anesthetic regimen to optimize patient recovery and comfort, though larger multi-center trials are warranted to standardize clinical infusion protocols.

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Conflicts of interest

The authors declare that they have no competing interests.

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Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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