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Effect of Intravenous Dexmedetomidine on Postoperative Analgesia at Tertiary care Teaching Center

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Abstract

Background: Effective postoperative pain control remains a clinical challenge, and opioid-based analgesia is often limited by adverse effects. Dexmedetomidine, a selective alpha-2 adrenergic agonist, possesses analgesic and sedative properties that may reduce opioid requirements after surgery. This study evaluated the effect of intravenous dexmedetomidine on postoperative pain scores, analgesic consumption, and hemodynamic stability. **Methods:** In this prospective, randomized, double-blind, placebo-controlled trial, 100 ASA I–II patients undergoing elective lower abdominal surgery under general anesthesia were allocated into two equal groups. Group D received intravenous dexmedetomidine (1 µg/kg loading dose over 10 minutes, followed by 0.5 µg/kg/hour infusion until skin closure); Group C received an equal volume of normal saline. Postoperative pain (visual analogue scale, VAS), time to first rescue analgesia, 24-hour tramadol consumption, sedation (Ramsay scale), hemodynamic parameters, and adverse effects were recorded. **Results:** VAS scores were significantly lower in Group D at all postoperative time points ($p<0.001$). Time to first rescue analgesia was significantly prolonged in Group D (245.6 ± 42.3 min vs 98.4 ± 28.7 min, $p<0.001$), and total 24-hour tramadol consumption was significantly reduced (142.5 ± 32.6 mg vs 248.7 ± 38.4 mg, $p<0.001$). Group D showed higher sedation scores and a higher incidence of bradycardia, but fewer episodes of nausea and vomiting. **Conclusion:** Intraoperative intravenous dexmedetomidine significantly improves postoperative analgesia and reduces opioid consumption, with an acceptable and manageable hemodynamic side-effect profile.

Keywords: dexmedetomidine; postoperative analgesia; alpha-2 agonist; opioid-sparing; visual analogue scale

INTRODUCTION

Postoperative pain remains one of the most common and inadequately managed complications of surgery, affecting patient comfort, early mobilization, and overall recovery (1). Despite advances in multimodal analgesia protocols, a substantial proportion of surgical patients continue to experience moderate to severe pain in the immediate postoperative period (2). Opioids remain the cornerstone of postoperative pain management; however, their use is frequently constrained by adverse effects such as respiratory depression, excessive sedation, nausea, vomiting, ileus, and the risk of tolerance or dependence (3). These limitations have driven continued interest in opioid-sparing adjuncts capable of providing effective analgesia while minimizing opioid-related morbidity.

Alpha-2 adrenergic agonists have emerged as valuable adjuncts in perioperative practice owing to their sedative, anxiolytic, sympatholytic, and analgesic properties (4). Dexmedetomidine, a highly selective alpha-2 adrenoceptor agonist with an alpha2:alpha1 selectivity ratio considerably greater than clonidine, has gained increasing popularity in anesthesia and critical care since its clinical introduction (5). Its analgesic action is thought to involve activation of alpha-2 receptors in the locus coeruleus and the dorsal horn of the spinal cord, inhibiting the release of substance P and norepinephrine and thereby attenuating nociceptive transmission through descending inhibitory pathways (6).
Unlike opioids, dexmedetomidine produces sedation and analgesia without clinically significant respiratory depression, making it an attractive option in the perioperative setting (5). Several investigators have shown that perioperative administration of dexmedetomidine produces stable sedation and reduces the requirement for other sedative and analgesic agents (7). Beyond its intraoperative benefits, growing evidence suggests that its analgesic effect extends into the postoperative period, reducing pain scores and rescue analgesic consumption (8).

Arain and Ebert demonstrated that postoperative dexmedetomidine infusion provided analgesia comparable to morphine patient-controlled analgesia, with a more favorable side-effect profile (8). Similarly, Gurbet et al. reported that intraoperative dexmedetomidine infusion significantly reduced perioperative analgesic requirements and postoperative

pain scores compared with placebo (9). A systematic review and meta-analysis of systemic alpha-2 agonists further confirmed that dexmedetomidine significantly reduces postoperative morphine consumption and pain intensity, while modestly increasing the risk of bradycardia and hypotension (10).
Despite this accumulating evidence, optimal dosing regimens, timing of administration, and the magnitude of opioid-sparing benefit continue to vary across surgical populations and anesthetic techniques, and controlled clinical evaluation in specific surgical settings remains warranted.

Therefore, the present study was designed to evaluate the effect of intravenous dexmedetomidine, administered as a loading dose followed by a continuous intraoperative infusion, on postoperative pain scores, total opioid consumption, time to first rescue analgesia, sedation level, and hemodynamic and adverse-effect profile, compared with placebo, in patients undergoing elective surgery under general anesthesia.

MATERIALS AND METHODS

This prospective, randomized, double-blind, placebo-controlled study was conducted in the Department of Anaesthesiology of a tertiary care teaching hospital after approval from the Institutional Ethics Committee and prospective trial registration. Written informed consent was obtained from all participants before enrollment.

A total of 100 patients aged 18–60 years, of American Society of Anesthesiologists (ASA) physical status I or II, scheduled for elective lower abdominal surgery under general anesthesia, were enrolled. Exclusion criteria included known hypersensitivity to dexmedetomidine, cardiac arrhythmias, uncontrolled hypertension, hepatic or renal impairment, pregnancy, body mass index greater than 35 kg/m², chronic opioid or sedative use, and patient refusal.

Sample size was calculated assuming a 20% reduction in 24-hour postoperative opioid consumption between groups, with 80% power and an alpha error of 0.05, yielding a minimum requirement of 45 patients per group; 50 patients were enrolled per group to allow for possible dropouts.

Patients were randomly allocated into two equal groups using a computer-generated random number table, with allocation concealed in sealed opaque envelopes: Group D (dexmedetomidine) and Group C (control/saline). The study drug was prepared by an anesthesiologist not involved in patient assessment, so that both the patient and the assessing investigator remained blinded to group allocation.
In the operating room, standard monitoring including electrocardiography, non-invasive blood pressure, and pulse oximetry was instituted. After induction of general anesthesia using a standardized protocol (intravenous fentanyl, propofol, and a neuromuscular blocking agent, followed by maintenance with a volatile anesthetic agent in an oxygen–air mixture), Group D received intravenous dexmedetomidine as a loading dose of 1 µg/kg infused over 10 minutes, followed by a maintenance infusion of 0.5 µg/kg/hour continued until skin closure. Group C received an equal volume of normal saline administered in an identical manner.

Intraoperative heart rate and mean arterial pressure were recorded at baseline, after induction, and at 15-minute intervals until completion of surgery. Postoperatively, patients were assessed by a blinded observer in the recovery room and surgical ward at 0, 2, 4, 6, 12, and 24 hours. Pain intensity was assessed using a 10-point visual analogue scale (VAS), where 0 indicated no pain and 10 indicated the worst imaginable pain. Rescue analgesia with intravenous tramadol 1 mg/kg was administered whenever the VAS score exceeded 4, and the time to first rescue analgesic request and total analgesic consumption over 24 hours were recorded. Sedation level was assessed using the Ramsay sedation scale. Adverse effects, including nausea, vomiting, bradycardia (heart rate <50 beats/min), hypotension (mean arterial pressure reduction >20% from baseline), and dry mouth, were noted and managed per institutional protocol.
Statistical analysis was performed using SPSS software (version 25.0). Continuous variables were expressed as mean ± standard deviation and compared using the unpaired Student's t-test; categorical variables were expressed as frequencies and percentages and compared using the chi-square test. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 100 patients completed the study, with 50 patients in each group. No patient was excluded after randomization, and there were no dropouts.

Table 1. Demographic and baseline characteristics

Parameter	Group D (n=50)	Group C (n=50)	p-value
Age (years, mean ± SD)	38.6 ± 10.2	39.4 ± 9.8	0.68
Sex (M/F)	28/22	26/24	0.70
Weight (kg, mean ± SD)	64.3 ± 8.1	63.8 ± 7.6	0.75

ASA status (I/II)	34/16	32/18	0.68
Duration of surgery (min, mean ± SD)	95.4 ± 18.6	92.8 ± 17.2	0.47

Baseline demographic and surgical characteristics, including age, sex distribution, weight, ASA status, and duration of surgery, were comparable between the two groups (p>0.05 for all parameters), confirming that the groups were well matched and that any differences in postoperative outcomes can reasonably be attributed to the study intervention rather than baseline imbalance.

Table 2. Postoperative VAS pain scores at rest

Time (hours)	Group D (mean ± SD)	Group C (mean ± SD)	p-value
0	1.8 ± 0.6	3.2 ± 0.8	<0.001
2	2.1 ± 0.7	4.5 ± 0.9	<0.001
4	2.6 ± 0.8	5.1 ± 1.0	<0.001
6	3.0 ± 0.9	5.6 ± 1.1	<0.001
12	2.4 ± 0.7	4.2 ± 0.9	<0.001
24	1.6 ± 0.5	2.8 ± 0.7	<0.001

VAS pain scores were significantly lower in Group D than in Group C at every postoperative time point assessed. Pain scores in both groups rose initially after surgery, peaking around 6 hours, before gradually declining by 24 hours; however, the magnitude of pain at every interval was consistently and significantly lower in patients who received dexmedetomidine, indicating a sustained analgesic effect extending well beyond the intraoperative infusion period.

Table 3. Postoperative analgesic consumption

Parameter	Group D (n=50)	Group C (n=50)	p-value
Time to first rescue analgesia (min, mean ± SD)	245.6 ± 42.3	98.4 ± 28.7	<0.001
Total 24-hour tramadol consumption (mg, mean ± SD)	142.5 ± 32.6	248.7 ± 38.4	<0.001
Number of rescue doses required (mean ± SD)	1.4 ± 0.6	2.8 ± 0.8	<0.001

Patients in Group D required their first rescue analgesic dose substantially later than those in Group C, more than doubling the analgesia-free interval. Correspondingly, total 24-hour tramadol consumption and the mean number of rescue doses required were significantly reduced in the dexmedetomidine group, demonstrating a clear opioid-sparing effect.

Table 4. Intraoperative hemodynamic parameters

Time point	HR Group D	HR Group C	MAP Group D	MAP Group C
Baseline	82 ± 8	84 ± 7	88 ± 6	89 ± 5
After induction	74 ± 7	80 ± 8	80 ± 7	86 ± 6
30 minutes	68 ± 6	78 ± 7	76 ± 6	84 ± 5
60 minutes	65 ± 5*	79 ± 6	74 ± 5*	85 ± 6
End of surgery	70 ± 6	81 ± 7	78 ± 6	87 ± 5

HR = heart rate (beats/min); MAP = mean arterial pressure (mmHg). *p<0.05 vs Group C.

Group D showed a modest, statistically significant reduction in heart rate and mean arterial pressure compared with Group C across the intraoperative period, most pronounced at 60 minutes after induction. These reductions remained within a clinically acceptable physiological range and did not necessitate emergency intervention in any patient, reflecting the expected sympatholytic action of dexmedetomidine.

Table 5. Postoperative sedation and adverse effects

Parameter	Group D (n=50)	Group C (n=50)	p-value
Ramsay sedation score (mean ± SD)	3.2 ± 0.6	1.8 ± 0.4	<0.001
Nausea/vomiting, n (%)	6 (12%)	16 (32%)	0.014
Bradycardia, n (%)	5 (10%)	0 (0%)	0.027
Hypotension, n (%)	4 (8%)	1 (2%)	0.16
Dry mouth, n (%)	8 (16%)	2 (4%)	0.045

Patients receiving dexmedetomidine had significantly higher sedation scores, consistent with its known sedative property, but remained easily arousable throughout the observation period. The incidence of postoperative nausea and vomiting was significantly lower in Group D, likely reflecting reduced opioid consumption. Bradycardia and dry mouth occurred significantly more often in Group D, though all episodes of bradycardia were transient and resolved without pharmacological treatment; the difference in hypotension did not reach statistical significance.

DISCUSSION

The present randomized, double-blind, placebo-controlled study demonstrated that intravenous dexmedetomidine, administered as a loading dose followed by a continuous intraoperative infusion, significantly improved postoperative analgesia, as reflected by lower VAS pain scores, prolonged time to first rescue analgesic, and reduced total opioid consumption over 24 hours compared with placebo. These findings are consistent with previous reports describing the opioid-sparing and analgesic properties of dexmedetomidine in the perioperative period (8,9).

The analgesic effect observed in this study can be explained by the action of dexmedetomidine on alpha-2 adrenoceptors located in the locus coeruleus and the dorsal horn of the spinal cord, where it inhibits nociceptive transmission through activation of descending noradrenergic inhibitory pathways and suppression of substance P release (6). This central antinociceptive mechanism, distinct from the mu-opioid receptor pathway, likely accounts for the prolonged analgesic effect observed even after discontinuation of the infusion at the end of surgery, a finding similar to that reported by Gurbet et al., who demonstrated reduced perioperative analgesic requirements with intraoperative dexmedetomidine infusion (9). The significant reduction in total tramadol consumption and the prolonged time to first rescue analgesia observed in Group D corroborate the findings of Blaudszun et al., whose meta-analysis of systemic alpha-2 agonists reported a clinically meaningful reduction in postoperative morphine consumption and pain intensity scores across multiple surgical populations (10). Similarly, Lin et al. reported that combining dexmedetomidine with morphine for patient-controlled analgesia improved pain control while reducing opioid-related side effects such as nausea and vomiting, paralleling the lower incidence of postoperative nausea and vomiting observed in the dexmedetomidine group in the present study (11).

The higher Ramsay sedation scores noted in Group D reflect the inherent sedative property of dexmedetomidine, which, while beneficial for patient comfort, requires careful postoperative monitoring, particularly in settings with limited nursing supervision (5). Hemodynamically, although Group D exhibited a modest reduction in heart rate and mean arterial pressure during the intraoperative period, the changes remained within a clinically acceptable range, and the incidence of bradycardia, while statistically significant compared with control, was self-limiting and required no pharmacological intervention in this study. This finding aligns with reviews identifying bradycardia and hypotension as the most consistently reported adverse effects of dexmedetomidine, warranting cautious use in patients with pre-existing bradyarrhythmias or hemodynamic instability (12).

This study has certain limitations. The relatively modest, single-center sample size and short follow-up period of 24 hours limit generalizability to broader surgical populations and longer-term outcomes. Additionally, only a single fixed dosing regimen was studied, and dose-response relationships were not explored. Future multicentric trials with larger sample sizes, varied dosing protocols, and longer postoperative follow-up are warranted to further define the optimal role of dexmedetomidine in multimodal postoperative analgesia protocols.

CONCLUSION

This study demonstrates that intravenous dexmedetomidine, when administered intraoperatively as a loading dose followed by a continuous infusion, significantly enhances postoperative analgesia by lowering pain scores, prolonging the duration of effective analgesia, and decreasing total opioid consumption, without causing clinically significant hemodynamic compromise. Dexmedetomidine may therefore be considered a valuable adjunct in multimodal postoperative pain management protocols, particularly for reducing opioid-related adverse effects such as nausea and vomiting. However, careful patient selection and continuous hemodynamic monitoring are advised given its propensity to cause bradycardia.

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