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Research Article

Dexmedetomidine in Fast-Track Cardiac Care for Patients Undergoing Coronary Artery Bypass Graft Surgery

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ABSTRACT

Background: Dexmedetomidine offers established cardioprotective benefits, yet its specific impact on ventilation weaning and broader recovery metrics in cardiac surgery warrants a clearer definition.
Methods: We enrolled 100 elective CABG patients in a double-blind randomized trial. Subjects received either a dexmedetomidine regimen (1 µg/kg loading dose, 0.3 µg/kg/h maintenance) or standard care. The study prioritized mechanical ventilation duration as the primary endpoint. We further analyzed cardiac injury markers alongside interleukin-6 levels and length of stay.
Results: The median duration of mechanical ventilation was shorter in the dexmedetomidine group (210 vs. 310 minutes; p=0.004). This recovery benefit also found its way to biological indices, with the intervention group showing lower median amounts of CK-MB (65.00 vs 129.00 U/L; p<0.001), troponin I (0.07 vs 4.04 ng/mL; p<0.001) and five pack interleukin-6 (40.00 vs 70.00 pg/mL; p=0·004). As a result, patients had shorter times in the ICU (23.0 ± 24.9 vs. 32.25 ± 38.3 hours; p=0.023) and hospital (9 ± 5.2 vs 11±7 days; p<0.001). However, the drug disturbed hemodynamics stability with higher rates of hypotension (44% vs 22%; p=0.019), bradycardia (62% vs 24%; p<0.001) and need for temporary pacing (50% vs 16%; p<0.001).
Conclusion: Administering dexmedetomidine accelerates extubation and attenuates myocardial injury. These benefits justify its use, provided the anesthesia team proactively manages the accompanying hemodynamic instability.

Introduction

Coronary artery bypass grafting remains the standard intervention for advanced coronary disease, yet it subjects patients to significant perioperative risk. The obligatory cardiopulmonary bypass triggers a potent systemic inflammatory response and ischemia-reperfusion injury that compromises organ integrity. These physiological insults result into myocardial dysfunction and other postoperative complications which delay rapid recovery [1–3]. Early extubation and reduced length of ICU stay are the key goals behind enhance recovery protocols, but achieving them without too much hemodynamic compromise requires a specific pharmacological approach. Selectively

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increasing the level of sedation and refraining inflammation through the action mechanism of dexmedetomidine that offers postproceeding stress relating perioperatively. This agent is noteworthy in that it lowers the incidence of postoperative delirium by 30-40% in various surgical populations (4). It is well established that dexmedetomidine mitigates ischemia-reperfusion injury through differing cellular pathways. It protects mitochondrial and endothelial integrity, and upregulates autophagy to markedly restrain oxidative stress and ferroptosis [3,5–8]. Clinical studies have recently shown that dexmedetomidine reduces postoperative cardiac biomarkers and inflammatory cytokines, including troponin, CK-MB, IL-6 and TNF-α [9–11]. The agent decreases the need for opioids to help promote fast emergence from anesthesia. Its incorporation into protocols for fast-track approaches dramatically decreases mechanical ventilation time and shortens ICU length of stay [7]. operative dexmedetomidine reduced one-year mortality from 7.95% to 3.17% in a cohort of 1134 cardiac patients (p=0.07). It came along with a significant reduction in postoperative complications and delirium [1].

Nevertheless, fear of dose-dependent bradycardia and hypotension limits its common use. Such hemodynamic changes often require pharmacologic support or temporary pacing [5,12-13]. The ideal dosing strategy that offers a balance between the cardioprotective and hemodynamic risks has not yet been established. However, recent evidence indicates there are improvements in hemodynamic control and suppression of postoperative arrhythmias compared to lower doses when using regimens recommended by guidelines [7,14-15]. While these cardioprotective mechanisms are well characterized in experimental models, their clinical translation to on-pump CABG surgery has not yet been established. Confirmation from rigorous prospective trials is now required to establish if these pathways translate into clinically meaningful gains in global recovery endpoints and inflammatory surrogate measures.

This randomized controlled trial evaluates the efficacy and safety of perioperative dexmedetomidine in adults undergoing on-pump CABG. We designated time to extubation as the primary end point. To gauge cardioprotection and recovery quality, we further assessed cardiac (troponin I, CK-MB) and inflammatory (IL-6) biomarkers, length of stay, delirium incidence, hemodynamic stability, and major adverse events.

Methods

This prospective, double-blind, randomized controlled trial was conducted at a teaching hospital in Tehran, Iran, in strict adherence to the Declaration of Helsinki and CONSORT guidelines. The study protocol was approved by the Iranian Registry of Clinical Trials (IRCT20120910010800N13) and the Ethics Committee of Shahid Beheshti University of Medical Science (IR.SBMU.MSP.REC.1404.253), and written informed consent was obtained from all subjects before enrollment.

Adults (>18 years) scheduled for primary elective on-pump CABG were assessed. Eligibility required normal preoperative hepatic and renal functions. Candidates with severe left ventricular dysfunction (ejection fraction < 30%), pre existing conduction abnormalities, severe arrhythmias, prior cerebrovascular accidents, or significant neurological impairment were excluded. Additional exclusion criteria included chronic sedative or opioid use (>3 months), smoking history, and hypersensitivity to dexmedetomidine. A total of 100 participants were randomized in a 1:1 allocation ratio to the dexmedetomidine or control groups. Block randomization with variable block sizes of 4 and 6 was used, and the randomization sequence was generated using the randomizeR package in R software (R Foundation for Statistical Computing, Vienna, Austria).

Allocation concealment was ensured using sealed, opaque, sequentially numbered envelopes. The study agents were prepared by an independent anesthesia nurse who was not involved in clinical management or data collection. Dexmedetomidine (4 µg/mL) and a visually matched 0.9% saline placebo were prepared in identical 50 mL syringes. Each syringe was labeled only with the randomization code, date, and the phrase “Study Drug.” Complete blinding of patients, anesthesia and surgical teams, ICU staff, and outcome assessors was thereby ensured. Unblinding was permitted solely in the event of life-threatening emergencies; however, no such events occurred during the trial. Standard monitoring, including five-lead electrocardiography, pulse oximetry, invasive arterial pressure, and bispectral index monitoring (target 40–60), was established upon arrival in the operating room. Anesthesia was induced using a standardized regimen of midazolam, fentanyl, etomidate, and rocuronium and maintained with isoflurane and intermittent doses of rocuronium and fentanyl in a volatile based, opioid sparing approach.

Cardiopulmonary bypass was performed using standard heparinization, temperature, and hemodynamic management. Anesthesia was maintained during bypass, and separation from bypass was guided by transesophageal echocardiography with vasoactive support as required, followed by protamine reversal. Surgery was conducted via median sternotomy under mild hypothermic cardiopulmonary bypass (32–34 °C). Myocardial protection was achieved with cold-blood cardioplegia, and a mean arterial pressure of 50–70 mmHg was targeted during bypass. Before induction, a dexmedetomidine loading dose (1 µg/kg) was administered to the intervention group over 10–15 minutes, followed by a continuous maintenance

infusion of 0.3 µg/kg/h throughout surgery. The control group received a volume and rate matched normal saline placebo during both phases. Heart rate and mean arterial pressure were continuously monitored throughout the procedure. Intraoperative hypotension, defined as a mean arterial pressure < 60 mmHg or a > 20% decrease from baseline, was managed with fluid boluses and vasopressors (phenylephrine or norepinephrine). With hemodynamically significant bradycardia (heart rate <50 beats/min), atropine or temporary pacing was performed.

The main endpoint was time to extubation, defined as the time elapsed from ICU admission to endotracheal tube removal. Secondary outcomes were myocardial injury and inflammation: serum creatine kinase-MB (CK-MB), cardiac troponin I (cTnI) and interleukin-6 (IL-6) were assessed at baseline and 24 hours after surgery. The hemodynamic stability was evaluated by the occurrence of intraoperative hypotension, bradycardia and temporary pacing. Secondary outcomes included ICU length of stay, postoperative atrial arrhythmias, ventricular arrhythmias and pain intensity measured using the visual analog scale at transfer to the ward. Postoperative delirium was evaluated with the Confusion Assessment Method for the ICU (CAM ICU), and adverse events, including 30-day mortality and major complications, were systematically recorded.

Following the methodology specified by Bindu etal. Power calculations indicated that a sample size of 23 patients per group (power=90%, α=0.05) would be sufficient to detect hemodynamic changes (16). This is equivalent to a 150 min difference in mechanical ventilation duration but taking into consideration heterogeneity of ventilation times observed in many patients undergoing CABG, our study was powered to be able to detect differences ≥100 minutes, using pooled standard deviation (conservative estimate) 150 minutes. Given this information we calculated that a minimum of 40 patients per group would be required for 80% power. Allowing for a 20% attrition rate, 50 patients were enrolled in each group (N = 100). All statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

The distribution of continuous variables was assessed using the Shapiro–Wilk test. As most variables, including biomarkers and time intervals, were not normally distributed, data were expressed as medians (interquartile range [IQR]) and between group comparisons were performed using the Mann–Whitney U test. Categorical variables were presented as frequencies (percentages) and analyzed using the chi square test or Fisher's exact test, as appropriate. Statistical significance was defined as a P value < 0.05.

Results

A total of 100 patients undergoing elective cardiac surgery were enrolled and randomly assigned in a 1:1 ratio to receive dexmedetomidine or standard care. Baseline characteristics were comparable between the two groups. Age distribution was similar, with a median of 68.00 years (IQR=14.00) in the control group and 66.50 years (IQR=14.00) in the dexmedetomidine group (U=1112.0, p=0.341). Similarly, Body Mass Index did not differ significantly with respective medians of 25.01 kg/m² (IQR=5.58) and 26.05 kg/m² (IQR=3.68) (U=1407.0, p=0.279). Groups were well balanced with respect to sex distribution and the presence of comorbidity profiles, including hypertension, diabetes mellitus and prior cardiac interventions.

Operative characteristics between groups were similar (Table.1). Cardiopulmonary bypass time did not differ significantly, with a median of 126.17 minutes (IQR=19.58) in the control group versus 130.25 minutes (IQR=26.67) in the dexmedetomidine group (U=1100.5, *Z*=-1.036, p=0.303, r=0.10). Together with aortic cross-clamp duration which was similar between the two groups (78.12 minutes [IQR=14.51] vs 79.99 minutes [IQR=25.21], U=1127.0, p=0.396, r=0.08). Table 2 illustrates that the dexmedetomidine group was associated with higher degree of hemodynamic instability with significantly lower incidence of intraoperative hypotension (44.0% vs. 22.0%; $\chi^2 = 5.473$, p = 0.019) and bradycardia (62.0% vs. 24%, χ^2=14.729, P<.001). Consequently, the requirement for temporary pacing was significantly greater in the dexmedetomidine group compared to controls (50.0% vs. 16.0%; $\chi^2 = 13.071$, p < 0.001). While baseline biomarkers were comparable, significant differences emerged 24 hours postoperatively (Table 3). The dexmedetomidine group had lower median CK-MB (65.00 U/L, IQR=104.25 vs 129.00 U/L, IQR=111.38; U = 605.5, p <0.001) and cardiac troponin I levels (0.07 ng/mL, IQR=2.52 vs 4.04 ng/mL, IQR=2.85; U=451.0, p<0·001). As another measure of the inflammatory response, postoperative IL-6 concentrations were also significantly lower in the dexmedetomidine group (40.00 pg/mL (IQR=34.20 vs 60.00 pg/mL (IQR=66.70; U = 836.5, p = 0.004)), despite having similar preoperative values between groups. Patients in the dexmedetomidine group had better recovery outcomes, with a significantly shorter time on mechanical ventilation (median 210.00 min, IQR=50.00 vs. 310.00 min, IQR=135.00; U = 835.5, p=0.004) and less ICU length of stay (median 23 hours, IQR=7.50 vs. median 32:25 hours; IQR=20:0; U =9210, p=0.022). Total hospital stays also decreased significantly in the intervention group (median 9.00 days, IQR=2.00 vs. 11.00 days, IQR=4.25; U = 644.0, p < 0.001). Pain scores at ICU discharge and patient satisfaction upon ward

transfer showed no significant differences between groups (Table 4).

Table 1- Baseline demographic and clinical characteristics of study participants

Variable	Control	Dexmedetomidine	Statistic	P value	Effect Size
Age, years Median (IQR)	68.00 (14.00)	66.50 (14.00)	U = 1112.0	0.341	r = 0.095
BMI, kg/m² Median (IQR)	25.01 (5.58)	26.05 (3.68)	U = 1407.0	0.279	r = 0.108
Male sex, n (%)	42 (84.0%)	41 (82.0%)	$\chi^2 = 0.071$	0.790	V = 0.027
Hypertension, n (%)	45 (90.0%)	44 (88.0%)	$\chi^2 = 0.102$	0.749	V = 0.032
Diabetes mellitus, n (%)	36 (72.0%)	32 (64.0%)	$\chi^2 = 0.735$	0.391	V = 0.086
Previous cardiac intervention, n (%)	7 (14.0%)	10 (20.0%)	$\chi^2 = 0.638$	0.424	V = 0.080

IQR, interquartile range; BMI, body mass index; U, Mann-Whitney U statistic; χ^2, Chi-square statistic; r, effect size for Mann-Whitney U test; V, Cramér's V.

Table 2- Intraoperative hemodynamic events and surgical parameters

Variable	Control	Dexmedetomidine	Statistic	P value	Effect Size
Surgery duration, min Median (IQR)	373.00 (131.75)	360.00 (101.25)	U = 1176.0	0.610	r = 0.051
CPB time, min Median (IQR)	126.17 (19.58)	130.25 (26.67)	U = 1100.5	0.303	r = 0.10
Aortic Cross-Clamp Time, min Median (IQR)	78.12 (14.51)	79.99 (25.21)	U = 1127.0	0.396	r = 0.085
Hypotension, n (%)	11 (22.0%)	22 (44.0%)	$\chi^2 = 5.473$	0.019	V = 0.234
Bradycardia, n (%)	12 (24.0%)	31 (62.0%)	$\chi^2 = 14.729$	<0.001	V = 0.384
Temporary pacemaker, n (%)	8 (16.0%)	25 (50.0%)	$\chi^2 = 13.071$	<0.001	V = 0.362

IQR, interquartile range; CPB, cardiopulmonary bypass; U, Mann-Whitney U statistic; χ^2, Chi-square statistic; r, effect size for Mann-Whitney U test; V, Cramér's V.

Table 3- Cardiac Biomarkers and IL-6

Variable	Control	Dexmedetomidine	Statistic	P value	Effect Size
CK-MB preop, U/L Median (IQR)	13.00 (10.00)	13.00 (8.00)	U = 1157.0	0.518	r = 0.065
CK-MB postop 24h, U/L Median (IQR)	129.00 (111.38)	65.00 (104.25)	U = 605.5	<0.001	r = 0.445
cTnI preop, ng/mL Median (IQR)	0.01 (0.00)	0.01 (0.00)	U = 1200.0	0.673	r = 0.042
cTnI postop 24h, ng/mL Median (IQR)	4.04 (2.85)	0.07 (2.52)	U = 451.0	<0.001	r = 0.552
IL-6 preop, pg/mL Median (IQR)	5.70 (12.55)	10.00 (15.00)	U = 1053.0	0.172	r = 0.136
IL-6 postop 24h, pg/mL Median (IQR)	70.00 (66.70)	40.00 (34.20)	U = 836.5	0.004	r = 0.286

IQR, interquartile range; CK-MB, creatine kinase-myocardial band; cTnI, cardiac troponin I; IL-6, interleukin-6; U, Mann-Whitney U statistic; r, effect size.

Table 4- Postoperative Recovery

Variable	Control	Dexmedetomidine	Statistic	P value	Effect Size
Time to Extubation, min Median (IQR)	310.00 (135.00)	210.00 (50.00)	U = 835.5	0.004	r = 0.288
ICU stay, hours Median (IQR)	32.25 (20.00)	23.00 (7.50)	U = 921.0	0.022	r = 0.228
Delirium, n (%)	7 (14.0%)	4 (8.0%)	$\chi^2 = 0.919$	0.338	$\Phi = -0.096$
Arrhythmias, n (%)	35 (70.0%)	21 (42.0%)	$\chi^2 = 7.955$	0.005	$\Phi = -0.282$
Hospital stay, days Median (IQR)	11.00 (4.25)	9.00 (2.00)	U = 644.0	<0.001	r = 0.424
Pain score at ICU discharge Median (IQR)	4.00 (2.00)	4.00 (1.25)	U = 1203.0	0.731	r = 0.034

Total Morphine Consumption at 24 hours, mg Median (IQR)	23.50 (11.25)	21.00 (10.50)	U = 986.5	0.069	r = 0.182
Satisfaction score Median (IQR)	4.00 (1.25)	4.00 (2.00)	U = 1204.0	0.743	r = 0.034

IQR, interquartile range; ICU, intensive care unit; VAS, Visual Analog Scale; U, Mann-Whitney U statistic; χ^2, Chi-square statistic; r, effect size for the Mann-Whitney U test; Φ, Phi coefficient.

Postoperative pain severity at the time of ward transfer did not differ significantly between the cohorts (Control: median 4.0, IQR=2.0 vs. Dexmedetomidine: median 4.0, IQR=1.25; U = 1203.0, Z = -0.343, p = 0.731), yielding a negligible effect size (r = -0.034). While the dexmedetomidine group exhibited lower total morphine consumption (MEDIAN: 21.00 mg, IQR=10.50) compared to controls (Median: 23.50 mg, IQR=11.25), this trend did not reach statistical significance (U = 986.5, p = 0.069; r = 0.18). The overall incidence of ICU delirium was 11%, affecting 14% of control patients and 8% of those in the dexmedetomidine group. This difference did not reach statistical significance (chi2 = 0.919, p = 0.338; Fisher's exact test p = 0.525), and the effect size remained negligible (phi = -0.096). Although the odds ratio favored the intervention (OR = 0.534; 95% CI: 0.146–1.954), the confidence interval implies no definitive association between dexmedetomidine administration and delirium prevention in this cohort. Postoperative arrhythmias affected 56% of the total cohort, occurring significantly less frequently in the dexmedetomidine group compared to controls (42% vs. 70%; chi2 = 7.955, p = 0.005; Fisher's exact test p = 0.008). This reduction demonstrated a moderate effect size (phi = -0.282). The analysis yielded an odds ratio of 0.310 (95% CI: 0.136–0.708), indicating that dexmedetomidine administration reduced the odds of developing postoperative arrhythmias by approximately 69%. As shown in (Table 5), the incidence of postoperative complications, such as bleeding or tamponade, remained comparable between groups (Fisher's exact test p = 0.638). Although two deaths occurred in the control group versus none in the dexmedetomidine arm, this difference did not reach statistical significance (p = 0.495).

Discussion

This randomized controlled trial demonstrates a significant cardioprotective effect of perioperative administration of dexmedetomidine in patients undergoing on-pump cardiac surgery. The intervention is associated with decreased markers of myocardial injury, inflammatory response and faster recovery profiles. But these advantages were accompanied by increased intraoperative hemodynamic instability, which underscores the need for careful anesthetic management.

Echoing current findings that support the cardioprotective effectiveness of the agent, compared with controls the cohort treated with dexmedetomidine had significantly lower postoperative cardiac injury markers. In particular reduced cardiac troponin I (0.07 ng/mL vs 4.04 ng/mL; p < 0.001) and CK-MB concentrations (65.00 U/L vs 129.00 U/L; p < 0.001) compared to the control group were observed [8,17–19]. Meta-analyses in more heterogeneous pediatric populations demonstrate significantly lower myocardial injury biomarkers at 24 to 48 hours postoperatively [17]. These results are only partly shown in adult cardiac surgery studies, as the extent of biomarker attenuation is influenced by the dose and timing of application [10,18,20–22]. These data suggest that dexmedetomidine, by up-regulating autophagic activity and inhibiting oxidative stress and ferroptosis, ameliorates ischemia-reperfusion injury through a mechanistic pathway via maintenance of mitochondrial and endothelial glycocalyx integrity [3]. Aortic cross-clamp and total CPB times were similar between groups. Because myocardial enzyme release depends mainly on how long the heart is under ischemic arrest, and because both Troponin I and CK-MB levels were decreased after dexmedetomidine adjunctive therapy over placebo groups of patients, these improvements strongly suggest that there is a pharmacological cardioprotective effect of dexmedetomidine.

The 43% decrease in postoperative IL-6 level (40.00 vs. 70.00pg/mL; p=0.004) provides objective evidence of a substantial dampening effect on the systemic inflammatory response triggered by cardiopulmonary bypass setting. On the other hand, a 2025 randomized trial done by Zdravković et al. observed that without a loading dose (0.5 μg/kg/h), the continuous administration could not attenuate postoperative IL-6 surge, producing similar responses in both intervention (73·0 pg/mL) and control groups (72·4 pg/mL) [19]. Totonchi et al. also reported no clinical anti-inflammatory efficacy when a loading dose was not included [23]. However, another multilayer systematic review with data from multiple clinical trials (17) found that dexmedetomidine has an overall significant effect in reducing inflammation: it reduces the pro-inflammatory mediators such as IL-6, TNF-α and NF-κB significantly at most of the postoperative points. This divergence likely relates to the dosing schedule. The use of a loading dose seems necessary to suppress inflammatory mediators, while maintenance only approaches are ineffective. The significant IL-6 reduction observed in our cohort is consistent with the bulk of literature supporting anti-inflammatory properties of dexmedetomidine [15,17-18].

Mechanical ventilation duration was significantly lower in the dexmedetomidine group (210 vs. 310 min; p=0.004), which translates to a 32% reduction. Clinically, this decrease is important as long duration of ventilation has been reported to guarantee ventilator-associated pneumonia, respiratory deconditioning and hemodynamic instability. These findings are consistent with a 2023 meta-analysis by Poon et al. that provided evidence supporting intraoperative dexmedetomidine accelerating extubation time in adult cardiac surgery [24]. Similarly, Ysenbaardt et al. in a retrospective cohort following coronary artery bypass graft surgery [25]. Kerroum et al. also showed that the combination of dexmedetomidine with ERAS protocols for cardiac surgery enhances early recovery by promoting respiratory weaning and postoperative recovery [7]. Dexmedetomidine leads to better respiratory outcomes by a mechanism utilizing non-GABAergic systems not shared by propofol and benzodiazepines. This mechanism may create feasible both "cooperative sedation," permitting rapid arousal upon stimulation, with an effective anxiolytic and analgesic effect [26].

Table 5- Postoperative complications and in-hospital mortality

Outcome	Control	Dexmedetomidine	Test	P value	Effect Size
Any complication Median (IQR)	14 (28.0%)	11 (22.0%)	Fisher's exact test	0.638	V = 0.162
Mortality Median (IQR)	2 (4.0%)	0 (0.0%)	Fisher's exact test	0.495	V = 0.143

IQR, interquartile range; V, Cramér's V.

This specific sedation profile preserves airway reflexes and permits earlier neurological assessment, expediting extubation. Furthermore, the drug's opioid-sparing properties reduce cumulative opioid requirements, thereby minimizing respiratory depression and preventing delayed weaning [27].

Alpha-2 agonism also diminishes the surgical stress response and sympathetic hyperactivity, leading to optimized respiratory mechanics and decreased oxygen demand in the immediate postoperative period. [28]. Finally, the marked reduction in IL-6 levels seen here indicates that anti-inflammatory effects of dexmedetomidine may play a role in lessening pulmonary inflammation secondary to bypass and hence resulting in preserved lung function and gas exchange. The 100-minute reduction in time spent on mechanical ventilation noted here was larger than the reduced times reported in earlier studies. This result corroborates findings from the literature that Dexmedetomidine reduces time to extubation or weaning from mechanical ventilation in elective CABG cohorts [7,24-25].

However, Singh et al., have presented some contradictory evidence; no significant difference in duration of mechanical ventilation between the dexmedetomidine and control groups was found [29], a prospective study on CABG patients. Cheng et al. decreased extubation to a similar extent; however, this gain was tentatively abolished in multimorbid elderly patients [14]. These inconsistencies indicate that the extent of respiratory benefit is determined according to the drug regimen, timing of administration, age of the patient and baseline pulmonary reserve. These outcomes are also additionally modified by concurrent comorbidities that affect respiratory mechanics and institutional protocols. Dexmedetomidine shortened duration of ICU length of stay (mean 23.0 vs. median 32.25 hours; p=0.023), a clinically meaningful improvement in resource utilization. Nonetheless, there are conflicting data in the literature regarding this endpoint. Numerous studies describe this paradox of dexmetomidine speeds extubation, but prolonged ICU stay [14,25]. A non-significant trend toward longer ICU duration in the dexmedetomidine group was also seen in a risk-adjusted analysis of 1,134 patients [1], while no difference was indicated by other investigations (30). In contrast, our results are consistent with studies which have reported a markedly decreased ICU length of stay [7]. Dexmedetomidine significantly reduces length of ICU stay compared with controls, as shown in a meta-analysis conducted in 2018 [6]. In addition, a retrospective cohort of 871 patients underlined the key role of dosing strategy and showed that as compared to lower regimens, guideline-recommended doses subsequently led to shorter ICU stay [21].

Dexmedetomidine greatly reduced hospital length of stay (9 vs. 11 days; p<0.001), an 18% reduction, enhancing both cost-effectiveness and throughput. Previous data concerning this outcome are mixed, with significant reductions reported in some studies and negligible differences after adjustment for confounding factors in others [7,14,29]. However, our findings corroborate the role of dexmedetomidine in reducing hospital length of stay. These findings imply that where dexmedetomidine is used in perioperative regimens, discharges are quickened.

The major drawback of this dexmedetomidine regimen is substantially increased rates of intraoperative hypotension (44% vs. 22%; p=0.019), bradycardia (62% vs. 24%; p<0.001) and temporary pacemaker requirements (50% vs. 16%; p<0,001). These findings are consistent with the known dose-dependent hemodynamic profile of alpha2-agonists and represent a direct physiological consequence of sympatholytic activity [21,31] The pressure and heart rate induced by

dexmedetomidine is maintained at much lower levels than standard care [32]. While Hatemi et al. showed stable hemodynamics [33), subsequently, most studies do not support these findings. The most common side effect of dexmedetomidine continues to be bradycardia [34]. Our cohort incidence of 62% is consistent with the general literature; these rates vary based on dose and demographics. This higher risk of bradycardia in various ICU populations when comparing dexmedetomidine against propofol was also confirmed by a meta-analysis published in 2022. [35]. Conversely, Soltani et al. occurred only in 2.6% of the patients undergoing off-pump surgery [32]. The rather large difference must imply that our regimen was more effective in causing sympatholysis. Recent data suggest that the guideline-recommended dosing (µg/kg/h) improves hemodynamic control; one retrospective cohort study from 2025 with an aggregate of 871 cardiac surgery patients showed adherence to guidelines greatly reduced perioperative heart rate and blood pressure variability [21].

The main adverse effects of this dexmedetomidine protocol are significantly higher rates of intraoperative hypotension (44% vs 22%; p=0.019), bradycardia (62% vs 24%; p<0.001) and temporary pacemaker requirements (50% vs 16%; p<0.001). These findings are consistent with the known dose-dependent hemodynamics of α_2-agonists and represent direct sympatholytic effects [18]. Importantly, this significant requirement for temporary pacing was seen although the doses of infusion remained below the recommended range of 0.2–0.7 µg/kg/h [5].

Dexmedetomidine-induced hemodynamic instability can be controlled with close monitoring, early volume resuscitation and careful vasopressor administration. In current off-pump CABG settings, the titration of infusions to mean arterial pressure targets reduced complications without compromising arrhythmia prophylaxis [18,32]. Using similar dynamic protocols could reduce the degree of temporary pacing and spare some of the cardioprotective effects from dexmedetomidine.

Although the analgesic effect of dexmedetomidine was observed, there was no statistically significant difference in visual analogue scores at transfer from recovery to the ward between groups (median VAS 4.0; p=0.731). This differs from most of the literature; for example, El-Safty et al. found that using this drug resulted in a 30% reduction in opioid needs during CABG making it an effective analgesic adjuvant [36]. Similarly, Zientara et al. showed that dexmedetomidine provides efficient pain control in facilitating early extubation after fast track off-pump CABG surgery without jeopardizing patient's comfort [37]. Multiple other studies also supporting the safe and effective admittance of dexmedetomidine into opioid-sparing protocols in anesthesia [7,30].

Even though dexmedetomidine is well known to reduce the requirement for opioids, the present trial showed only a non-significant trend towards less morphine consumption. It is plausible that multiple methodological factors are behind this discrepancy. One con of this trial was that the duration between intraoperative administration and pain assessment at ward transfer may have disallowed peak serologic differentiations, which are most often yielded and assessable during immediate postoperative or ICU periods. Second, the widespread use of multimodal analgesia in both groups likely obscured small differences in pain perception. Third, the study did not systematically quantify total postoperative opioid load which would have allowed for a more sensitive assessment of sparing effects. Despite these limitations, the literature strongly supports the incorporation of dexmedetomidine into multimodal cardiac anesthetic approaches, particularly when minimizing opioid consumption is a clinical goal.

Delirium incidence was numerically lower in the dexmedetomidine group (8% vs. 14%; OR = 0.534, 95% CI: 0.146–1.954; p=0.338), but not statistically significant and likely a reflection of inadequate power due to the low aggregate event rate (11%). However, this trend replicates previous evidence as dexmedetomidine decreases the incidence and duration of delirium mainly in elderly patients [5,30,38]. Singh et al [29] attribute selective alpha-2 agonism as the factor which produces these neuroprotective effects. Thus, although statistical weaknesses exist here, the data clearly affirm dexmedetomidine as a viable agent for delirium prevention in high-risk cardiac surgery.

We found that perioperative dexmedetomidine administration provided a clinically meaningful decrease in postoperative arrhythmias (43% vs. 70%; p=0.005), with an odds reduction of approximately 69% (OR = 0.310, 95% CI: 0.136-0.708). These results are consistent with the systematic review and meta-analysis by Wang et al. [34] reaching the same conclusion regarding the efficacy of dexmedetomidine on decreasing atrial fibrillation and ventricular tachycardia, and data from Soltani et al. regarding off-pump procedures. Zientara et al. Another study [37] also showed lower rates of new-onset atrial fibrillation and successful fast-track recovery. This protection is mechanistically related to central alpha-2 agonism involving sympatholysis, decreasing circulating catecholamines and preserving autonomic balance [32]. Therefore, dexmedetomidine provides effective prophylaxis against arrhythmia; however, the risk of bradycardia requires intensive monitoring to prevent hemodynamic deterioration.

Although there were significant advances in biomarkers and recovery parameters, complication rates were not significantly different between groups. This implies that although dexmedetomidine provides demonstrable myocardial and systemic protection, but it

does not significantly attenuate the early postoperative complications (such as bleeding and multi-organ dysfunction).

This study was not powered to find a difference in mortality, but the results—two deaths in controls compared with none in the dexmedetomidine group—are consistent with reported trends. A particularly important point is that a large prospective trial with 1,134 patients demonstrated that dexmedetomidine use perioperatively halved the rates of 1-year mortality (3.17% versus 7.95%) (1). Likewise, dexmedetomidine reduces short-term mortality after adult cardiac surgery [24]. In contrast, large-scale studies have reported conflicting results; specifically, two multicenter RCTs concluded that perioperative dexmedetomidine did not reduce mortality or complications in adults undergoing cardiopulmonary bypass [39].

This study presents several limitations. The modest sample size constrained the statistical power necessary to detect differences in low-frequency outcomes, including mortality and severe complications. Additionally, the high incidence of temporary pacing suggests that standard dosing protocols may require downward adjustment or more precise titration alongside early chronotropic support. The single-center design further restricts the generalizability of these findings. Finally, the short follow-up period precluded the assessment of long-term endpoints, such as late mortality or major adverse cardiac events.

This study affirms dexmedetomidine in enhancing myocardial protection and early recovery after cardiac surgery. These outcomes can be maximized through careful hemodynamic management and adherence to guideline-based doses (0.2–0.7 μg/kg/h) titrated downward following intraoperative responses. These results highlight the need for physiologically guided, dynamic titration as opposed to fixed infusion rates - an approach most beneficial for very rapid (fast-track) strategies that favor early extubation.

Future studies should clarify the dose-response and effort optimization for hemodynamic protocols that curb bradycardia but maximize therapeutic benefit. In addition, definitive evidence of the effect on hard clinical endpoints can only be obtained in large-scale multicenter trials with longer follow-up.

Conclusion

Dexmedetomidine is a promising adjunct to fast-track cardiac surgery protocols, providing significant myocardial protection and early recovery. Larger, multicenter trials with longer follow-up are warranted to further determine the appropriate dosing strategies and longer-term clinical efficacy.

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