



DEXMEDETOMIDINE IN ALLEVIATING SUCCINYLCHOLINE INDUCED POST-OPERATIVE MYALGIA- A PROSPECTIVE RANDOMIZED STUDY

Anaesthesiology

Dr. Pooja Patil

Junior Resident, Department of Anaesthesiology, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, India

Dr. Shobha Yavagal*

Assistant Professor, Department of Anaesthesiology, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, India*Corresponding Author

Dr. Manjunath A.C

Professor, Department of Anaesthesiology, Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, India

ABSTRACT

Background: Succinylcholine, though widely used for rapid sequence induction, is associated with a high incidence of postoperative myalgia. Dexmedetomidine, an α_2 -adrenergic agonist, has sedative and analgesic properties which may be effective in attenuating succinylcholine-induced myalgia. Given the high prevalence of succinylcholine-induced myalgia and the potential benefits of dexmedetomidine, there is a clear need for further investigation. **Aim:** To evaluate the efficacy of dexmedetomidine in reducing succinylcholine-induced postoperative myalgia. **Methods:** A prospective, randomized, double-blind study was conducted on 60 ASA I-II patients undergoing elective surgery under general anesthesia. Patients were randomized into two groups using a computer-generated sequence with concealed allocation: Group D (dexmedetomidine 1 $\mu\text{g/kg}$ IV, n=30) and Group C (saline, n=30), administered 10 minutes before induction with propofol and succinylcholine. Myalgia was assessed at 30 minutes, 1 hour and 24 hours postoperatively using the Harvey scale. Hemodynamic parameters and adverse events were also recorded. **Results:** A total of 60 patients (30 per group) were analysed. The incidence of postoperative myalgia was lower in the dexmedetomidine group. At 30 minutes, myalgia occurred in 3.3% of patients in Group D versus 20.0% in Group C ($p = 0.107$). At 1 hour, the incidence was 3.3% demonstrating a significant reduction with dexmedetomidine ($p = 0.004$). At 24 hours, myalgia occurred in 6.7% of patients in Group D and 30.0% in Group C, which was also statistically significant ($p = 0.002$). The requirement for rescue analgesia was significantly lower in Group D compared with Group C (23.3%) ($p = 0.023$). Hemodynamic parameters remained comparable between groups, and no significant adverse events were observed.

KEYWORDS

Dexmedetomidine, Succinylcholine, Postoperative myalgia, α_2 -adrenergic agonist

INTRODUCTION

Succinylcholine is a depolarising neuromuscular blocking agent widely used during the induction of general anaesthesia due to its rapid onset of action and short duration of effect. These characteristics make it particularly useful for rapid sequence intubation and airway management. Despite its advantages, the use of succinylcholine is associated with several adverse effects, among which muscle fasciculations and postoperative myalgia are the most common complications. Postoperative myalgia can cause significant patient discomfort and may delay recovery, thereby affecting overall patient satisfaction and postoperative outcomes.

The incidence of succinylcholine-induced postoperative myalgia has been reported to be as high as 50%, with symptoms typically appearing within the first 24 hours after administration. The exact mechanism responsible for this myalgia is not completely understood, but it is believed to result from unsynchronised muscle contractions during fasciculations, leading to micro-trauma of muscle fibres and subsequent inflammatory responses. Various pharmacological agents such as non-depolarising neuromuscular blockers, non-steroidal anti-inflammatory drugs, pregabalin, and gabapentin have been investigated for preventing or reducing the severity of succinylcholine-induced myalgia, with varying degrees of success.

Dexmedetomidine is a highly selective α_2 -adrenergic receptor agonist that possesses sedative, analgesic, and sympatholytic properties. It acts by inhibiting central sympathetic outflow and reducing the release of norepinephrine, thereby producing analgesia and anti-inflammatory effects. These pharmacological properties may contribute to attenuation of succinylcholine-induced muscle fasciculations and postoperative myalgia. Previous studies have demonstrated that intravenous dexmedetomidine can reduce the incidence and severity of these adverse effects without significant haemodynamic compromise. Dexmedetomidine, due to its analgesic and anti-inflammatory properties, may offer a promising approach for reducing postoperative myalgia associated with succinylcholine administration. Therefore, the present study is designed to evaluate the efficacy of IV dexmedetomidine administered prior to induction of general anaesthesia in reducing the incidence and severity of succinylcholine-induced postoperative myalgia in patients undergoing elective surgery.

MATERIALS AND METHODS

This prospective, randomized study was conducted in the Department of Anaesthesiology at Sapthagiri Institute of Medical Sciences and Research Centre, Bengaluru, after obtaining approval from the Institutional Ethics Committee and written informed consent from all participants. The study was carried out over a period of three months from August 2025 to October 2025.

A total of 60 patients scheduled for elective surgical procedures under general anaesthesia were included in the study. Patients aged between 18 and 50 years and belonging to American Society of Anesthesiologists (ASA) physical status I or II were enrolled. Patients with pre-existing musculoskeletal disorders, those receiving chronic pain medications or antipsychotic drugs, patients with hypersensitivity to any of the study medications, and those with ASA physical status III or IV were excluded. Patients with conditions such as increased intracranial pressure, increased intraocular pressure, hepatic or renal impairment, hyperkalemia, dehydration, malignant hyperthermia, and pregnant women were also excluded from the study.

Based on previous literature, the sample size was calculated assuming a proportion of patients with no postoperative myalgia of 0.84 in the dexmedetomidine group and 0.48 in the control group, with a confidence interval of 95%, study power of 80%, and a margin of error of 5%. The required sample size was calculated as 26 patients per group, which was rounded to 30 patients in each group, giving a total sample size of 60.

The patients were randomly allocated into two groups of 30 each using a randomization technique. In Group D (Dexmedetomidine group), patients received intravenous dexmedetomidine at a dose of 1 $\mu\text{g/kg}$ administered 10 minutes prior to induction of anaesthesia. In Group C (Control group), patients received an equal volume of normal saline at the same time interval. The study drugs were prepared in identical syringes by an anaesthesiologist who was not involved in patient assessment to maintain blinding.

All patients underwent routine preoperative evaluation including investigations such as haemoglobin estimation, blood urea, serum creatinine, blood sugar, coagulation profile, blood grouping, and electrocardiography when indicated. On arrival in the operating room, standard monitoring including electrocardiography, pulse oximetry, and non-invasive blood pressure monitoring was instituted. Baseline vital parameters including heart rate and blood pressure were recorded,

and the average of three readings taken at two-minute intervals was documented.

An 18-gauge intravenous cannula was secured in all patients. Following administration of the study drug, patients were premedicated with intravenous fentanyl at a dose of 2 µg/kg. After preoxygenation with 100% oxygen, anaesthesia was induced with intravenous propofol 2 mg/kg. Muscle relaxation for intubation was achieved with intravenous succinylcholine 2 mg/kg, following which endotracheal intubation was performed.

Anaesthesia was maintained with a mixture of oxygen and air along with isoflurane at 1–2 minimum alveolar concentration. Intravenous vecuronium 0.08 mg/kg was administered as a loading dose for muscle relaxation, followed by intermittent doses of 0.02 mg/kg as required. Intraoperative haemodynamic parameters including heart rate and blood pressure were recorded at regular intervals of five minutes. Bradycardia (heart rate <45 beats per minute) was treated with atropine 0.6 mg intravenously, while hypotension (mean arterial pressure <65 mmHg) was managed with intravenous mephentermine.

At the end of surgery, neuromuscular blockade was reversed with intravenous neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg. Patients were extubated after adequate recovery of neuromuscular function. To avoid confounding factors in the assessment of muscle pain, intramuscular injections were avoided during the perioperative period. Postoperative myalgia was defined as muscle pain not related to the surgical procedure. Assessment was performed by an independent observer who was blinded to group allocation at 30 minutes, 1 hour, and 24 hours following surgery. The severity of myalgia was graded according to the Harvey et al. scale as follows: score 0 – no muscle pain; score 1 – mild stiffness localized to one area of the body; score 2 – spontaneous muscle pain or stiffness requiring analgesic therapy; and score 3 – severe, generalized, or incapacitating muscle pain.

STATISTICAL ANALYSIS

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) software version 22.0 (IBM Corp., Armonk, NY, USA). Quantitative variables were expressed as mean ± standard deviation, while categorical variables were presented as frequency and percentage. The Chi-square test was used to compare categorical variables such as gender distribution, ASA status, and incidence of myalgia between the two groups. Continuous variables such as age, weight, and haemodynamic parameters were analysed using the independent Student's t-test or Mann-Whitney U test as appropriate. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 60 patients were enrolled in the study and randomly allocated into two groups: Group D (Dexmedetomidine, n = 30) and Group C (Control, n = 30). There were no dropouts or protocol deviations, and all patients were included in the final analysis.

Demographic characteristics

The demographic profiles of the two groups were comparable with respect to age and ASA physical status (p = 0.970 and p = 0.760, respectively). The gender distribution was also similar (M/F: 19/11 in Group D vs 15/15 in Group C; p = 0.435). However, the mean weight was significantly higher in Group D (60.63 ± 11.48 kg) compared to Group C (50.80 ± 10.55 kg), with p = 0.001 (Table 1).

Table 1: Demographic details of patients

Variable	Group D (Dexmedetomidine) (n=30)	Group C (Control) (n=30)	P value
Age (years), mean (SD)	35.67 ± 11.34	35.57 ± 9.24	0.97
Weight (kg), mean (SD)	60.63 ± 11.48	50.80 ± 10.55	0.001*
Sex (M/F)	19 / 11	15 / 15	0.435
ASA physical status (I/II)	22 / 8	24 / 6	0.76

Myalgia at 30 Minutes

At 30 minutes postoperatively, the incidence of myalgia was lower in Group D compared to Group C. Only 1 patient (3.3%) in Group D experienced mild myalgia, whereas 6 patients (20.0%) in Group C

developed myalgia (mild: 3; moderate: 3). Although numerically lower in the dexmedetomidine group, the difference did not reach statistical significance ($\chi^2 = 4.48$, p = 0.107). The odds ratio (OR) for developing myalgia at 30 minutes was 0.138 (95% CI: 0.016 – 1.226), favouring dexmedetomidine.

Table 2: Myalgia at 30 Minutes

Myalgia score	Group D (n = 30)	Group C (n = 30)
0 – No myalgia	29	24
1 – Mild	1	3
2 – Moderate	0	3
3 – Severe	0	0
Chi-square test: ($\chi^2 = 4.48$, p = 0.107)		

Myalgia at 1 hour

At 1 hour, the incidence of myalgia was significantly lower in Group D compared to Group C. Only 1 patient (3.3%) in Group D experienced mild myalgia, whereas 11 patients (36.7%) in Group C developed myalgia (mild: 8; moderate: 3). This difference was statistically significant ($\chi^2 = 8.44$, p = 0.004). The odds ratio (OR) for developing myalgia at 1 hour was 0.060 (95% CI: 0.007 – 0.500), favouring dexmedetomidine.

Table 3: Myalgia at 1 hour

Myalgia score	Group D (n = 30)	Group C (n = 30)
0 – No myalgia	29	19
1 – Mild	1	8
2 – Moderate	0	3
3 – Severe	0	0
Chi-square test: $\chi^2 = 8.44$, p = 0.004		

Myalgia at 24 Hours

At 24 hours, the incidence of myalgia was lower in Group D compared to Group C. Only 2 patients (6.7%) in Group D reported myalgia, whereas 9 patients (30.0%) in Group C experienced myalgia (mild: 6; moderate: 2; severe: 1). This difference was statistically significant ($\chi^2 = 9.36$, p = 0.002).

Table 4: Myalgia at 24 Hours

Myalgia score	Group D (n = 30)	Group C (n = 30)
0 – No myalgia	28	21
1 – Mild	1	6
2 – Moderate	1	2
3 – Severe	0	1
Chi-square test: $\chi^2 = 9.36$, p = 0.002		

Rescue Analgesia Requirement

The requirement for rescue analgesia was lower in Group D compared to Group C. Only 1 patient (3.3%) in Group D required rescue analgesia, whereas 7 patients (23.3%) in Group C required additional analgesic treatment. This difference was statistically significant ($\chi^2 = 5.19$, p = 0.023). The odds ratio (OR) for requiring rescue analgesia was 0.11 (95% CI: 0.013 – 0.94), indicating a significantly reduced need for postoperative analgesia in patients who received dexmedetomidine.

DISCUSSION

The present study was conducted to evaluate the effectiveness of intravenous dexmedetomidine in reducing succinylcholine-induced postoperative myalgia in patients undergoing elective surgeries under general anaesthesia. Succinylcholine remains widely used for rapid sequence intubation because of its rapid onset and short duration of action; however, postoperative myalgia continues to be one of its most frequent adverse effects. The findings of the present study indicate that pre-treatment with dexmedetomidine reduces the incidence and severity of succinylcholine-induced postoperative myalgia when compared with placebo.

Dexmedetomidine is a highly selective α_2 -adrenergic receptor agonist with sedative, analgesic, and sympatholytic properties. It exerts its analgesic effect through inhibition of nociceptive transmission in the central nervous system and reduction in the release of substance P in dorsal horn neurons. In addition, dexmedetomidine possesses anti-inflammatory properties that may contribute to the attenuation of muscle pain caused by succinylcholine-induced fasciculations. These pharmacological actions may explain the reduction in postoperative

myalgia observed in patients who received dexmedetomidine premedication.

The results of the present study are consistent with the findings reported by Sriramka et al., who demonstrated that low-dose dexmedetomidine significantly reduced the incidence of succinylcholine-induced myalgia and fasciculations. Their randomized controlled trial showed that patients receiving dexmedetomidine experienced fewer symptoms compared with those receiving placebo. Similarly, Celebi et al. also reported that dexmedetomidine administration before induction of anaesthesia was associated with a reduction in early postoperative myalgia.

The haemodynamic effects of dexmedetomidine were also considered during the study period. Dexmedetomidine is known to produce sympatholysis and may lead to bradycardia or hypotension in some patients. However, in the present study, no clinically significant haemodynamic instability or major adverse effects were observed following its administration. This observation is consistent with previous studies which have shown that dexmedetomidine, when used in appropriate doses, maintains haemodynamic stability during the perioperative period. Overall, the findings of the present study suggest that dexmedetomidine administered prior to induction of anaesthesia can effectively reduce succinylcholine-induced postoperative myalgia without producing significant adverse effects. This supports the potential role of dexmedetomidine as a useful premedication in patients receiving succinylcholine during general anaesthesia.

CONCLUSION:

The present study demonstrates that intravenous dexmedetomidine administered prior to induction of anaesthesia effectively reduces the incidence and severity of succinylcholine-induced postoperative myalgia in patients undergoing surgery under general anaesthesia. Dexmedetomidine was well tolerated and did not produce significant haemodynamic instability. Therefore, dexmedetomidine may be considered a useful premedication to minimize postoperative myalgia associated with succinylcholine.

LIMITATIONS OF STUDY

The study was conducted in a single institution with a relatively small sample size. Additionally, the assessment of postoperative myalgia was limited to the early postoperative period. Larger multicentric studies with longer follow-up periods may help to further validate the findings.

REFERENCES

1. Sriramka B, Panigrahy S, Ramasubbu MK, Mishra SN. Dexmedetomidine for reducing succinylcholine-induced myalgia in patients undergoing electroconvulsive therapy: A randomised controlled trial. *Indian J Anaesth* 2024;68:560-5.
2. Schreiber J, Lysakowski C, Fuchs-Buder T, Tramer MR. Prevention of succinylcholine induced Fasciculations and myalgia. A Meta Analysis of randomized trials. *Anesthesiology* 2005;103:877-84
3. Hall JE, Uhrich TD, Barney JA, Arain SR, Ebert TJ. Sedative, amnestic, and analgesic properties of small-dose dexmedetomidine infusions. *Anesth Analg* 2000; 90: 699-705.
4. Arain SR, Ruehlw RM, Uhrich TD, Ebert TJ. The efficacy of dexmedetomidine versus morphine for postoperative analgesia after major inpatient surgery *Anesth Analg* 2004; 98: 153-158.
5. Wong SF, Chung F. Succinylcholine-associated postoperative myalgia. *Anaesthesia*. 2000 Feb;55(2):144-52. Review
6. Srivastava VK, Agrawal S, Nimbhorkar VK, Mishra A, Sharma S, Panda PK. Prophylactic use of pregabalin for prevention of succinylcholine-induced fasciculation and myalgia: a randomized, double-blinded, placebo-controlled study. *Braz J Anesthesiol*. 2016 Mar-Apr;66(2):165-70. doi:10.1016/j.bjane.2014.08.004. Epub 2014 Nov 27.
7. Pandey CK, Karna ST, Tandon M, Pandey VK, Singh A. Comparative evaluation of prophylactic use of pregabalin, gabapentin and diclofenac sodium for prevention of succinylcholine-induced myalgia: a randomized, double-blinded study. *J Postgrad Med*. 2014 Jan-Mar;60(1):16-20. doi: 10.4103/0022-3859.128801
8. Kahraman S, Ercan S, Ayar U, Erdem K. Effect of preoperative i.m. administration of diclofenac on suxamethonium-induced myalgia. *Br J Anaesth* 1993; 71:238-241