e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(11); 445-448

Original Research Article

Evaluation of two Different Doses of Dexmedetomidine Infusion on Oxygenation, Lung Mechanics in Morbid Obese Patients: A Prospective Study

Sushma Singh[1], P. Kamesh Chandra[2], Nirvi Sharma[3]

[1]Assistant Professor, Dept of Anaesthesia, GMC, KORBA CHHATTISGARH, India
[2]Senior Consultant Anaesthesiologist, Department of Anaesthesiology and Critical Care, KIMS Seethammadhara, Visakhapatnam, Andhra Pradesh, India
[3]Professor, Department of Occupational Therapy, Jaipur Occupational Therapy College, Jaipur, Rajasthan, India

Received: 25-08-2024 / Revised: 23-09-2024 / Accepted: 26-10-2024
Corresponding Author: Dr. Sushma Singh
Conflict of interest: Nil

Abstract:

Background: Dexmedetomidine, a selective alpha-2 adrenoceptor agonist, is increasingly used in anesthesia for its sedative and analgesic effects without respiratory depression. In morbidly obese patients, respiratory management is challenging due to reduced lung compliance and oxygenation. This study evaluates the effect of two different doses of dexmedetomidine infusion on oxygenation and lung mechanics in morbidly obese patients undergoing surgery.

Materials and Methods: A prospective study was conducted on 60 morbidly obese patients (BMI ≥ 40 kg/m²) scheduled for elective surgery under general anesthesia. Patients were randomized into two groups: Group A received a dexmedetomidine infusion of 0.4 µg/kg/hr, and Group B received 0.7 µg/kg/hr after an initial loading dose of 1 µg/kg over 10 minutes. Oxygenation parameters (PaO_2, SpO_2) and lung mechanics (peak inspiratory pressure, compliance) were recorded at baseline, after induction, and hourly during surgery. Statistical analysis was performed using a two-way ANOVA, with significance set at $p < 0.05$.

Results: Group B showed a statistically significant improvement in PaO_2 levels compared to Group A, with mean values of 95 ± 10 mmHg versus 88 ± 12 mmHg ($p = 0.03$). Peak inspiratory pressure was lower in Group B (22 ± 3 cm H_2O) than in Group A (25 ± 4 cm H_2O), indicating better lung compliance ($p = 0.02$). No significant difference in heart rate or mean arterial pressure was observed between the two groups.

Conclusion: A higher dose of dexmedetomidine infusion (0.7 µg/kg/hr) improved oxygenation and lung mechanics in morbidly obese patients without adverse cardiovascular effects. This dose may offer a safer anesthetic regimen for this patient population, enhancing respiratory outcomes during surgery.

Keywords: Dexmedetomidine, Morbid Obesity, Oxygenation, Lung Mechanics, General Anesthesia, Respiratory Compliance.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

The management of anesthesia in morbidly obese patients presents unique challenges, particularly in maintaining adequate oxygenation and lung compliance due to the altered respiratory mechanics in this population [1]. Morbid obesity, defined as a body mass index (BMI) of 40 kg/m² or higher, is associated with reduced functional residual capacity, increased airway resistance, and decreased lung compliance, which collectively complicate ventilation and increase the risk of perioperative hypoxemia [2,3].

Various pharmacologic agents have been explored to improve respiratory mechanics in these patients, yet few provide the balance of sedation, analgesia, and respiratory stability needed during surgery [4]. Dexmedetomidine, a selective alpha-2 adrenoceptor agonist, has gained popularity in anesthetic practice due to its sedative, analgesic, and anxiolytic properties without significant respiratory depression [5]. It acts on the central nervous system by inhibiting norepinephrine release, providing sedation with minimal respiratory compromise, which is particularly advantageous for morbidly obese patients [6,7].

Studies have shown that dexmedetomidine reduces the requirement for other anesthetic agents and facilitates smoother recovery, but its specific effects on oxygenation and lung mechanics in the morbidly obese population remain underexplored [8]. In animal and clinical studies,

dexmedetomidine has demonstrated beneficial effects on oxygenation and lung compliance by reducing inflammatory responses and improving hemodynamic stability [9,10]. However, the optimal dosing strategy to achieve these outcomes without adverse effects has yet to be determined. While some studies suggest a lower infusion dose may be sufficient, others propose that a higher dose may further enhance lung mechanics and oxygenation, particularly in patients with compromised respiratory function [11,12].

This prospective study aims to evaluate the effects of two different doses of dexmedetomidine infusion on oxygenation and lung mechanics in morbidly obese patients undergoing general anesthesia. By comparing the impact of a standard dose versus a higher dose of dexmedetomidine, this study seeks to determine an optimal dose that improves respiratory outcomes while maintaining hemodynamic stability in this high-risk population.

Materials and Methods

A total of 60 morbidly obese patients (BMI ≥ 40 kg/m²), aged 18–60 years, scheduled for elective surgical procedures under general anesthesia were enrolled. Inclusion criteria included patients classified as ASA (American Society of Anesthesiologists) physical status II or III. Patients with known hypersensitivity to dexmedetomidine, severe cardiac or pulmonary disease, or requiring emergency surgery were excluded. Written informed consent was obtained from all participants.

Randomization and Group Allocation

Patients were randomly allocated into two groups (Group A and Group B) using a computer-generated randomization table. Group A received a dexmedetomidine infusion at a dose of 0.4 µg/kg/hr, while Group B received 0.7 µg/kg/hr following an initial loading dose of 1 µg/kg administered over 10 minutes.

Anesthesia Protocol: All patients underwent standard preoperative evaluation and fasting guidelines. Premedication with oral ranitidine (150 mg) and alprazolam (0.5 mg) was given the night before surgery. In the operating room, baseline hemodynamic parameters, including heart rate (HR), mean arterial pressure (MAP), and oxygen saturation (SpO_2), were recorded. Anesthesia was induced with intravenous propofol (2 mg/kg), fentanyl (2 µg/kg), and vecuronium bromide (0.1 mg/kg) to facilitate endotracheal intubation. Maintenance anesthesia included sevoflurane (1.5–2.0%), 50% oxygen in air, and intermittent doses of vecuronium.

Intervention

After induction, patients in Group A received dexmedetomidine infusion at 0.4 µg/kg/hr, and those in Group B received 0.7 µg/kg/hr. The infusion was continued throughout the surgery. Ventilation was adjusted to maintain end-tidal CO_2 between 35 and 40 mmHg.

Outcome Measures

The primary outcomes were oxygenation parameters (arterial oxygen partial pressure [PaO_2] and peripheral oxygen saturation [SpO_2]) and lung mechanics (peak inspiratory pressure [PIP] and dynamic lung compliance [Cdyn]). These parameters were recorded at baseline, after induction, and hourly during surgery. Secondary outcomes included hemodynamic stability, assessed by HR and MAP.

Sample Size Calculation

The sample size was calculated to detect a minimum difference of 10 mmHg in PaO_2 between groups with 80% power and a significance level of 0.05. Considering a dropout rate of 10%, 30 patients per group were required.

Statistical Analysis

Data were analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Comparisons between groups were performed using an independent t-test for continuous variables and the chi-square test for categorical variables. A p-value of <0.05 was considered statistically significant.

Results

The study enrolled 60 patients, with 30 in each group (Group A and Group B). The demographic characteristics, including age, gender distribution, and BMI, were comparable between the two groups ($p > 0.05$). The results of oxygenation parameters, lung mechanics, and hemodynamic stability are summarized below.

Table 1: Demographic Characteristics

Parameter	Group A (n = 30)	Group B (n = 30)	p-value
Age (years)	45 ± 10	47 ± 12	0.56
Gender (Male/Female)	18/12	17/13	0.82
BMI (kg/m²)	42.5 ± 3.2	43.0 ± 3.5	0.43

Table 2: Oxygenation Parameters

Time point	PaO_2 (mmHg) Group A	PaO_2 (mmHg) Group B	SpO_2 (%) Group A	SpO_2 (%) Group B	p-value
Baseline	88 ± 8	89 ± 7	97 ± 2	97 ± 2	0.74
After Induction	85 ± 10	90 ± 9	96 ± 2	98 ± 1	0.03*
1 Hour After Surgery	88 ± 9	95 ± 10	96 ± 2	98 ± 1	0.02*

***p < 0.05 indicates statistical significance.**

Table 3: Lung Mechanics

Time point	PIP (cm H_2O) Group A	PIP (cm H_2O) Group B	Cdyn (mL/cm H_2O) Group A	Cdyn (mL/cm H_2O) Group B	p-value
Baseline	28 ± 4	27 ± 5	35 ± 5	36 ± 6	0.65
After Induction	26 ± 3	24 ± 4	37 ± 4	40 ± 5	0.04*
1 Hour After Surgery	25 ± 4	22 ± 3	38 ± 4	42 ± 6	0.03*

***p < 0.05 indicates statistical significance.**

Table 4: Hemodynamic Parameters

Time point	HR (bpm) Group A	HR (bpm) Group B	MAP (mmHg) Group A	MAP (mmHg) Group B	p-value
Baseline	78 ± 5	79 ± 6	85 ± 8	84 ± 9	0.81
After Induction	74 ± 4	73 ± 5	82 ± 7	81 ± 8	0.64
1 Hour After Surgery	76 ± 4	75 ± 5	83 ± 6	82 ± 7	0.72

Key Findings:

1. **Oxygenation:** Group B showed significantly higher PaO_2 levels and SpO_2 percentages after induction and 1 hour after surgery ($p < 0.05$).
2. **Lung Mechanics:** Peak inspiratory pressure (PIP) was lower, and dynamic lung compliance (Cdyn) was higher in Group B compared to Group A at all postoperative time points ($p < 0.05$).
3. **Hemodynamic Stability:** There was no significant difference in heart rate (HR) or mean arterial pressure (MAP) between the two groups ($p > 0.05$).

These findings suggest that a higher dose of dexmedetomidine infusion (0.7 µg/kg/hr) significantly improves oxygenation and lung mechanics in morbidly obese patients during surgery while maintaining hemodynamic stability.

Discussion

The findings of this study demonstrate that a higher dose of dexmedetomidine infusion (0.7 µg/kg/hr) significantly improves oxygenation and lung mechanics in morbidly obese patients undergoing surgery, compared to a lower dose (0.4 µg/kg/hr). These results support the use of dexmedetomidine as a promising adjunct to enhance respiratory outcomes in this high-risk population.

Morbidly obese patients often present with reduced functional residual capacity, increased airway resistance, and impaired oxygenation, which complicate perioperative respiratory management [1]. Dexmedetomidine, a selective alpha-2 adrenergic agonist, has been shown to exert protective effects on pulmonary function through its anti-inflammatory, sedative, and sympatholytic properties [2]. The significant improvement in PaO_2 and SpO_2 observed in Group B aligns with previous research, which highlights the role of dexmedetomidine in enhancing oxygenation by reducing shunt fraction and improving ventilation-perfusion matching [3].

Lung mechanics were also significantly improved with the higher dose of dexmedetomidine, as evidenced by lower peak inspiratory pressure (PIP) and higher dynamic compliance (Cdyn). These findings are consistent with earlier studies that reported enhanced pulmonary compliance and reduced airway pressures with dexmedetomidine administration during mechanical ventilation [4,5]. The probable mechanism involves its ability to modulate inflammatory responses and reduce airway hyperreactivity, which are critical factors in improving lung mechanics [6].

Despite these respiratory benefits, hemodynamic stability was maintained in both groups, with no significant differences in heart rate (HR) or mean arterial pressure (MAP). This is particularly noteworthy, as higher doses of dexmedetomidine are sometimes associated with bradycardia or hypotension due to its sympatholytic effects [7].

The absence of significant hemodynamic disturbances in this study suggests that the selected dose range is both effective and safe for morbidly obese patients. Previous studies comparing varying doses of dexmedetomidine have highlighted the need for dose optimization to balance its benefits and side effects. For example, Bao et al. [8] observed that a higher infusion dose (0.7 µg/kg/hr) resulted in superior oxygenation compared to lower

doses in patients with compromised lung function, corroborating our findings. Similarly, Wu et al. [9] reported improved lung compliance and reduced inflammatory markers with dexmedetomidine in thoracic surgeries, further supporting its role in respiratory protection. However, our study has certain limitations. The relatively small sample size and single-center design may limit the generalizability of the findings.

Additionally, the study focused only on intraoperative outcomes; long-term effects of dexmedetomidine on respiratory recovery and overall clinical outcomes were not evaluated. Future research with larger, multicentric trials and extended follow-up periods is warranted to confirm these findings and explore the broader implications of dexmedetomidine use in obese patients.

Conclusion

This study demonstrates that a higher dose of dexmedetomidine infusion (0.7 µg/kg/hr) improves oxygenation and lung mechanics in morbidly obese patients during surgery without compromising hemodynamic stability.

These findings provide valuable insights into optimizing perioperative respiratory management in this challenging patient population.

References

1. Biring MS, Lewis MI, Liu JT, Mohsenifar Z. Pulmonary physiologic changes of morbid obesity. Am J Med Sci. 1999; 318(5):293-7.
2. Salome CM, King GG, Berend N. Physiology of obesity and effects on lung function. J Appl Physiol. 2010; 108(1):206-11.
3. Pelosi P, Gregoretti C. Perioperative management of obese patients. Best Pract Res Clin Anaesthesiol. 2010; 24(2):211-25.
4. Mahmoud M, Mason KP. Dexmedetomidine: review, update, and future considerations of pediatric perioperative and periprocedural applications. Anesth Analg. 2015; 121(6):1704-14.
5. Zhang X, Li D, Cai H, Zhu L. Effects of dexmedetomidine on lung injury in septic patients. Int J Clin Exp Med. 2015; 8(4):5820-7.
6. Gerlach AT, Dasta JF. Dexmedetomidine: an updated review. Ann Pharmacother. 2007; 41(2):245-52.
7. Afonso J, Reis F. Dexmedetomidine: current role in anesthesia and intensive care. Rev Bras Anestesiol. 2012; 62(1):118-33.
8. Bao R, Li A, Shao Q. Effects of dexmedetomidine on lung mechanics and oxygenation in mechanically ventilated morbidly obese patients. J Anesth. 2019; 33(3):419-26.
9. Wu X, Lu Y, Zhang X. Protective effects of dexmedetomidine on pulmonary function in patients undergoing thoracoscopic surgery. Medicine (Baltimore). 2017;96(35)
10. Ma H, Zhang G, Mou Z. The effect of dexmedetomidine on respiratory function in patients undergoing laparoscopic surgery. Acta Anaesthesiol Scand. 2013; 57(10):1195-202.
11. Sanders RD, Maze M. Alpha2-adrenoceptor agonists. Curr Opin Investig Drugs. 2007; 8(1):25-33.
12. Riker RR, Shehabi Y, Bokesch PM, et al. Dexmedetomidine vs midazolam for sedation of critically ill patients: a randomized trial. JAMA. 2009; 301(5):489-99.