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Comparison of the Effect of Induction of General Anesthesia with Propofol Vs Ketamine + Midazolam on Perfusion Index in Cardiac Patients Undergoing Non-Cardiac Surgery

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ABSTRACT

Hemodynamic stability is a critical concern in cardiac patients. The choice of induction agent significantly influences cardiac output and tissue perfusion. **Objectives:** To compare the effect of induction with ketamine and midazolam vs. propofol on perfusion index in cardiac patients under general anesthesia. **Methods:** This quasi-experimental study was done from January 2024 to June 2024 at the Department of Anesthesia, Mayo Hospital, Lahore. Total 146 patients undergoing non-cardiac surgery were included after taking written consent. Patients were assigned to two equal Groups: M (midazolam and ketamine) or Group P (propofol). Outcome parameters, including SBP, MAP, and perfusion index, were recorded pre- and post-induction. Data were collected using a proforma and analyzed with SPSS-26. **Results:** Post-induction, mean SBP in Group M was higher, 126.02 ± 12.58 mmHg, vs Group P, 120.61 ± 13.09 mmHg, p-value=0.01. Post-induction mean MAP in Group M and P calculated was 74.17 ± 10.06 mmHg, and 71.02 ± 9.82 mmHg, p-value=0.050. Post-induction PI in Group M was also higher, 1.88 ± 0.40, vs Group P, 1.69 ± 0.28, p-value=0.001. **Conclusions:** Findings of the current study suggest that induction with ketamine and midazolam may lead to increased post-induction mean perfusion index as compared to propofol in cardiac patients. Therefore, assists in maintaining peripheral perfusion in cardiac patients, which could be beneficial for postoperative recovery and minimizing complications.

INTRODUCTION

Modern surgery requires general anesthesia to perform intricate procedures while maintaining patient comfort and safety [1]. Because it establishes the foundation for steady hemodynamics and adequate tissue perfusion throughout the procedure, the induction phase is very crucial. Selecting the right induction agent is crucial for cardiac patients, since excellent outcomes depend on maintaining optimum perfusion [2]. Due to their underlying cardiovascular problems, cardiac patients might become more complex during the perioperative phase and pose particular challenges [3]. Cardiovascular stability and tissue oxygenation, two important factors, are greatly influenced by the induction agent. The perfusion index serves as an indicator of peripheral perfusion and tissue

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oxygenation [4]. An increase in PI suggests enhanced peripheral blood flow and oxygenation, while a decrease may indicate compromised perfusion [5]. A prior review examined how different induction agents affect hemodynamics in patients with IHD. Overall, all agents caused significant drops in blood pressure, heart rate, and cardiac index. However, the midazolam group uniquely avoided increases in blood pressure during induction [6]. Propofol, a lipid-soluble, non-opioid, non-barbiturate sedative-hypnotic with a quick onset and brief duration of action, is commonly used for induction. Propofol can have a substantial influence on the cardiovascular system despite its advantages. Patients with congenital heart disease may be more susceptible to arrhythmias. Propofol's suppression of the sympathetic nervous system and disruption of baroreflex-regulating systems, which are critical for preserving cardiovascular stability, are blamed for these cardiovascular consequences [7]. Ketamine is an anesthetic that has hypnotic, amnestic, and analgesic effects. It has a powerful analgesic impact with no cardiovascular or respiratory depression. However, because it tends to enhance myocardial oxygen demand and its arrhythmogenic effects, utilizing ketamine alone for induction in cardiac patients raises concerns [8]. A benzodiazepine with a quick onset and short half-life, midazolam has hypnotic, amnestic, anticonvulsant, and anxiolytic effects [9]. Propofol can be used effectively at lower doses when combined with ketamine to reduce its negative effects. According to a prior study, midazolam and ketamine together produced more hemodynamic stability compared to midazolam and propofol, although both combinations had comparable results in terms of post-anesthesia recovery and patient comfort [10].

Although previous studies have compared the hemodynamic effects of various induction agents in cardiac populations, most have focused on systemic parameters rather than direct measures of peripheral tissue perfusion. Therefore, this study aims to address the knowledge gap by investigating the effect of induction with ketamine and midazolam compared to propofol on perfusion index in cardiac patients undergoing general anesthesia.

METHODS

This quasi-experimental study was conducted after obtaining ethical approval from the IRB (367/RC/KEMU) of King Edward Medical University. Sample size of 146 (73 in each group) was estimated at 5% significance level and 95% power, based on the expected mean perfusion index of 1.5 ± 1.3 in the ketamine and midazolam group and 2.4 ± 2.4 in the propofol group [11]. This study was done from January 2024 to June 2024, and for patient enrollment, a non-probability consecutive sampling method was used. Written informed consent was obtained from patients before enrollment. Patients aged 40 to 65 years of both genders, belonging to ASA I or II, and diagnosed as cardiac patients undergoing elective non-cardiac surgery under general anesthesia were included. Cardiac patients were defined as having a history/medical record suggestive of CAD, ECG showing ischemic changes (ST segment depression/T wave inversions/LBBB/RBBB/Q waves), or Echocardiography with EF < 45% / segmental wall motion abnormalities. Patients with congenital or valvular heart disease, Raynaud's disease, uncontrolled diabetes mellitus or hypertension, known allergies to study drugs, those on vasoactive drugs such as calcium channel blockers or ARBs, individuals with substance abuse, and those suffering from severe systemic non-cardiac disease were excluded. Each patient's demographic data was recorded. A detailed medical history and thorough clinical examination were performed, and all relevant baseline investigations were done to ensure compliance with inclusion and exclusion criteria. Patients were assigned to two equal groups according to assigned drug: Group M (ketamine and midazolam) or Group P (propofol). All patients were kept NPO for at least eight hours before induction. Upon arrival in OT, the IV line was secured, and standard monitoring was established. A pulse oximeter sensor was attached to the forefinger of one hand, while noninvasive BP monitoring was applied to the opposite arm. A core body temperature probe was also attached to prevent hypothermia-induced vasoconstriction. Perfusion index, HR, BP, and MAP were recorded during the pre-induction phase. ECG and SpO_2 monitoring were initiated, and baseline vital signs were documented. Patients were preoxygenated with 100% oxygen at 6 L/min and premedicated with IV nalbuphine 0.1 mg/kg and dexamethasone 0.1 mg/kg. Induction in Group M was performed using intravenous ketamine 0.5 mg/kg combined with midazolam 0.4 mg/kg, whereas Group P received propofol 2 mg/kg administered slowly until loss of verbal response. After confirming adequate bag-mask ventilation, muscle relaxation was achieved with atracurium IV 0.5 mg/kg. Patients were ventilated with 100% oxygen and isoflurane at 1.2 MAC for five minutes, during which data were recorded for all parameters at five minutes post-induction, similar to pre-induction measurements. PI was recorded using a pulse oximeter before and 5 minutes after induction. It was measured after 5 minutes of induction. Endotracheal intubation was performed by an experienced anesthetist using an appropriately sized tube, and proper placement was confirmed by auscultation and end-tidal CO_2 monitoring. Mechanical ventilation was maintained with VT 6–8 mL/kg, RR 12 breaths/minute, I: E 1:2, and oxygen 2 L/min, while anesthesia was maintained with oxygen and isoflurane.

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MAP below 60 mmHg or systolic BP below 90 mmHg was considered severe hypotension and managed with intravenous fluids and adrenaline as required. Bradycardia, defined as a decrease in heart rate by 20% below baseline, was treated with atropine. Intraoperative management continued as necessary throughout the procedure. At the end of surgery, anesthesia was reversed with IV neostigmine (0.03mg/kg) and atropine (0.01mg/kg). After satisfactory recovery, patients were extubated and shifted to PACU, where they were observed for two hours. All data were recorded on a predesigned proforma.

Data were investigated in SPSS version 26. Normality of continuous variables was assessed using the Shapiro–Wilk test before analysis. Quantitative variables were presented as mean + standard deviation. Qualitative variables as frequency and percentage. The data were stratified by age, gender, BMI, and ASA classification. After stratification, comparisons of PI between groups were conducted using an independent samples t-test; a p-value of <0.050 was considered statistically significant.

RESULTS

Both groups were compared in terms of demographic characteristics. The mean age in Groups M and P was calculated to be 54.68 ± 7.00 years and 54.34 ± 7.32 years (p>0.05). Gender distribution found 49.3% males and 50.7% females in Group M and 50.7% males and 49.3% females in Group P (p>0.050). Mean BMI was similar between groups (25.76 ± 2.74 kg/m^2 vs. 25.28 ± 3.17 kg/m^2; p>0.050). Distribution of ASA physical status was also comparable, with 54.8% of patients classified as ASA I and 45.2% as ASA II in Group M, compared to 58.9% and 41.1%, respectively, in Group P (p>0.050) (Table 1).

Table 1: Comparison of Baseline Characteristics among Study Groups

Variables	Group M (Midazolam + Ketamine), n=73, Mean ± SD/n (%)	Group P (Propofol), n=73, Mean ± SD/ n (%)	p-value
Age (Years)	54.68 ± 7.00	54.34 ± 7.32	0.775
Male	36 (49.3%)	37 (50.7%)	<0.001
Female	37 (50.7%)	36 (49.3%)	
BMI (kg/m^2)	25.76 ± 2.74	25.28 ± 3.17	0.329
ASA Status I	40 (54.8%)	43 (58.9%)	0.738
ASA Status II	33 (45.2%)	30 (41.1%)	

Comparison of hemodynamic parameters revealed no significant difference in pre-induction systolic BP (p=0.94), MAP (p=0.45), and perfusion index (p=0.57). However, post-induction SBP was significantly higher in Group M (126.02 ± 12.58 mmHg) compared to Group P (120.61 ± 13.09 mmHg; p=0.01). MAP was also significantly higher in the Midazolam + Ketamine group (74.17 ± 10.06 mmHg) vs the Propofol group (71.02 ± 9.82 mmHg; p=0.05). Post induction, a significant increase in PI was observed in Group M (1.88 ± 0.40) compared to Group P (1.69 ± 0.28; p=0.001) (Table 2).

Table 2: Comparison of Hemodynamic Parameters among Groups

Variables	Phase	Group M (Midazolam + Ketamine) n=73 (Mean ± SD)	Group P (Propofol) n=73 (Mean ± SD)	Mean Difference (95% CI)	p-value
Systolic Blood Pressure (mmHg)	Pre-Induction	128.35 ± 12.24	128.49 ± 12.09	−0.14 (−4.03 to 3.75)	0.940
	Post-Induction	126.02 ± 12.58	120.61 ± 13.09	5.41 (1.18 to 9.64)	0.001*
Mean Arterial Pressure (mmHg)	Pre-Induction	75.82 ± 10.34	77.12 ± 10.73	−1.30 (−4.70 to 2.10)	0.450
	Post-Induction	74.17 ± 10.06	71.02 ± 9.82	3.15 (−0.12 to 6.42)	0.050*
Perfusion Index (PI)	Pre-Induction	1.60 ± 0.25	1.57 ± 0.28	0.03 (−0.06 to 0.12)	0.570
	Post-Induction	1.88 ± 0.40	1.69 ± 0.28	0.19 (0.08 to 0.30)	0.001*

*Statistically significant at p<0.050

Stratified analysis of post-induction perfusion index demonstrated significant differences in specific subgroups. Among males, Group M showed significantly higher PI values compared to Group P (p=0.004), while in females the difference was not statistically significant (p=0.11). When stratified by ASA status, significant differences were observed in both ASA I (p=0.040) and ASA II (p=0.01) patients, favoring the Midazolam + Ketamine group. With respect to age, patients aged ≤50 years (p=0.050) and those aged >50–65 years (p=0.006) both demonstrated significantly higher PI values in Group M. Similarly, when analyzed by BMI, patients with BMI >25–30 kg/m^2 showed significant difference (p=0.004), whereas those with BMI between 20–<25 kg/m^2 did not (p=0.080) (Table 3).

Table 3: Stratified Comparison of Post-Induction Perfusion Index (PI) Between Study Groups

Stratification Variables	Category	Group M (Midazolam + Ketamine) Mean ± SD	Group P (Propofol) n=73 (Mean ± SD)	Mean Difference (95% CI)	p-value	Interaction p-value
Gender	Male	1.92 ± 0.41	1.68 ± 0.29	0.24 (0.08 to 0.40)	0.004*	0.030*
	Female	1.83 ± 0.38	1.72 ± 0.27	0.11 (−0.03 to 0.25)	0.110	
ASA Status	I	1.90 ± 0.39	1.70 ± 0.26	0.20 (0.01 to 0.39)	0.040*	0.020*
	II	1.86 ± 0.42	1.65 ± 0.30	0.21 (0.06 to 0.36)	0.010*	

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Age (years)	≤50	1.84 ± 0.37	1.70 ± 0.25	0.14 (0.00 to 0.28)	0.050*	0.010*
	>50–65	1.91 ± 0.43	1.66 ± 0.30	0.25 (0.08 to 0.42)	0.006*	
BMI (kg/m^2)	20–<25	1.82 ± 0.36	1.73 ± 0.27	0.09 (–0.02 to 0.20)	0.080	0.040*
	≥25–30	1.94 ± 0.44	1.65 ± 0.29	0.29 (0.10 to 0.48)	0.004*	

DISCUSSION

Poor peripheral perfusion has been associated with poor surgical outcomes according to recent studies [12]. Improved peripheral perfusion enhances tissue oxygenation, which is essential for better wound healing, reduced infection rates, and decreased in-hospital mortality [13]. In the present study, post-induction mean perfusion index was significantly higher in Group M (ketamine + midazolam) at 1.88 ± 0.40 compared to Group P (propofol) at 1.69 ± 0.28 (p=0.001), indicating better peripheral perfusion with the ketamine–midazolam combination. These findings are consistent with Zhou *et al.* who reported that PI values remained consistently higher in patients receiving ketamine than those receiving propofol at several time points—during induction (mean difference = 1.01, p=0.007), pre-intubation (mean difference = 1.46, p=0.001), and post-intubation (mean difference = 1.28, p=0.010) [11]. Similarly, Mohamed *et al.* also observed an overall increase in PI with both dexmedetomidine and midazolam, though the difference was not significant (p=0.610), suggesting that sedative agents can improve PI to varying extents [14]. In contrast, Nakasuji and colleagues found that PI measurements after intravenous propofol induction remained comparable between high- and low-risk groups during the initial three minutes, with increases becoming evident only at four and five minutes post-induction (4.5 ± 1.7 vs. 6.1 ± 2.4 and 4.3 ± 1.8 vs. 6.5 ± 2.5, respectively). This finding reflects propofol's transient and delayed vasodilatory effect, which may explain the comparatively lower PI observed in our propofol group [15]. Ketamine's ability to maintain blood pressure and heart rate can counteract the depressant effects of midazolam, potentially leading to more stable hemodynamic parameters during anesthesia induction and maintenance [16, 17]. In the current study, both SBP and MAP were significantly higher in Group M (p=0.010 and p=0.050, respectively) as compared to Group P. Afghaniyan *et al.* also reported that propofol significantly decreases systolic and mean arterial blood pressure (p<0.001), particularly in patients undergoing CABG, as compared to midazolam [18]. Atchley *et al.* conducted a study on high-risk patients in the ICU and found similar results, showing that propofol was associated with significantly higher rates of hypotension (p<0.001) and bradycardia (p<0.001) compared with ketamine combined with dexmedetomidine [19]. Baysal *et al.* conducted a study on children who underwent cardiac catheterization and found that propofol use led to significant decreases in mean arterial pressure (p=0.001) and oxygen saturation, whereas ketamine/midazolam maintained more stable hemodynamic parameters with non-significant changes in mean arterial pressure (p=0.544) and a moderate reduction in heart rate [20].

Limitations of the current study include the relatively small sample size, which may limit the generalizability of the findings to a broader cardiac patient population. Additionally, variations in individual responses to anesthetic agents due to co-morbidities and baseline hemodynamic status may also have influenced the results. Finally, the use of non-invasive monitoring for perfusion index may not capture all hemodynamic changes compared to invasive measurements.

CONCLUSIONS

Findings of the current study suggest that induction with ketamine and midazolam may lead to increased post-induction mean perfusion index as compared to propofol in cardiac patients. These findings highlight the potential of using ketamine and midazolam for improving or maintaining peripheral perfusion in cardiac patients, which could be beneficial for postoperative recovery and minimizing complications.

Authors' Contribution

Conceptualization: MAK, FA
Methodology: FA, SY, SB
Formal analysis: SA
Writing and Drafting: SA, MF
Review and Editing: MAK, FA, SY, SB, SA, MF, HMUM

All authors approved the final manuscript and take responsibility for the integrity of the work

Conflicts of Interest

All the authors declare no conflict of interest.

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REFERENCES

[1] Linassi F and Kreuzer M. General Anesthesia Research. Anesthesia and Analgesia. 2020 Jul; 131(1): e13-4. doi: 10.1213/ANE.0000000000004824.

[2] Nordine M, Pille M, Kraemer J, Berger C, Brandhorst P, Kaeferstein P *et al.* Intraoperative Beat-to-Beat Pulse Transit Time (PTT) Monitoring Via Non-Invasive Piezoelectric/Piezocapacitive Peripheral Sensors Can Predict Changes in Invasively Acquired Blood Pressure in High-Risk Surgical Patients. Sensors. 2023 Mar; 23(6): 3304. doi: 10.3390/s23063304.

DOI: https://doi.org/10.54393/pjhs.v7i5.3654

[3] Russotto V, Tassistro E, Myatra SN, Parotto M, Antolini L, Bauer P *et al.* Peri-Intubation Cardiovascular Collapse in Patients Who Are Critically Ill: Insights from the INTUBE Study. American Journal of Respiratory and Critical Care Medicine. 2022 Aug; 206(4): 449-58. doi: 10.1164/rccm.202111-2575OC.

[4] Coutrot M, Dudoignon E, Joachim J, Gayat E, Vallee F, Depret F. Perfusion Index: Physical Principles, Physiological Meanings and Clinical Implications in Anesthesia and Critical Care. Anesthesia, Critical Care and Pain Medicine. 2021 Dec; 40(6): 100964. doi: 10.1016/j.accpm.2021.100964.

[5] Mostafa H, Shaban M, Hasanin A, Mohamed H, Fathy S, Abdelreheem HM *et al.* Evaluation of Peripheral Perfusion Index and Heart Rate Variability as Early Predictors for Intradialytic Hypotension in Critically Ill Patients. BioMed Central Anesthesiology. 2019 Dec; 19(1): 242. doi: 10.1186/s12871-019-0917-1.

[6] Vitovskyi AR and Loskutov OA. Changes in Hemodynamic Parameters with Different Anesthesia Induction Agents in Elderly Patients with Coronary Heart Disease. Emergency Medicine. 2024 Dec; 20(7): 594-600. doi: 10.22141/2224-0586.20.7.2024.1778.

[7] Li B, Pop C, Johnson M, Sklar MC, Lawler PR, Elliott AM. Hemodynamic Effects of Propofol. Journal of the American College of Cardiology: Advances. 2025 Aug; 4(8): 101769. doi: 10.1016/j.jacadv.2025.101769.

[8] Goddard K, Sampson C, Ghadban R, Stilley J, Goddard KB, Bedy SM. Effect of Ketamine on Cardiovascular Function During Procedural Sedation of Adults. Cureus. 2021 Mar; 13(3). doi: 10.7759/cureus.14228.

[9] Mihalj M, Karlović Z, Vladić-Spaić D, Matić B, Mikulić I, Mikulić V *et al.* Effects of Midazolam Co-Induction to General Anesthesia: A Randomized Clinical Trial. Medicine. 2022 Nov; 101(45): e31400. doi: 10.1097/MD.0000000000031400.

[10] Uludağ Ö, Doğukan M, Kaya R, Tutak A, Dumlupınar E. Comparison of the Effects of Midazolam-Ketamine or Midazolam-Propofol Combinations on Hemodynamic Stability, Patient Comfort, and Post-Anesthesia Recovery in Children Undergoing Sedation for Magnetic Resonance Imaging Procedures. Ain-Shams Journal of Anesthesiology. 2020 Jan; 12(1). doi: 10.1186/s42077-019-0037-7.

[11] Zhou N, Liang X, Gong J, Li H, Liu W, Zhou S *et al.* S-ketamine Used During Anesthesia Induction Increases the Perfusion Index and Mean Arterial Pressure After Induction: A Randomized, Double-Blind, Placebo-Controlled Trial. European Journal of Pharmaceutical Sciences. 2022 Dec; 179: 106312. doi: 10.1016/j.ejps.2022.106312.

[12] Højlund J, Petersen DR, Agerskov M, Foss NB. The Peripheral Perfusion Index Discriminates Hemodynamic Responses to Induction of General Anesthesia. Journal of Clinical Monitoring and Computing. 2023 Dec; 37(6): 1533-40. doi: 10.1007/s10877-023-01035-z.

[13] Højlund J, Agerskov M, Clemmesen CG, Hvolris LE, Foss NB. The Peripheral Perfusion Index Tracks Systemic Hemodynamics During General Anesthesia. Journal of Clinical Monitoring and Computing. 2020 Dec; 34(6): 1177-84. doi: 10.1007/s10877-019-00420-x.

[14] Mohamed Atef Refaat M, Ali Elkafrawy L, Elkabarity RH, Hafez AF. Effect of Dexmedetomidine vs Midazolam on the Microcirculation of Septic Patients Who Are Mechanically Ventilated. Egyptian Journal of Anesthesia. 2022 Dec; 38(1): 459-65. doi: 10.1080/11101849.2022.2109826.

[15] Nakasuji M and Nakasuji K. Causes of Arterial Hypotension During Anesthetic Induction with Propofol Investigated with Perfusion Index and Clearsighttm in Young and Elderly Patients. Minerva Anestesiologica. 2021 Mar; 87(6): 640-7. doi: 10.23736/S0375-9393.21.15226-5.

[16] Amri I, Arif SK. Perbandingan Efek Deksmedetomidin 0, 75 µg/kgBB Dengan Fentanil 2 µg/kgBB Intravena Terhadap Kebutuhan Dosis Induksi Propofol Dan Respon Hemodinamik Selama Tindakan Laringoskopi Dan Intubasi Trakhea. Jurnal Kesehatan Tadulako. 2017 Jan; 3(1): 1-84.

[17] Smischney NJ, Seisa MO, Morrow AS, Ponce OJ, Wang Z, Alzuabi M *et al.* Effect of Ketamine/Propofol Admixture on Peri-Induction Hemodynamics: A Systematic Review and Meta-Analysis. Anesthesiology Research and Practice. 2020; 2020(1): 9637412. doi: 10.1155/2020/9637412.

[18] Afghaniyan P, Farhadian M, Tarbiat M, Bakhshaei MH, Salimbahrami SA. Comparing the Hemodynamic Effects of Midazolam, Etomidate, And Propofol Following Anesthesia Induction in Coronary Artery Bypass Graft Surgery: A Double-Blind Randomized Clinical Trial. The Journal of Tehran University Heart Center. 2024 Apr; 19(2): 89. doi: 10.18502/jthc.v19i2.16197.

[19] Atchley E, Tesoro E, Meyer R, Bauer A, Pulver M, Benken S. Hemodynamic Effects of Ketamine Compared with Propofol or Dexmedetomidine as Continuous ICU Sedation. Annals of Pharmacotherapy. 2022 Jul; 56(7): 764-72. doi: 10.1177/10600280211051028.

[20] Baysal A, Polat TB, Yalcin Y, Celebi A. The Use of Basic Parameters for Monitoring the Hemodynamic Effects of Midazolam and Ketamine as Opposed to Propofol During Cardiac Catheterization. Cardiology in the Young. 2014 Apr; 24(2): 351-8. doi: 10.1017/S1047951108001935.