e-ISSN: 3023-6738

Eurasian Journal of

Anesthesiology & Intensive Care

Volume: 3 Issue: 2 Year: 2026

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Original Article

doi
10.51271/EAJAIC-0050

Evaluation of the effectiveness of Pleth Variability Index (PVI) and Internal Jugular Vein Collapsibility Index (IJV-CI) measurement methods in predicting hypotension following spinal anesthesia

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Cite this article: Akgül GÖ, Akçaboy ZN, Akçay F, Yalçın Yılmaz G. Evaluation of the effectiveness of Pleth Variability Index (PVI) and Internal Jugular Vein Collapsibility Index (IJV-CI) measurement methods in predicting hypotension following spinal anesthesia. *Eur J Anesthesiol Intens Care.* 2026;3(2):27-33. doi:10.51271/EAJAIC-0050

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Received: 13/08/2025 ◆ **Accepted**: 23/11/2025 ◆ **Published**: 16/05/2026

ABSTRACT

Aims: Hypotension following spinal anesthesia is a significant issue in clinical practice. The aim of this study is to evaluate the efficacy of Pleth Variability Index (PVI) and Internal Jugular Vein Collapsibility Index (IJV-CI) in predicting the risk of hypotension that may develop in patients undergoing spinal anesthesia while breathing spontaneously.

Methods: After ethics committee approval, 116 patients scheduled for elective orthopedic surgery in the supine position under spinal anesthesia were included. Hypotension was defined as a drop in mean arterial pressure (MAP) below 60 mmHg or more than 20% below the baseline value. Patients who developed hypotension were designated as Group A, and those who remained normotensive as Group B. Preoperative and intraoperative PVI and IJV-CI values were measured for all patients and effectiveness of these values in predicting the risk of hypotension compared.

Results: Hypotension was observed in 69 (59.5%) patients. No statistically significant differences were observed between the groups in the comparison of PVI, and IJV-CI values at any time interval. No correlation between MAP values and PVI, and IJV-CI values at baseline (T0) and post-spinal block (T1) time points was identified.

Conclusion: Our findings demonstrated that neither pleth PVI nor IJV-CI exhibited significant efficacy in predicting hypotension among spontaneously breathing patients.

Keywords: Post-spinal hypotension, Pleth Variability Index, Internal Jugular Vein Collapsibility Index, spontaneous breathing

INTRODUCTION

Spinal anesthesia is commonly used in surgical operations to be performed in the pelvis, lower abdomen, and lower extremities. Hypotension is observed in approximately 15.3% to 33% after spinal anesthesia and can lead to decreased perfusion in organs and ischemic conditions.[1] This hypotension results from a decrease in systemic vascular resistance due to sympathetic blockade[2] and has been associated with increased mortality. To prevent hypotension and mitigate its adverse effects, recommended measures include fluid loading, patient repositioning, and the use of positive inotropic agents.[3,4] In patients with low cardiac reserve or in those who cannot tolerate hypotension or would be adversely affected by it, the ability to predict post-spinal anesthesia hypotension is crucial. Early appropriate intervention in such patients can reduce intraoperative and postoperative complications.[5]

Continuous monitoring of physiological parameters with basic hemodynamic monitoring is mandatory during anesthesia. However, these methods alone are not sufficient to predict the risk of hypotension and cannot directly assess oxygen delivery or cardiac output (CO). Proper fluid management is very important when ensuring intraoperative stability, and it is faster and easier to use non-invasive methods during fluid management. Pulse oximetry plethysmographic waveform variables and ultrasonography (USG) can be used for this purpose.

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The Perfusion Index (PI) generated from pulse oximetry is used to interpret peripheral perfusion dynamics due to changes in peripheral vascular tone. It is calculated as the ratio of pulsatile blood flow to non-pulsatile blood flow in peripheral tissue. Many studies have found that a higher baseline PI is associated with a greater decrease in blood pressure and the need for higher doses of positive inotropes.[6]

The Perfusion Index (PVI) is the automated measurement of dynamic changes in the PI, particularly those influenced by respiration. Numerous studies have suggested that PVI can be used as a dynamic parameter for goal-directed fluid therapy in mechanically ventilated patients. While many studies have focused on mechanically ventilated patients, research on spontaneously breathing patients is limited. Since spinal anesthesia-induced hypotension can be related to the patient's volume status, pre-spinal anesthesia measurement of PVI may be used to predict patients at risk for hypotension.[7]

Evaluation of the inferior vena cava (IVC) and internal jugular vein (IJV) using USG is among the recently preferred predictive methods for assessing patients' volume status due to its non-invasive, rapid, and complication-free nature. During spontaneous breathing, inspiration causes negative intrathoracic pressure, increasing venous return and right ventricular filling. There are a limited number of studies examining the efficacy of IJV USG in predicting post-spinal hypotension.[4]

The aim of this study is to evaluate the efficacy of PVI and Internal Jugular Vein Collapsibility Index (IJV-CI), which are non-invasive and dynamic measurement methods, in predicting the risk of hypotension that may develop in patients undergoing spinal anesthesia with spontaneous respiration.

METHODS

The study was conducted with the approval of the Ankara City Hospital No. 2 Clinical Researches Ethics Committee (Date: 29.09.2021, Decision No: E2-21-874). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. 116 patients, aged 18-65 years (ASA class I-II) who were scheduled for elective orthopedic surgery under spinal anesthesia in the supine position were included and written informed consent was obtained from all participants. Patients with an ASA score of 3 or higher, a body mass index of 40 or higher, history of cardiac arrhythmia, peripheral vascular disease, severe heart failure, pregnancy, emergency surgery, a position other than supine, sedation requirement and unsuccessful spinal block were excluded from the study.

The patients were taken to the operating table and rested in the supine position for 5 minutes. Standard monitoring with a GE Healthcare Carescape B 450® monitor was applied including non-invasive arterial blood pressure measurement, five-lead electrocardiogram. Oxygen saturation measurement was done with Masimo Radical-7 Pulse CO-Oximeter®, and probe placed on the index finger of the dominant hand. Time intervals for measurements were determined as follows: baseline values (T0), after spinal anesthesia in supine position (T1), at the 5th (T5), 10th (T10), 15th (T15), 30th (T30), 60th (T60) minutes. After the resting period, baseline heart rate, non-invasive systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), peripheral oxygen saturation (SpO2), PI and PVI were measured and recorded.

All ultrasonographic measurements were performed by the same anesthesiologist using the same ultrasound device (Sonosite S-Nerve®), while patients were breathing spontaneously in the supine position. Using a linear probe, the right IJV was visualized at the level of the cricoid cartilage, using B mode in a transverse plane perpendicular to the skin, without applying pressure to the jugular vein as much as possible. The IJV was verified using compression and color Doppler sampling. Dynamic diameter changes were measured using M-mode over a 20-second period of spontaneous respiration, recording maximum (end-expiration) and minimum (end-inspiration) dimensions. The right IJV-CI was calculated using the formula:

Collapsibility Index (%)=

[(max IJV diameter-min IJV diameter)/max IJV diameter]×100

Venous access was established through a 20-gauge intravenous catheter on the non-dominant hand, but no pre-spinal fluid loading was performed. Immediately following spinal block administration, intravenous infusion of 0.9% NaCl at 10 ml/kg/hr was initiated. Spinal anesthesia was performed in the sitting position, after disinfection and sterile draping, at the L3-4 or L4-5 intervertebral space using a 25-Gauge spinal needle (Braun®), administering 12.5 mg of 0.5% hyperbaric bupivacaine. The patient was then positioned supine and remained in the supine position throughout the procedure. The level of sensory block was assessed using the pinprick test up to T6-T8 level. Motor block was evaluated using the Bromage Scale. Measurements were conducted at predetermined intervals and recorded throughout the operation.

Hypotension was defined as a decrease in MAP below 60 mmHg or a reduction of more than 20% from baseline. Patients who developed hypotension were classified as Group A, while those who did not were classified as Group B. When MAP fell below 60 mmHg, 5 mg ephedrine was administered, and when heart rate dropped below 50 bpm, 0.5 mg atropine was given.

Statistical Analysis

G Power 3.1.9.2 Package program was used for sample size calculation before starting the study. Based on a previous study,[8] it was calculated that a total of 112 patients should be included in the study with an effect size of d=0.26, 80% power and 0.05 error level, assuming that a 2.3 point difference between the preoperative PVI values of patients who developed and did not develop hypotension would be considered significant.

IBM SPSS Statistics 20 software was used for statistical analysis. Mean±standard deviation and median values

were presented for continuous data while frequencies and percentages were presented for categorical data. The Shapiro-Wilk test was used to evaluate the normality of continuous variables. For comparison of continuous variables between two groups, the t-test was used for normally distributed data, while the Mann-Whitney U test was used for non-normally distributed data. For comparisons across three groups, the Kruskal-Wallis test was employed, and the source of any detected differences was explored using Kruskal-Wallis multiple comparison test. Friedman test was used to compare the repeated values measured during the operation (at T0, T1, T5, T10, T15, T30, T60 times) and followed by a post-hoc analysis via Friedman multiple comparison test. Factors affecting the decrease in MAP were analyzed using Multivariate Logistic Regression analysis. A value of $p<0.05$ was accepted as statistically significant.

RESULTS

A total of 116 patients who underwent elective orthopedic surgery in the supine position under spinal anesthesia were included in the study, and all patients were analyzed properly. Hypotension was observed in 69 patients. Patients were divided into two groups as those who developed hypotension (Group A, n=69) and those who did not (Group B, n=47). No statistical difference was observed in comparisons of age, gender, body weight, height, BMI, surgical duration, and fasting duration between the groups. Only ASA scores were found to be higher in Group A, which resulted in a statistically significant difference ($p=0.025$). Evaluation of patients' comorbidities revealed a significant difference between the groups only in terms of diabetes mellitus ($p=0.011$), whereas no significant differences were observed for hypertension or coronary artery disease. (Table 1).

Table 1. Comparison of demographic data, surgical duration, fasting duration, asa scores, and comorbidities between groups

		Group A (n=69)	Group B (n=47)	p
Age (years)		48.39±15.33	43.91±14.84	0.095
Weight (kg)		75.42±14.01	78.36±16.17	0.300
Height (m)		1.66±0.08	1.67±0.10	0.342
BMI (kg/m²)		27.87±5.17	28.40±4.99	0.580
Duration of surgery minute		88.41±18.99	88.19±15.65	0.989
Fasting duration (hr)		8.10±0.38	8.17±0.43	0.704
Gender	Female	45 (65.2%)	25 (53.2%)	0.194
	Male	24 (34.8%)	22 (46.8%)	
ASA	I	28 (40.6%)	29 (61.7%)	0.025
	II	41 (59.4%)	18 (38.3)	
HT	No	38 (55.1%)	30 (63.8)	0.347
	Yes	31 (44.9)	17 (36.2)	
DM	No	52 (75.4)	44 (93.6)	0.011
	Yes	17 (24.6)	3 (6.4)	
CAD	No	67 (97.1)	46 (97.9)	1.000
	Yes	2 (2.9)	1 (2.1)	

Data are shown as mean±SD or number (percentages) of patients. BMI: Body-mass index, hr: Heart rate, ASA: American Society of Anesthesiologists, HT: Hypertension, DM: Diabetes mellitus, CAD: Coronary artery disease

No statistically significant differences were observed between the groups in terms of block level ($p=0.776$) and the level of spinal anesthesia administration ($p=0.342$).

In Group A, who developed hypotension and bradycardia, ephedrine was administered to 20 patients, while atropine was administered to 5 patients.

Comparisons of baseline (T0) MAP values between the groups were statistically significant ($p=0.001$), with MAP values being higher in Group A patients. However, when comparing baseline (T0) PI, PVI, and IJV-CI values between the groups, no statistical difference was observed, despite the values being higher in Group A patients (Table 2).

Table 2. Comparison of baseline (T0) MAP, PI, PVI and IJV-CI values of patients between groups

	Group A (n=69)	Group B (n=47)	p
T0-MAP	108.46±11.42	100.06±13.33	0.001
T0 PI	2.36±1.83	2.33±1.42	0.636
T0 PVI	16.96±6.52	15.62±5.81	0.299
T0 IJV-CI	22.48±9.81	19.87±10.11	0.187

Data are shown as mean±SD (standard derivation), MAP: Mean arterial pressure, PI: Perfusion Index, PVI: Pleth Variability Index, IJV-CI: Internal Jugular Vein Collapsibility Index

The time intervals during which patients' MAP decreased by more than 20% and/or remained below 60 mmHg are given in Figure 1. It was observed that 65.2% of the patients experienced a decrease after spinal block (T1) compared to T0.

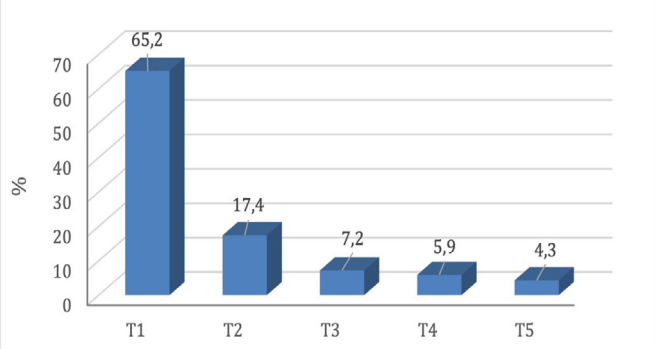

Figure 1. Distribution of hypotension onset times

No statistically significant differences were observed between the groups in the comparison of PI, PVI, and IJV-CI values at any time interval throughout the procedure (Figure 2).

Based on studies[9] that found a higher incidence of hypotension in patients with PVI values >15, we re-evaluated the T0- PVI values in our study patients, categorizing them as those with PVI values of 15 and above, and those below 15. No difference was found in the rate of hypotension between groups. In a study[10] where IJV-CI was used to evaluate fluid responsiveness, a cutoff point of 36% was calculated for IJV-CI. When we re-evaluated our study patients as those with a T0- IJV-CI value equal or greater than 36 and those with T0-IJV-CI<36, no statistically significant difference was found between the groups in terms of hypotension rates (Table 3).

In patients categorized as hypotensive (Group A), an investigation was conducted to determine the presence of a correlation between MAP values and PI, PVI, and IJV-CI values at baseline (T0) and post-spinal block (T1) time points (which is the most common time for hypotension). No statistically significant correlation was identified (Table 4).

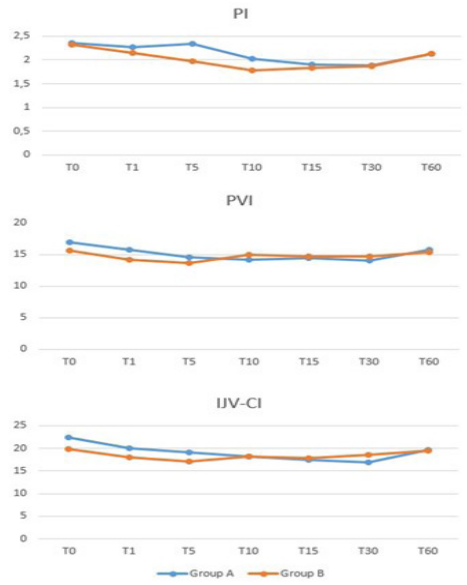

Figure 2. PI, PVI and IJV-CI values in groups
PI: Perfusion Index, PVI: Pleth Variability Index, IJV-CI: Internal Jugular Vein Collapsibility Index

Table 3. Comparison of hypotension rates according to the determined cut-off values of T0-PVI (<15/≥15) and initial IJV-CI (<36%/≥36%)

	Group A (n=69)		Group B (n=47)		p
	n	%	n	%	
T0-PVI <15	30	56.6	23	43.4	0.562
T0-PVI ≥15	39	61.9	24	38.1	
T0-IJV-CI <36%	64	59.3	44	40.7	0.486
T0-IJV-CI ≥36%	5	62.5	3	37.5	

PVI: Pleth Variability Index, IJV-CI: Internal Jugular Vein Collapsibility Index

Table 4. Correlation analysis between MAP and PI, PVI, and IJV-CI values in hypotensive patients (Group A)

		T0 PI	T0 PVI	T0 IJV-CI
T0 MAP	r	0.027	0.063	-0.024
	p	0.828	0.605	0.846
		T1 PI	T1 PVI	T1 SIJV-CI
T1 MAP	r	-0.025	0.152	-0.195
	p	0.841	0.213	0.107

MAP: Mean arterial pressure, PI: Perfusion Index, PVI: Pleth Variability Index, IJV-CI: Internal Jugular Vein Collapsibility Index

In order to examine the risk factors affecting the decrease in MAP, the independent variables age, gender, ASA score, presence of DM, and T0 IJV-CI values, which were found to be significant in the univariate analysis (variables with p<0.20), were included in the multivariate logistic regression analysis and the final model was obtained. The effect of these variables on the decrease in MAP was not found to be significant (Table 5).

Table 5. Multivariate logistic regression analysis of factors associated with decrease in mean arterial pressure

Factors	Regression coefficient (SE)	OR	95 % CI		p
Age (>50)	-0.010 (0.019)	1.010	0.829	1.058	0.576
Gender (female)	0.394 (0.445)	1.482	0.608	3.615	0.378
ASA II	0.888 (0.605)	2.429	0.742	7.959	0.143
DM	1.227 (0.730)	3.411	0.815	14.273	0.093
T0 IJV-CI	0.038 (0.021)	1.039	0.996	1.083	0.073

SE: Standard error, OR: Odds ratio, CI: Confidence interval, ASA: American Society of Anesthesiologists, DM: DM: Diabetes mellitus, IJV-CI: Internal Jugular Vein Collapsibility Index

DISCUSSION

In this study, we aimed to evaluate the predictive capacity of noninvasive measurements PI, PVI and IJV-CI, in the prediction of hypotension, which is an important complication in spinal anesthesia. Our findings revealed that these parameters were not effective in predicting hypotension in spontaneously breathing patients.

In recent years, many studies have been conducted on parameters that would allow predicting post-spinal hypotension in order to prevent hypotension, which is one of the most common and serious complications of spinal anesthesia. It should be kept in mind that if fluid deficit is present which is not uncommon, considering the long fasting periods in the preoperative period for many patients, then post-spinal hypotension frequently occurs. While attempting to correct the fluid deficit, it is necessary to avoid excessive volume overload especially in patients with heart failure. Therefore, it is important to assess the intravascular volume status accurately, quickly, and without complications before administering anesthesia. Many measurement methods can be used for hemodynamic monitoring. Information about CO can be obtained with invasive methods such as arterial catheter, central venous catheter, and pulmonary artery catheter, but the application of these invasive methods takes time and has risks of infection and complications. In addition, PVI and IJV-CI measurement methods can also be used for non-invasive evaluation of the patient's intravascular volume status. Due to its high accessibility and the limited number of studies available in the current literature, this study aimed to evaluate the effectiveness of IJV-CI measurements by comparing them with PVI measurements.

In studies, the incidence of hypotension after spinal anesthesia administration has been reported to be between 15.3% and 33%.[1] This variation is attributable to the different definitions of hypotension, different local anesthetics and their doses chosen, and differences in the level of intervention. In our study, similar to Kılıç et al.,[4] hypotension was defined as a decrease of 20% or more in MAP compared to the baseline value and/or a decrease in MAP below 60 mmHg. As in the study of Kılıç et al.,[4] the incidence of post-spinal hypotension was observed at a high rate of 59.5% in our study, and we believe this is due to our definition of hypotension.

In recent studies, pulse oximetry, a dynamic method for hemodynamic monitoring of peripheral perfusion, has been increasingly used. In particular, the parameters PI and PVI have been monitored to predict fluid responsiveness.[11] It has been reported that by monitoring peripheral perfusion trends with PI and PVI measurements, postoperative hypotension can be predicted and prevented.[12-15] Based on these studies, we aimed to evaluate the baseline PI and PVI values of patients to predict preoperative residual volume status, which is one of the significant causes of post-spinal hypotension.

In their study using pulse oximetry to predict post-spinal hypotension, Yokose et al.[16] found that PVI and PI values of hypotensive patients were higher than those of non-hypotensive patients, similar to our study's findings, but noted that this difference was not statistically significant.

Sun et al.[17] investigated the PI and PVI parameters to predict post-spinal anesthesia hypotension and found that the baseline PVI values of patients who developed hypotension were higher, but did not observe a significant difference in baseline PI. Additionally similar to the findings of our study, they concluded that PVI had poor diagnostic accuracy, stating that it was not a useful predictor of hypotension.

In a study conducted by Küpeli et al.[8] on the geriatric patient population, the incidence of post-spinal hypotension was observed at a high rate of 56.4%, similar to our findings. Similarly, it was found that baseline PVI values were higher in patients who developed post-spinal hypotension, but it was reported that this finding did not reach statistical significance.[8]

In their study investigating whether preoperative volume status assessment could predict the risk of hypotension, Kuwata et al.[18] found that 64% of patients undergoing spinal anesthesia for cesarean section developed hypotension. To predict hypotension, they determined a PVI threshold value of 18, after spinal anesthesia rather than at baseline. Consequently, the researchers reported that PVI after spinal anesthesia was a good predictor of post-spinal hypotension in patients undergoing caesarean delivery.

Yüksek et al.[9] evaluated the ability of PVI values in the geriatric patient population to predict hypotension that may develop after general anesthesia and found a higher incidence of hypotension in patients with a baseline PVI value >15.45%. In their study to predict hypotension that may occur after general anesthesia induction, Tsuchiya et al.[7] emphasized that patients with a preoperative PVI value of 15 or higher had a >25 mmHg decrease in MAP, suggesting that PVI could predict hypotension. Based on these studies, when we re-evaluated the patients in our study by grouping them as those with T0-PVI values of 15 and above and those below 15, we could not find a significant difference in the incidence of hypotension between the groups.

The use of USG in assessing intravascular volume status has increased in recent years due to its non-invasive nature, bedside applicability, and low cost. In particular, the ultrasonographic evaluation of the IVC for this purpose, using collapsibility and distensibility index values calculated from respiratory variations in diameter, has been shown to be beneficial in the management of critically ill patients by guiding selected intravascular hydration strategies.[19]

Ceruti et al.[20] performed IVC-CI measurements to determine the amount of volume replacement before spinal anesthesia and thus prevent hypotension. They determined the fluid replacement cut-off point as 36%, based on the results in the literature. In patients with IVC-CI greater than 36%, they found a slight correlation between the increase in post-spinal IVC-CI and the decrease in MAP, despite adequate fluid administration before spinal anesthesia.

In their study, Jaremko et al.[21] performed IVC-ex, IVC-in, and IVC-CI measurements before spinal anesthesia and after administering 500 ml of isotonic saline infusion to predict post-spinal hypotension, and divided the patients into two groups as hypotensive and non-hypotensive. They reported that, no statistically significant differences were detected between changes in IVCex, IVCin, and IVC-CI comparing hypotensive and non-hypotensive patients at the baseline and after the interventions. Additionally according to ROC analysis, IVC-CI is found not effective in the prediction of severe hypotension during spinal anesthesia in spontaneously breathing patients. In our study, instead of IVC measurements, we aimed to investigate whether IJV-max, IJV-min, and IJV-CI parameters could be used to predict post-spinal hypotension, based on the premise that extrathoracic veins can provide information about intrathoracic pressure and volume changes, and therefore performed measurements on the right IJV. Similar to Jaremko's study, there was no statistically significant difference in baseline measurements between the hypotensive and non-hypotensive groups. This could be because intravascular volume status is not the sole determinant of blood pressure; changes in systemic vascular resistance, which are closely related to sympathetic activity, are also major determinants of hypotension. Additionally, in both studies, the effects of respiratory variations on vascular diameter may not have been evaluated as effectively as in mechanical ventilation due to the patients' spontaneous breathing.

In their study, Mačiulienė et al.[22] measured IVC-in and IVC-ex before and after spinal anesthesia administration in patients undergoing elective knee arthroplasty, and divided the patients into hypotensive and non-hypotensive groups. No statistically significant changes were shown in IVC-in, IVC-ex, and IVC-CI measurements between these groups, compared to baseline and other time points. However, similar to our study, they found a higher collapsibility index in the hypotensive group. Consistent with our study, they concluded that measuring venous diameters before spinal anesthesia was not a predictor of hypotension.

In their study evaluating fluid responsiveness in 34 mechanically ventilated patients, Iizuka et al.[23] measured IJV, SCV, and IVC diameters and collapsibility values. Subsequently, they performed a passive leg-raising test on the patients. An 8% increase in stroke volume, calculated by arterial pulse contour analysis, was considered indicative of fluid responsiveness, and the patients were divided into two groups to examine the role of venous measurements in predicting fluid responsiveness. They showed that right IJV collapsibility was higher in the fluid-responsive group and found a threshold value of 11.4%. In our study, which was conducted on patients with equal fasting periods and the same fluid strategy, patients in the post-spinal hypotension group had a higher collapsibility index, as Iizuka also reported. Although this difference was not statistically significant, we believe that administering fluid before the procedure to patients with a high collapsibility index before spinal anesthesia could reduce the incidence of hypotension.

Haliloğlu et al.[10] demonstrated that in spontaneously breathing septic patients exhibiting fluid responsiveness, both the IVC and internal jugular vein collapsibility index (IJV-CI) values were significantly elevated. They also reported that IJV-CI could be used to assess volume responsiveness in non-mechanically ventilated patients. Furthermore, a cutoff value of 36% was calculated for IJV-CI. In our current study, we re-evaluated whether the proportion of patients with IJV-CI

>36% differed between groups. However, we did not find a statistically significant difference.

Similar to our study, Kılıç et al.,[4] in their investigation evaluating the efficacy of IJV in predicting post-spinal hypotension in spontaneously breathing patients, reported a 46.8% incidence of hypotension. In contrast to other studies in literature[10,23] and also to our findings, this study demonstrated lower collapsibility index values in hypotensive patients compared to non-hypotensive patients.

Limitations

This single-center study was limited to patients undergoing orthopedic surgical procedures, which may restrict the generalizability of the results. Furthermore, intraoperative blood loss was not considered as a variable, and this factor could have affected the incidence of intraoperative hypotension.

CONCLUSION

This study aimed to evaluate the predictive accuracy of the PVI, derived from plethysmographic waveforms, and the IJV-CI, measured via non-invasive USG, for the incidence of hypotension following spinal anesthesia. While prior investigations have indicated potential utility in mechanically ventilated or specific surgical populations, our findings demonstrate that neither PVI nor IJV-CI may reliably predict hypotension in spontaneously breathing patients. Consequently, large-scale, prospective studies are needed to validate these observations and explore the integration of other hemodynamic and clinical variables to refine preoperative risk stratification.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the approval of the Ankara City Hospital No. 2 Clinical Researches Ethics Committee (Date: 29.09.2021, Decision No: E2-21-874).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights-including the right to withdraw at any time without consequence. All participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: GÖA; Design: ZNA; Control: GÖA; Data Collection and/or Processing: GÖA, GYY; Analysis and/or Interpretation: ZNA; Literature Review: GYY; Article Writing: ; Critical Review: All Authors.

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MHA MEDIHEALTH ACADEMY

Eurasian Journal of
Anesthesiology & Intensive Care

Original Article

doi
10.51271/EAJAIC-0051

Comparative impact of general and epidural anesthesia on postoperative outcomes in percutaneous nephrolithotomy

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Cite this article: Telli S, Özenç E, Kepekçi AB. Comparative impact of general and epidural anesthesia on postoperative outcomes in percutaneous nephrolithotomy. Eur J Anesthesiol Intens Care. 2026;3(2):34-40. doi:10.51271/EAJAIC-0051

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Received: 10/11/2025 ◆ **Accepted**: 23/12/2025 ◆ **Published**: 16/05/2026

ABSTRACT

Aims: This study aimed to compare the effectiveness and safety of epidural anesthesia (EA) versus general anesthesia (GA) in patients undergoing percutaneous nephrolithotomy (PCNL).

Methods: This retrospective observational study included 50 patients who underwent PCNL under either EA or GA. The groups were compared regarding demographic data, intraoperative and postoperative parameters, including hemodynamic stability, bleeding, complications, Visual Analog Scale (VAS) pain scores, and postoperative analgesic requirements.

Results: No significant differences were found between groups in terms of demographics, stone location, or size. Fluid requirement was significantly higher in the EA group ($p<0.01$). Although intraoperative bleeding and hypotension were more frequent in the GA group, differences were not statistically significant. Peak heart rate at the 10th minute was significantly higher in the GA group ($p<0.05$), and systolic and diastolic blood pressures were lower at some time points ($p<0.05$). Oxygen saturation was significantly higher in the GA group at the 5th minute only ($p<0.01$). Postoperative VAS scores were significantly lower in the EA group ($p<0.01$), and analgesic need was higher in the GA group. Ephedrine use was greater in the EA group ($p<0.05$), with no significant difference in atropine use or catheter site.

Conclusion: EA is a safe and effective alternative to GA for PCNL procedures, as it provides superior postoperative pain control, reduces analgesic requirements, and maintains hemodynamic stability. Although EA is associated with higher fluid and ephedrine requirements, its overall benefits make it a viable option for appropriately selected patients.

Keywords: Epidural anesthesia, general anesthesia, hemodynamic stability, percutaneous nephrolithotomy, postoperative pain control

INTRODUCTION

Urinary stone disease is a common health problem affecting 1% to 20% of the global population, with a higher prevalence in men.[1,2] Over the past four decades, the need for open surgery in kidney stone treatment has markedly declined, with minimally invasive techniques such as extracorporeal shock wave lithotripsy (SWL), percutaneous nephrolithotomy (PCNL), retrograde intrarenal surgery (RIRS), and laparoscopic approaches emerging as effective alternatives. Introduced by Fernström and Johansson[3] in 1976, PCNL has since become the standard surgical treatment for renal stones, replacing open surgery in many cases. Advances in technology and surgical experience have further refined the PCNL technique, leading to fewer complications, shorter hospital stays, and improved clinical outcomes.[4,5]

PCNL can be performed safely under general anesthesia (GA) or regional techniques such as spinal (SA), epidural (EA), or combined spinal-epidural anesthesia (CSEA). While GA ensures airway protection and facilitates hemodynamic control, it carries potential risks including neuroendocrine stress responses, airway trauma, pulmonary complications, allergic reactions, nerve injuries, and postoperative nausea

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and vomiting.[6,7] In contrast, regional anesthesia preserves spontaneous breathing and airway reflexes, provides effective postoperative analgesia, facilitates early mobilization, reduces the risk of thromboembolism, and shortens hospital stay.[7,8] In addition, it has also been shown to be superior to GA in reducing postoperative mortality and serious complications.[9,10]

The literature shows that EA provides a similar duration of anesthesia in patients undergoing PCNL while avoiding the disadvantages of GA. Therefore, this study aims to compare the effects of EA and GA in patients undergoing PCNL on surgical outcomes, hemodynamic changes, pain levels, analgesic requirements within the first 24 hours post-surgery, and the frequency of complications.

METHODS

Ethics

The study included 50 patients who underwent PCNL surgery, with 27 receiving GA and 23 receiving EA, all managed by the same surgical and anesthesia teams. This study has been approved by the Clinical Researches Ethics Committee of Haseki Training and Research Hospital (Date: 05.02.2014, Decision No: 55). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Participants

This retrospective observational study included patients who underwent PCNL for renal calculi at the Urology Clinic of a Training and Research Hospital between June 2012 and June 2014. Patients aged between 18 and 75 years and had an ASA score of ≤III, were included in the study. Exclusion criteria included patients with serious cardiac conditions such as bradycardia, advanced heart block, sick sinus syndrome, cardiogenic shock, significant heart failure (ejection fraction <30%), symptomatic mitral or aortic valve disease, or cardiac tamponade. Additional exclusion criteria were hypovolemia, liver failure, acute intermittent porphyria, coagulopathies, progressive neurological diseases, and, in the EA group, failure to achieve a sensory block level of T6 within 30 minutes of the injection.

Procedure

All data were collected retrospectively from patient files and subjected to detailed analysis. Review of patient records revealed that all patients received standard monitoring and were administered intravenous midazolam (0.05 mg/kg) for premedication. In the GA group, patients received 1 mcg/kg fentanyl, 7 mg/kg thiopental, and 0.6 mg/kg rocuronium intravenously. Following endotracheal intubation, ventilation was adjusted to a tidal volume of 7 ml/kg, and anesthesia was maintained with sevoflurane at approximately 1 MAC using a Dräger Primus anesthesia machine, titrated according to the patient's cardiac response. In the EA group, records indicated that the epidural space was accessed at the L3–L4 level using a 16G Tuohy needle. A mixture of 200 mg 2% prilocaine, 25 mg 0.25% bupivacaine, and 25 mcg fentanyl was administered. If regression of the sensory block from the T6 level occurred or the VAS score exceeded 3 during surgery, an additional 5–20 ml of 0.25% bupivacaine was given via the epidural catheter.

Data recorded during and after surgery for two groups of patients were analyzed from medical records. The systolic, diastolic, and mean arterial pressures, heart rate (HR), and SpO_2 values of both groups at the baseline, as well as at the 5^{th}, 10^{th}, 15^{th}, 20^{th}, 40^{th}, 60^{th}, and 75^{th} minutes, were extracted from the patient records. Hypotension was defined as a 20% decrease in blood pressure or systolic arterial pressure ≤90 mmHg. It was observed that in cases of hypotension, rapid fluid replacement was administered, and, when necessary, 5 mg/ml ephedrine was used. In the event of bradycardia, which was defined as a HR of <45 beats per minute, 0.5 mg of atropine was administered.

Intraoperative adverse events and complications were recorded from patient files. Preoperative and first postoperative hour hemogram results were analyzed. Visual Analog Scale (VAS) scores at 1, 4, 12, and 24 hours postoperatively were retrospectively evaluated. Surgical access was categorized as either subcostal or intercostal.

This study was derived from data from the author's doctoral thesis titled "Comparison of GA and EA in PCNL surgery" completed 12 years ago. The analyses in the thesis were conducted in accordance with the statistical approaches of the time. However, in this article, the same data set was re-analyzed using current statistical methods, and the findings were re-evaluated for scientific accuracy and validity. This restructured the study in accordance with current scientific standards.

Statistical Analysis

The data analyses were performed using the Statistical Package for the Social Sciences (SPSS) 18 package program. Descriptive statistics are expressed as the mean and standard deviation for normally distributed continuous variables, and as numbers and percentages for categorical variables. Continuous variables were analyzed using the "Independent samples test" between groups, while "Chi-square analysis" was used to compare categorical variables. For intraoperative physiological parameters, including HR, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and oxygen saturation (SpO_2), between-group comparisons were conducted at multiple predefined time points. To control for the increased risk of type I error associated with multiple comparisons, Bonferroni correction was applied. Accordingly, the adjusted level of statistical significance was set at $p<0.0056$ (0.05 divided by nine time points). Statistical significance was otherwise accepted at $p<0.05$.

Sample Size

With an effect size of 0.72, a total sample of 50 patients (23 EA, 27 GA), and a significance level of 0.05, the power of our study was calculated as 80.42% using G*Power 3.1.7 (University of Kiel, Kiel, Germany), indicating adequate statistical power to detect significant group differences.

RESULTS

Age, gender, height, weight, body-mass index (BMI), ASA scores, and stone characteristics (location and size) were comparable between the EA and GA groups, with no statistically significant differences observed ($p>0.05$) (Table 1).

Anesthesia duration, intraoperative hemorrhage, side effects, and surgeon comfort were similar between the groups, with no statistically significant differences (p>0.05). However, fluid requirement was significantly higher in the EA group (p<0.01) (Table 2).

Table 1. Comparison of study groups in terms of demographic data, ASA Scores, and stone characteristics

	EA group (n=23)	GA group (n=27)	p-value
Age (years) mean±SD	45.91±14.04	43.44±11.73	0.138#
Height (cm) mean±SD	170.78±8.33	171.44±10.95	0.383#
Weight (kg) mean±SD	76.35±12.69	77.30±12.19	0.544#
BMI (kg/cm²) mean±SD	26.43±5.77	26.37±4.26	0.470#
ASA (n/%)			
1	15 (65.2%)	18 (66.7%)	0.277*
2	6 (26.1%)	9 (33.3%)	
3	2 (8.7%)	0 (0%)	
Placement of stones (n/%)			
Medium	1 (4.3%)	1 (3.7%)	
Inferior	7 (30.4%)	8 (29.6%)	0.622*
Pelvis	5 (21.7%)	11 (40.7%)	
Staghorn	5 (21.7%)	4 (14.8%)	
Inferior+pelvis	5 (21.7%)	3 (11.1%)	
Stone size mean±SD	2.51±1.31	2.56±0.85	0.687#

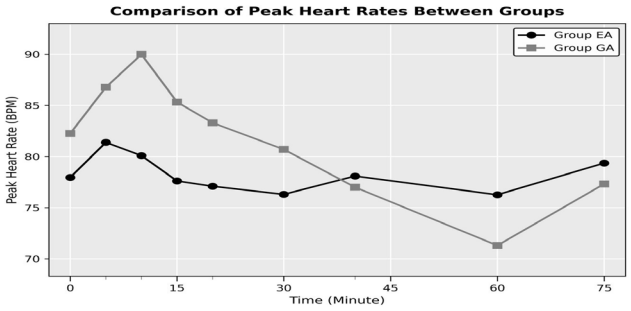
Data presented as mean (±SD) or number (n/%) of patients. ASA: American Society of Anesthesiologists, BMI: Body-mass ındex, EA: Epidural anesthesia, GA: General anesthesia, SD: Standard deviation. The p-value refers to the difference between the groups. p<0.05 is statistically significant. *Chi-square test, #Independent Sample-t test.

Table 2. Comparison of anesthesia duration, amount of fluid administered, postoperative hemorrhage, side effects, and surgeon comfort between groups

	EA group (n=23)	GA group (n=27)	p-value
Anesthesia duration (minute) mean±SD	77.83±26.23	78.15±17.10	0.621#
Amount of fluid administered (ml) mean±SD	1913.04±417.02	1659.26±510.09	<0.01#
Intraoperative hemorrhage (n/%)			
No	21 (91.3%)	22 (81.5%)	0.556*
Yes	2 (8.7%)	5 (18.5%)	
Side effects (n/%)			
None	16 (69.6%)	19 (70.4%)	
Nausea	1 (4.3%)	0 (0%)	0.335*
Hypotension	3 (13%)	7 (25.9%)	
Bradycardia	1 (4.3%)	1 (3.7%)	
Nausea and vomiting	2 (8.7%)	0 (0%)	
Surgeon comfort (n/%)			
Poor	1 (4.3%)	0 (0%)	
Moderate	2 (8.7%)	0 (0%)	0.154*
Good	20 (87%)	27 (100%)	

Data presented as mean (±SD) or number (n/%) of patients. EA: Epidural anesthesia, GA: General anesthesia, SD: Standard deviation. The p-value refers to the difference between the groups. p<0.05 is statistically significant. *Chi-square test, #Independent Sample-t test.

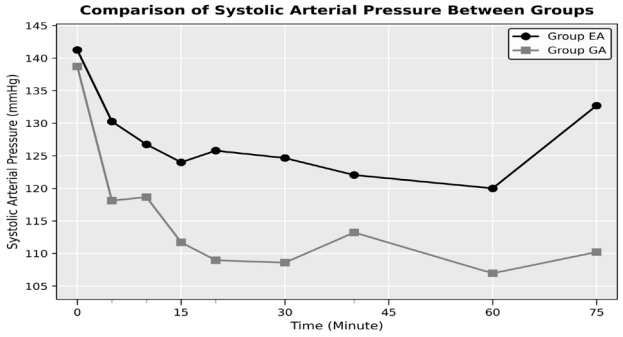
In the comparison of HR between the EA and GA groups, HR was nominally higher in the GA group at the 10th minute (p<0.05), while no significant differences were observed at the remaining time points, with comparable values between groups from the 30th minute onward (Figure 1). However, after Bonferroni correction for multiple comparisons (adjusted α=0.0056), the 10th-minute difference did not remain statistically significant.



Time (Minute)	Group EA	Group GA	P-value
0 Minute	77.96 ± 10.37	82.26 ± 13.09	0.201
5 Minute	81.39 ± 12.10	86.78 ± 14.08	0.152
10 Minute	80.09 ± 12.31	90.00 ± 15.78	0.016
15 Minute	77.61 ± 9.79	85.33 ± 18.19	0.064
20 Minute	77.09 ± 10.74	83.30 ± 17.43	0.131
30 Minute	76.30 ± 11.24	80.70 ± 17.05	0.281
40 Minute	78.09 ± 11.89	77.00 ± 15.08	0.776
60 Minute	76.26 ± 19.66	71.30 ± 18.64	0.367
75 Minute	79.36 ± 15.23	77.32 ± 11.52	0.601

Figure 1. Comparison of peak heart rates between groups
EA: Epidural anesthesia, GA: General anesthesia

Compared to the EA group, the GA group showed a significantly greater decline in SBP at the 5th, 15th, 20th, and 30th minutes (p<0.05). While differences diminished between the 40th and 60th minutes (p>0.05), at the 75th minute, the SBP in the EA group increased again, while the GA group remained at a lower level. At this point, the difference between the groups became highly significant (p<0.001) (Figure 2). However, after Bonferroni correction for multiple comparisons (adjusted α=0.0056), significant between-group differences persisted at the 20th, 30th, and 75th minutes, whereas the differences at the 5th and 15th minutes did not remain statistically significant. Notably, at the 75th minute, SBP increased in the EA group while remaining lower in the GA group, resulting in a highly significant difference (p<0.001).

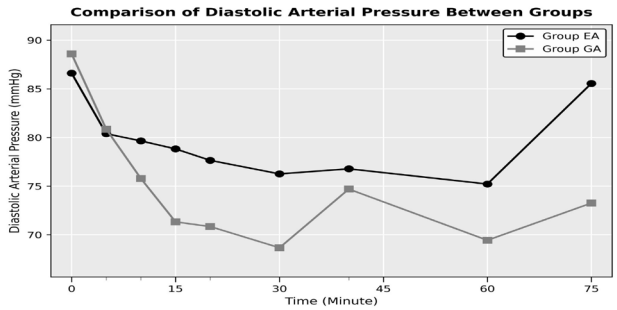


Time (Minute)	Group EA	Group GA	P-value
0 Minute	141.25 ± 20.23	138.7 ± 16.95	0.711
5 Minute	130.26 ± 15.94	118.11 ± 21.37	0.028
10 Minute	126.74 ± 16.54	118.65 ± 21.53	0.162
15 Minute	124.0 ± 18.84	111.70 ± 18.79	0.026
20 Minute	125.78 ± 19.10	108.96 ± 19.30	<0.01
30 Minute	124.65 ± 16.94	108.59 ± 17.38	<0.01
40 Minute	122.04 ± 15.94	113.22 ± 15.26	0.061
60 Minute	120.0 ± 21.35	106.95 ± 21.17	0.091
75 Minute	132.7 ± 15.27	110.21 ± 12.34	<0.001

Figure 2. Comparison of systolic arterial pressure between groups
EA: Epidural anesthesia, GA: General anesthesia

A statistically significant difference was found between EA and GA groups in terms of DBP at the 10th, 15th, 20th, 30th, and 75th minutes (p<0.05). Notably, at the 75th minute, the difference was highly significant (p<0.001). While DBP values remained more stable in the EA group during these periods,

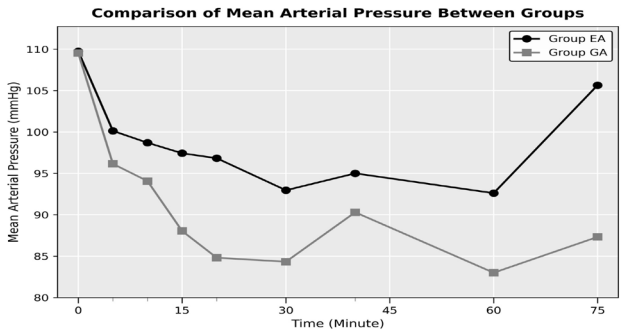
more pronounced decreases were observed in the GA group. However, no significant difference was detected between the groups at the 0th, 5th, 40th, and 60th minutes (p>0.05) (Figure 3). After Bonferroni correction for multiple comparisons across nine time points (adjusted α=0.0056), only the difference at the 75th minute remained statistically significant (p=0.001), whereas the differences observed at earlier time points did not retain statistical significance.



Time (Minute)	Group EA (Mean ± SD)	Group GA (Mean ± SD)	P-value
0 Minute	86.61 ± 14.17	88.59 ± 12.05	0.819
5 Minute	80.39 ± 13.13	80.85 ± 17.97	0.298
10 Minute	79.65 ± 10.48	75.78 ± 16.72	0.026
15 Minute	78.83 ± 13.92	71.33 ± 13.57	0.01
20 Minute	77.65 ± 14.12	70.85 ± 14.7	0.01
30 Minute	76.26 ± 14.89	68.67 ± 13.34	0.01
40 Minute	76.78 ± 12.93	74.7 ± 13.27	0.128
60 Minute	75.22 ± 21.9	69.44 ± 18.58	0.067
75 Minute	85.55 ± 12.54	73.26 ± 11.27	0.001

Figure 3. Comparison of diastolic arterial pressure between groups
EA: Epidural anesthesia, GA: General anesthesia

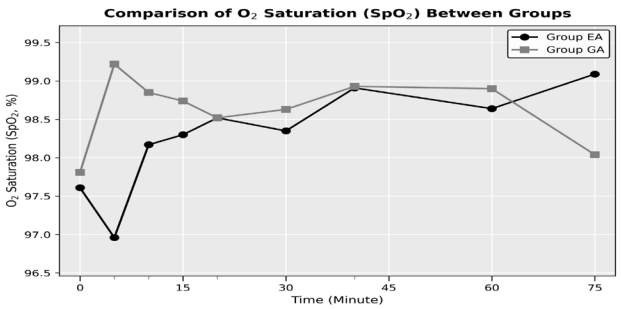
MAP showed an early decrease in both groups at the 5th minute; however, this reduction was more pronounced in the GA group, resulting in a significant difference between the groups at this time point (p<0.05). MAP was also significantly lower in the GA group at the 10th, 15th, 20th, 30th, 40th, 60th, and 75th minutes (p<0.05), with the most marked differences observed at the 30th and 75th minutes (p<0.001). In contrast, MAP values in the EA group demonstrated a more stable pattern throughout the intraoperative period. No significant difference was observed between the groups at baseline (0th minute) (p>0.05) (Figure 4). After Bonferroni correction for multiple comparisons across nine time points (adjusted α=0.0056), only the differences at the 15th, 20th, and 75th minutes remained statistically significant (all p<0.001), whereas the remaining time-point differences did not retain statistical significance.



Time (Minute)	Group EA	Group GA	P-value
0 Minute	109.74 ± 15.19	109.52 ± 14.60	0.344
5 Minute	100.13 ± 14.26	96.15 ± 19.53	0.047
10 Minute	98.70 ± 13.30	94.07 ± 20.65	0.030
15 Minute	97.43 ± 15.93	88.04 ± 15.35	<0.01
20 Minute	96.83 ± 17.26	84.81 ± 17.33	0.001
30 Minute	92.96 ± 19.00	84.33 ± 14.35	0.011
40 Minute	95.00 ± 14.35	90.30 ± 13.74	0.056
60 Minute	92.61 ± 25.66	83.00 ± 21.53	0.024
75 Minute	105.64 ± 13.11	87.32 ± 12.05	<0.001

Figure 4. Comparison of mean arterial pressure between groups
EA: Epidural anesthesia, GA: General anesthesia

When SpO_2 values were compared at different time points between the EA and GA groups, no statistically significant differences were observed at 0, 10, 15, 20, 30, 40, 60, and 75 minutes (p>0.05). However, at the 5th minute, the SpO_2 value in the GA group (99.22±1.05) was significantly higher than that in the EA group (96.96±3.82), and this difference was statistically significant (p<0.01) (Figure 5). After Bonferroni correction for multiple comparisons across nine time points (adjusted α=0.0056), this difference remained statistically significant (p=0.001), while no significant differences were observed at the remaining time points.



Time (Minute)	Group EA	Group GA	P-value
0 Minute	97.61 ± 1.92	97.81 ± 1.77	0.650
5 Minute	96.96 ± 3.82	99.22 ± 1.05	<0.01
10 Minute	98.17 ± 1.55	98.85 ± 1.06	0.191
15 Minute	98.30 ± 2.47	98.74 ± 1.16	0.590
20 Minute	98.52 ± 1.78	98.52 ± 1.12	0.583
30 Minute	98.35 ± 1.74	98.63 ± 1.07	0.839
40 Minute	98.91 ± 1.47	98.93 ± 1.03	0.565
60 Minute	98.64 ± 1.62	98.90 ± 1.05	0.672
75 Minute	99.09 ± 1.81	98.04 ± 1.06	0.261

Figure 5. Comparison of O_2 saturation (SpO_2) between groups
EA: Epidural anesthesia, GA: General anesthesia

Postoperative analgesic requirement was significantly lower in the EA group compared to the GA group (p<0.01), with fewer patients needing single or multiple doses. VAS scores were also significantly lower in the EA group at all measured time points (p<0.01). No significant differences were found between the EA and GA groups in terms of hematocrit values, nephrostomy access site, or atropine use (p>0.05). However, ephedrine was required significantly more often and in higher total doses in the EA group, while no patients in the GA group received ephedrine (p<0.05) (Table 3).

DISCUSSION

Due to the specific risks associated with GA, regional anesthesia techniques are increasingly preferred in PCNL to reduce anesthesia-related morbidity and postoperative complications, without compromising procedural efficacy.[6,7,11,12] Spinal anesthesia is considered safe and feasible, particularly in elderly patients with cardiopulmonary comorbidities;[13] however, its limitations in prolonged procedures and the challenges of conversion to GA in the prone position present practical concerns. EA, via catheter placement, offers the advantage of extending anesthesia intraoperatively and is a safe and effective alternative, especially in patients at high risk for difficult intubation or GA-related complications.[6,14] This study aimed to compare surgical, hemodynamic, and postoperative analgesia-related outcomes in patients undergoing PCNL under GA or EA.

In our study, the EA and GA groups were homogeneous in terms of demographic characteristics and anesthesia

Table 3. Comparison of study groups in terms of hematocrit levels, postoperative VAS scores, analgesic use, atropine and ephedrine administration, and PCNL catheter access site

	EA group (n=23)	GA group (n=27)	p-value
Preoperative HCT (mean±SD)	39.56±5.91	41.09±4.84	0.327#
Postoperative HCT (mean±SD)	35.51±5.66	36.43±4.78	0.542#
Change in HCT (ΔHCT) (mean±SD)	4.04±3.06	4.66±2.73	0.457#
Postoperative VAS (mean±SD)			
1st hour	0.26±0.68	3.19±1.90	<0.001#
4th hour	0.83±1.30	3.19±1.77	<0.001#
12th hour	0.57±1.08	3.07±1.83	<0.001#
24th hour	0.61±0.94	3.00±1.88	<0.001#
Use of post-op analgesia, n (%)			
None	15 (65.2%)	1 (3.7%)	<0.001*
1 dose	6 (26.1%)	6 (22.2%)	
2 doses	2 (8.7%)	11 (40.7%)	
3 doses	0 (0%)	6 (22.2%)	
4 doses	0 (0%)	2 (7.4%)	
6 doses	0 (0%)	1 (3.7%)	
Atropine use			
No, n (%)	19 (82.6%)	26 (96.3%)	0.256*
Yes, n (%)	4 (17.4%)	1 (3.7%)	
Amount (mg), mean±SD	0.15±0.35	0.01±0.09	0.068#
Ephedrine use			
No, n (%)	18 (78.3%)	27 (100%)	0.037*
Yes, n (%)	5 (21.7%)	0 (0%)	
Amount (mg), mean±SD	2.61±5.61	0.00±0.00	0.030#
PCNL catheter access site, n (%)			
Subcostal	21 (91.3%)	27 (100%)	0.401*
Intercostal	2 (8.7%)	0 (0%)	

Data presented as mean (±SD) or number (n/%) of patients. EA: Epidural anesthesia, GA: General anesthesia, HCT: Hematocrit, PCNL: Percutaneous nephrolithotomy, SD: Standard deviation, VAS: Visual Analog Scale. The p-value refers to the difference between the groups. p<0.05 is statistically significant. *Chi-square test, #Independent Sample-t test.

risk, ensuring comparability. Consistent with previous studies,[6,9,15–17] no significant differences were observed regarding anesthesia duration, postoperative hemorrhage, side effects, or surgeon comfort, suggesting that both techniques provide similar clinical outcomes and effectively support the surgical procedure.

EA has been associated with earlier mobilization and faster return to oral intake, though its effect on hospital stay varies across studies.[9,18] While some studies have reported significantly shorter hospital stays with regional anesthesia,[6,18,19] our study showed a shorter stay in the EA group; however, statistical analysis showed that this difference did not reach significance.

A study comparing hemodynamic responses during PCNL under GA and subarachnoid block (SAB) has shown that, although baseline values are similar, patients under GA exhibit significantly higher HR, SBP, DBP, and MBP from the early intraoperative period onward, indicating more pronounced and sustained cardiovascular responses compared to SAB.[16] Some studies have similarly reported that HR is significantly higher in the GA group, with these differences being particularly evident in the early stages.[20,21] Our study indicates that GA leads to an initial rise in HR over the first 30 minutes, which diminishes over time. This early rise may result from decreased systemic vascular resistance and reduced right atrial filling due to sympathetic blockade caused by neuraxial anesthesia. In contrast, EA is known to suppress sympathetic tone, leading to neurogenic bradycardia, contributing to initial bradycardia. However, fluid resuscitation and ephedrine administration likely restored sympathetic activity in the EA group, leading to HR levels comparable to those in the GA group.

Our study reveals significant differences in hemodynamic stability between GA and EA groups. While SBP, DBP, and MBP remained more stable in the EA group, the GA group exhibited marked decreases, particularly during the early intraoperative period. These fluctuations in the GA group are likely due to the vasodilatory effects of general anesthetics, whereas EA provides a more controlled sympathetic blockade, reducing blood pressure variability. The pronounced differences observed, especially at the 75th minute, underscore the sustained stabilizing effect of EA. These findings suggest that EA may offer superior hemodynamic control and be particularly advantageous in patients at risk for hypotensive episodes.

A review of current literature shows no evidence regarding the impact of the anesthesia method on SpO_2 levels in patients undergoing PCNL. The findings of our study indicate that while SpO_2 levels were generally comparable between patients receiving EA and GA, higher SpO_2 values were observed in the GA group during the early postoperative period (5th minute). Overall, SpO_2 remained stable in both groups, no clinically significant hypoxemia was detected, and both anesthesia techniques were deemed safe in maintaining adequate SpO_2.

While a study reports similar perioperative hemoglobin levels and no transfusion requirements between EA and GA groups,[15] another study has shown greater hemoglobin decline and blood loss in the GA group compared to SAB group.[16] In our study, both preoperative and postoperative HCT levels and ΔHCT were assessed, showing no statistically significant variation between groups. Although intraoperative bleeding was higher in the GA group, this difference was not statistically significant, and no patient required a blood transfusion. These findings indicate the hematological safety of both techniques during the perioperative period.

Multiple studies have demonstrated the efficacy of EA in reducing postoperative pain and analgesic requirements.[9,14,15,17,22] A study reported significantly lower pain scores in the EA group at the 1st and 3rd postoperative hours. However, no significant difference in pain scores was observed between the groups after the 12th postoperative hour.[15] Singh et al.[9] found significantly lower 24-hour analgesic use in the EA group in a randomized controlled trial of PCNL patients, and a meta-analysis by Liu et al.[22] confirmed reduced pain and analgesic needs with regional anesthesia. Consistent with these findings, our study showed significantly lower VAS scores at all time points. Additionally,

it was observed that the majority of patients in the EA group did not require postoperative analgesics, whereas the need for multiple doses of analgesia was significantly higher in the GA group. The present findings imply that EA is more effective in postoperative pain management, leading to reduced pain perception and a decreased requirement for additional analgesic medication.

Studies have shown that hypotension during SA or CSEA in PCNL is generally manageable with fluid therapy, though vasopressor support may be required, and significant MBP reductions have been observed compared to GA.[16,17,23] In line with the literature, our study found significantly higher fluid requirements and ephedrine use in the EA group, while no ephedrine was needed in the GA group. These results indicate that EA may cause greater susceptibility to hypotension due to sympathetic blockade, whereas GA appears to maintain more stable vascular tone.

The study's constraints include conducting it at a single center, a comparatively small cohort, and no prolonged follow-up period. Furthermore, the awareness of the surgical and anesthesia teams regarding which patients received which anesthesia could introduce observational bias. Although the data of the present study were collected between 2012 and 2014, both EA and GA remain commonly used and well-established options in current PCNL practice. Recent literature indicates that the comparative evaluation of these techniques continues to be relevant, as anesthesia choice may still affect perioperative hemodynamics, postoperative pain, complication profiles, and overall recovery. While advances in perioperative monitoring and management have occurred over time, they have not fundamentally changed the physiological mechanisms underlying EA and GA.[15,17,22] Accordingly, the patterns observed in our study-such as differences in postoperative analgesia, intraoperative hemodynamic parameters, and perioperative medication requirements-are generally in line with contemporary findings reported in the literature. Therefore, despite the historical nature of the data, the results of this study may still contribute to the ongoing discussion regarding anesthetic selection in PCNL procedures.

However, the similarity in demographic and clinical characteristics between the groups enhances the reliability of the findings. The detailed examination of hemodynamic parameters at various time points allowed for a comprehensive assessment of the cardiovascular effects of the anesthesia techniques. Additionally, the superiority of EA in postoperative pain management was clearly demonstrated.

Limitations

This study has several limitations that should be acknowledged. First, the retrospective, single-center design and relatively small sample size (n=50) carry an inherent risk of selection bias and may limit the generalizability of the findings. Second, although the fundamental physiological mechanisms[15,17,22] of the anesthetic techniques remain unchanged, the data were collected between 2012 and 2014; therefore, recent advances in enhanced recovery protocols may limit direct comparisons with contemporary perioperative practice. Finally, the study evaluated immediate postoperative outcomes and lacked a long-term follow-up to assess delayed complications or extended quality of recovery. Future prospective, randomized, multicenter trials with larger cohorts are warranted to confirm and extend these findings.

CONCLUSION

EA appears to be a safe and effective alternative to GA for PCNL, offering comparable surgical outcomes and greater hemodynamic stability. Its advantages include superior postoperative pain control and reduced analgesic requirements, along with sufficient anesthetic duration for managing large or complex stones. EA is particularly beneficial in patients for whom GA is contraindicated or endotracheal intubation is challenging. Although EA offers several advantages, including improved postoperative analgesia and hemodynamic stability, it is also associated with increased intraoperative fluid and vasopressor requirements. Therefore, careful patient selection and vigilant intraoperative hemodynamic monitoring are essential when EA is chosen, particularly in patients with limited cardiovascular reserve. While bleeding and hypotension rates were similar between groups, close monitoring of hemodynamic parameters remains essential. Overall, EA represents a favorable anesthetic option for selected patients, and these findings should be confirmed in larger, multicenter, and long-term studies.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study has been approved by the Clinical Researches Ethics Committee of Haseki Training and Research Hospital (Date: 05.02.2014, Decision No: 55).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: ST, EÖ; Design: ST, EÖ; Data Collection and/or Processing: ST, ABK; Analysis or Interpretation of Data: ST; Literature Research: ST, ABK; Writing Manuscript: ST; Critical Revision for Important Intellectual Content: ST, EÖ.

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MHA MEDIHEALTH ACADEMY

Eurasian Journal of
Anesthesiology & Intensive Care

Original Article

doi
10.51271/EAJAIC-0052

Effects of controlled hypotension on regional cerebral oxygen saturation and cognitive function in hypertensive patients undergoing tympanoplasty

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Cite this article: Akel CY, Geçer S, Sarıbudak HA. Effects of controlled hypotension on regional cerebral oxygen saturation and cognitive function in hypertensive patients undergoing tympanoplasty. *Eur J Anesthesiol Intens Care.* 2026;3(2):41-48. doi:10.51271/EAJAIC-0052

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Received: 27/02/2026 ◆ **Accepted**: 13/05/2026 ◆ **Published**: 16/05/2026

ABSTRACT

Aims: Controlled hypotension (CH) may impair tissue perfusion and inhibit autonomic regulation, potentially increasing the risk of cerebral ischemia in patients with hypertension due to altered autoregulation. This study aimed to compare the effects of CH on regional cerebral oxygen saturation (rSO_2) and cognitive function in hypertensive and normotensive patients undergoing tympanoplasty.

Methods: Sixty patients (ASA physical status I-II, aged 18-63 years) scheduled for elective tympanoplasty between January 2018 and December 2018, after obtaining approval from the Clinical Research Ethics Committee were enrolled in this prospective study (Date: 09.01.2018, Decision No: 2017/0410). Patients were allocated into two groups: normotensive (Group N, n=30) and hypertensive (Group H, n=30). Demographic characteristics, hemoglobin and hematocrit levels, anesthesia and surgical durations, time to extubation, total remifentanil consumption, additional analgesic requirements, and hemodynamic variables were recorded. Bilateral rSO_2 values (near-infrared spectroscopy), bispectral index (BIS), end-tidal carbon dioxide ($EtCO_2$), body temperature, and inspired/expired sevoflurane concentrations were recorded at 5-minute intervals intraoperatively. Cognitive function was assessed using the Mini-Mental State Examination (MMSE) 24 hours preoperatively and at postoperative 6 and 24 hours.

Results: After achieving target blood pressure, mean arterial pressure (MAP) values were comparable between groups. Although hypertensive patients demonstrated greater reductions in blood pressure from baseline, rSO_2 values did not differ significantly between groups. No patient developed postoperative cognitive dysfunction (POCD) based on MMSE scores at 6 or 24 hours. The incidence of adverse events was similar between groups.

Conclusion: Within a defined MAP range, CH did not result in decreased rSO_2 or POCD in hypertensive patients. These findings suggest that CH may be safely applied in this patient population.

Keywords: Near-infrared spectroscopy, controlled hypotension, cognitive dysfunction

INTRODUCTION

Hypotensive anesthesia involves the deliberate induction of controlled hypotension (CH) tailored to the patient's age, baseline blood pressure, and medical history. CH is defined as the intentional and reversible reduction of arterial blood pressure below baseline values while maintaining adequate cerebral, coronary, and renal perfusion.[1] It is commonly achieved by maintaining systolic blood pressure (SBP) at 80-90 mmHg and mean arterial pressure (MAP) at 50-65 mmHg.

Lowering arterial pressure reduces intraoperative bleeding and postoperative edema, improves surgical field visibility, and facilitates operative precision. Reduced bleeding also minimizes the need for traumatic hemostatic interventions and may limit tissue injury. CH is particularly advantageous in surgeries requiring a clear operative field, including middle ear surgery, endoscopic sinus surgery, plastic and reconstructive microsurgery, ophthalmic surgery, and neurosurgery. Tympanoplasty is one of the middle ear

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procedures in which hypotensive anesthesia is frequently applied to reduce bleeding.

Despite its benefits, CH may compromise vital organ perfusion and contribute to tissue ischemia or autonomic dysfunction. In hypertensive patients, vascular structural changes and impaired autoregulation may increase susceptibility to cerebral hypoperfusion. In normotensive individuals, cerebral blood flow is generally preserved during moderate hypotension through autoregulatory mechanisms; however, this compensation may be impaired in patients with chronic hypertension, potentially increasing the risk of cerebral ischemia.[2] Therefore, careful intraoperative monitoring of end-organ perfusion is warranted.

Near-infrared spectroscopy (NIRS) is a noninvasive monitoring technique that provides continuous assessment of regional cerebral oxygen saturation (rSO_2), representing the ratio of oxyhemoglobin to total hemoglobin, predominantly in the venous compartment. Although not routinely used in all procedures, NIRS has gained broader clinical application over the past two decades.[3] Its utility in detecting cerebral desaturation has been demonstrated in cardiac surgery, hypotensive anesthesia, and prolonged Trendelenburg positioning.[4,5]

Postoperative cognitive dysfunction (POCD) is defined as a decline in cognitive performance following surgery and anesthesia that may persist for weeks or months.[6] Clinical manifestations include memory impairment, psychomotor slowing, executive dysfunction, and mood disturbances. POCD is diagnosed using standardized neuropsychological testing and is considered a postoperative complication.[7] Prolonged or profound intraoperative hypotension is among the identified risk factors.

This study aimed to compare the effects of CH on rSO_2 and cognitive function in hypertensive and normotensive patients undergoing tympanoplasty, using continuous NIRS monitoring and perioperative Mini-Mental State Examination (MMSE) assessment. We hypothesized that CH would not result in a significant decrease in rSO_2 or deterioration in cognitive function in hypertensive patients compared with normotensive patients.

METHODS

This prospective study was approved by the Clinical Researches Ethics Committee of İstanbul Medeniyet University Göztepe Training and Research Hospital (Date: 09.01.2018, Decision No: 2017/0410). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. The study was conducted between January 2018 and December 2018. Sixty patients aged 18-63 years with American Society of Anesthesiologists (ASA) physical status I-II scheduled for elective tympanoplasty were enrolled. Exclusion criteria included ASA physical status ≥III, age <18 or >65 years, known allergy to study medications, conditions affecting preoperative cognitive function, and a preoperative MMSE score ≤23. All patients underwent preanesthetic evaluation and provided written informed consent. Patients were allocated into two groups according to baseline blood pressure: normotensive (Group N) and controlled hypertensive (Group H). This was a non-randomized observational comparison, and group allocation was based on baseline blood pressure status. Standard monitoring included three-lead electrocardiography, heart rate, noninvasive systolic, diastolic, and MAP, peripheral oxygen saturation (SpO_2), BIS). Bilateral NIRS sensors were applied to the frontal region to continuously measure rSO_2. Intravenous access was established with a 20-gauge cannula, and isotonic crystalloid infusion was initiated. Premedication consisted of midazolam 1 mg intravenous (IV) and fentanyl 1 µg/kg IV. After preoxygenation with 100% oxygen, anesthesia was induced with propofol 2 mg/kg IV and rocuronium 0.5 mg/kg IV. Following adequate neuromuscular blockade, endotracheal intubation was performed, and mechanical ventilation was initiated (tidal volume 7 mL/kg; respiratory rate 12 breaths/min). Anesthesia was maintained with 50% oxygen-air mixture and sevoflurane (target minimal alveolar concentration (MAC) 1-2), combined with remifentanil infusion (0.05 µg/kg/min in normotensive patients and 0.1 µg/kg/min in hypertensive patients), titrated to achieve target MAP. If CH could not be achieved at remifentanil doses up to 0.2 µg/kg/min, esmolol infusion (50-300 µg/kg/min) was planned as rescue therapy. Neuromuscular blockade was reversed at the end of surgery with neostigmine 0.03 mg/kg IV and atropine 0.015 mg/kg IV, and patients were extubated upon meeting standard criteria. Total remifentanil consumption was recorded. Demographic data, ASA classification, body weight, hemoglobin and hematocrit levels, baseline vital signs, rSO_2, and BIS values were documented. Hemodynamic parameters, rSO_2, BIS, $EtCO_2$, temperature, and inspired and expired sevoflurane fractions (FiSevo) were recorded after premedication, post-induction, post-intubation, and at 5-minute intervals intraoperatively. Anesthesia duration, surgical duration, time to discontinuation of anesthetic gases, time to extubation, total remifentanil consumption, and additional analgesic requirements were recorded. Cognitive function was assessed using the MMSE at 24 hours preoperatively and at postoperative 6 and 24 hours. Intraoperatively, MAP was maintained between 65-70 mmHg using sevoflurane and remifentanil infusion. If MAP decreased below 65 mmHg, hypotensive agents were reduced. Anesthesia depth was titrated to maintain BIS values between 40 and 60. Bradycardia was defined as heart rate <40 beats/min and treated with atropine 0.015 mg/kg IV if required. Ten minutes before the end of surgery, tramadol 1 mg/kg IV was administered for analgesia and metoclopramide 0.1 mg/kg IV for prophylaxis of nausea and vomiting. Patients with a visual analog scale (VAS) score >4 received paracetamol 10 mg/kg IV as rescue analgesia. Postoperative nausea and vomiting (PONV) were monitored; ondansetron 0.1 mg/kg IV was planned as rescue antiemetic therapy.

The primary outcome of the study was the comparison of intraoperative rSO_2 values between hypertensive and normotensive patients.

Secondary outcomes included postoperative cognitive function assessed by MMSE, intraoperative hemodynamic variables, anesthetic and analgesic requirements, and the incidence of perioperative adverse events.

Descriptive statistics were expressed as mean±standard deviation or median and interquartile range (IQR: Q1-Q3), as appropriate. Normality was assessed using histograms and the Kolmogorov-Smirnov test. Parametric variables were compared using the independent samples t-test, and nonparametric variables were analyzed using the Mann-Whitney U test. Categorical variables were compared using the chi-square test or Fisher's exact test. A p value <0.05 was considered statistically significant. All analyses were performed using IBM SPSS Statistics version 15.

RESULTS

Between January 2018 and December 2018, a total of 60 patients completed the study, including 30 normotensive and 30 hypertensive patients. Comparison of demographic characteristics and laboratory parameters between groups demonstrated that patients in the hypertensive group were significantly older and had higher body weight compared with the normotensive group (p<0.05). In addition, anesthesia duration, surgical duration, time to discontinuation of anesthetic gases, and total remifentanil consumption were significantly greater in the hypertensive group (p<0.05).

No significant differences were observed between groups with respect to other demographic or clinical variables (p>0.05) (Table 1).

Table 1. Comparison of demographic characteristics and laboratory parameters between groups

		Groups		
		Group-N	Group-H	p
		Mean±SD	Mean±SD	
Age		31±9.4	40±13.8	0.006*
Weight		68±12.7	77±15.4	0.011*
ASA, n (%)	ASA 1	28 (93.3%)	23 (76.7%)	0.145
	ASA 2	2 (6.7%)	7 (23.3%)	
Hemoglobin		13.6±2.2	14.3±1.6	0.215
Hematocrit		40.6±5.5	43.0±4.5	0.067
Duration of anesthesia		102±31.1	126±29.9	0.004*
Duration of surgery		90±30.8	113±30.8	0.004*
Time to discontinuation of anesthetic gases		99±30.9	122±30.1	0.004*
Time to extubation		6±2.5	5±1.9	0.100
Total remifentanil consumption		1002±579.3	151±838.9	0.008*
Additional analgesic requirement		80±16.5	86±16.3	0.151
Preoperative Mini-Mental Test score		28±1.6	28±1.4	0.746
Postoperative Mini-Mental Test score (6^{th} hour)		28±2.0	28±1.8	0.449
Postoperative Mini-Mental Test score (24^{th} hour)		28±1.7	28±1.8	0.413

SD: Standard deviation, ASA: American Society of Anesthesiologists

Comparison of SBP values between groups showed that median SBP at baseline (preinduction), after premedication, after induction, after intubation, after discontinuation of anesthesia, and after extubation was significantly higher in the hypertensive group than in the normotensive group (p<0.05).

In addition, median SBP at postoperative 5 and 10 minutes remained significantly higher in hypertensive patients compared with normotensive patients (p<0.05).

Heart rate (HR) values were compared between groups. Mean HR at baseline, after premedication, after induction, after intubation, after discontinuation of anesthesia, and after extubation was significantly higher in the hypertensive group compared with the normotensive group (p<0.05).

Additionally, mean HR at 5, 10, 120, and 125 minutes after intubation was significantly higher in hypertensive patients (p<0.05). No significant differences were observed between groups at other intraoperative time points (p>0.05) (Tables 2 and 3).

Mean left rSO_2 values were compared between groups. In the hypertensive group, mean left rSO_2 at 50 minutes after intubation was significantly lower than in the normotensive group (p<0.05).

No significant differences were observed between groups at other intraoperative time points (p>0.05) (Table 3, Figure 1).

Mean right rSO_2 values did not differ significantly between the hypertensive and normotensive groups at any measured time point (p>0.05) (Figure 1).

Median BIS values were also comparable between groups throughout the study period, with no statistically significant differences observed (p>0.05) (Figures 1 and 2).

Mean MAC values were comparable between the hypertensive and normotensive groups, with no statistically significant differences observed (p>0.05) (Figure 2).

Similarly, mean inspired FiSevo did not differ significantly between groups at any time point (p>0.05).

Mean expired FeSevo was significantly higher in the hypertensive group at 125 minutes after intubation (p<0.05). No other significant differences were observed between groups for FeSevo values (p>0.05).

Median $EtCO_2$ values were comparable between groups, with no significant differences detected (p>0.05). Mean body temperature values were also similar in both groups (p>0.05).

Postoperative hemodynamic parameters were significantly higher in the hypertensive group. Mean systolic, diastolic, and MAP values at postoperative 5, 10, and 15 minutes were significantly greater in hypertensive patients compared with normotensive patients (p<0.05).

Similarly, mean heart rate at postoperative 5, 10, and 15 minutes was significantly higher in the hypertensive group (p<0.05).

A decrease of ≥20% from baseline in left rSO_2 was analyzed between groups, with no significant difference observed (p>0.05) (Table 4).

Table 2. Comparison of MAP values between groups

	Groups		
	Group-N	Group-H	p
	Median (Q1-Q3)	Median (Q1-Q3)	
MAP baseline	92 (87-95)	108 (103-114)	<0.001*
Premedication	91 (85-95)	108 (103-110)	<0.001*
Induction	76 (71-84)	90 (77-97)	<0.001*
Intubation	103 (93-114)	115 (99-133)	0.017*
5th min	84 (76-90)	96 (88-106)	<0.001*
10th min	73 (68-79)	82 (73-91)	0.008*
15th min	70 (65-77)	75 (68-82)	0.019*
20th min	67 (65-76)	72 (68-79)	0.060
25th min	70 (66-77)	71 (66-78)	0.641
30th min	69 (66-73)	70 (68-75)	0.505
35th min	69 (65-74)	70 (67-74)	0.381
40th min	69 (65-73)	70 (66-75)	0.275
45th min	67 (65-72)	67 (65-70)	0.899
50th min	67 (65-70)	68 (65-71)	0.710
55th min	66 (65-70)	69 (65-72)	0.135
60th min	67 (65-70)	67 (65-69)	0.903
65th min	66 (65-70)	67 (65-68)	0.861
70th min	66 (64-69)	67 (65-70)	0.268
75th min	67 (63-70)	69 (65-72)	0.488
80th min	67 (64-73)	69 (65-73)	0.552
85th min	67 (64-72)	68 (66-72)	0.816
90th min	67 (65-69)	67 (65-71)	0.690
95th min	67 (63-72)	67 (65-71)	0.852
100th min	67 (64-71)	68 (65-73)	0.447
105th min	68 (63-71)	71 (65-75)	0.150
110th min	67 (60-75)	70 (66-75)	0.329
115th min	68 (65-78)	67 (65-76)	0.966
120th min	72 (68-75)	68 (66-73)	0.292
125th min	70 (70-76)	69 (67-76)	0.605
130th min	64 (53-74)	71 (65-77)	0.426
135th min	65 (54-75)	70 (66-78)	0.521
140th min	65 (54-76)	71 (67-82)	0.389
145th min	66 (57-74)	71 (65-89)	0.359
150th min	69 (58-79)	86 (68-97)	0.242
After discontinuation of anesthesia	82 (78-90)	94 (83-103)	0.010*
Extubation	95 (88-109)	105 (98-118)	0.008*

Mann-Whitney U test, *p<0.05. MAP: Mean arterial pressure

Table 3. Comparison of heart rate values between groups

	Groups		
	Group-N	Group-H	p
	Mean±SD	Mean±SD	
HR baseline	76±13	88±15	0.003*
Premedication	82±17	94±17	0.013*
Induction	78±13	87±14	0.009*
Intubation	89±13	100±17	0.009*
5th min	83±12	91±14	0.014*
10th min	78±11	85±15	0.046*
15th min	75±11	80±16	0.166
20th min	72±11	78±16	0.111
25th min	68±10	73±15	0.125
30th min	67±11	68±10	0.730
35th min	65±10	66±10	0.651
40th min	64±10	65±9	0.540
45th min	63±9	64±9	0.659
50th min	63±10	63±9	0.802
55th min	62±10	62±7	0.949
60th min	62±10	61±7	0.627
65th min	62±9	60±7	0.370
70th min	62±9	61±8	0.859
75th min	62±10	61±8	0.811
80th min	62±9	62±7	0.832
85th min	60±7	62±7	0.305
90th min	60±8	64±8	0.225
95th min	60±7	63±6	0.249
100th min	61±5	63±7	0.198
105th min	60±5	64±7	0.068
110th min	61±4	65±7	0.156
115th min	61±6	65±7	0.127
120th min	59±5	67±10	0.009*
125th min	59±5	68±10	0.039*
130th min	62±1	67±9	0.492
135th min	63±1	64±9	0.779
140th min	63±4	68±10	0.451
145th min	62±4	71±12	0.385
150th min	63±1	76±10	0.108
After discontinuation of anesthesia	76±20	89±24	0.027*
Extubation	90±13	102±18	0.005*

Independent samples t-test, p<0.05, SD: Standard deviation, HR: Heart rate

Similarly, the incidence of a ≥20% reduction from baseline in right rSO_2 did not differ significantly between groups (p>0.05).

DISCUSSION

In this study, we compared the effects of CH during elective tympanoplasty on intraoperative regional cerebral oxygenation and early and late postoperative cognitive function in hypertensive and normotensive patients. This topic is clinically important because hypertensive patients may have altered cerebral autoregulation, which could increase their vulnerability to cerebral hypoperfusion during controlled hypotension. Although the reduction in blood pressure from baseline was greater in hypertensive patients, no significant differences were observed in NIRS-derived rSO_2 values between groups. Similarly, MMT scores were comparable, indicating no difference in early postoperative cognitive function.

Because a greater reduction in arterial pressure was required to reach target MAP in hypertensive patients, higher doses of remifentanil were administered in this group. However, no additional hypotensive agent was required. Despite increased remifentanil consumption, no clinically significant bradycardia requiring intervention or delay in extubation was observed.

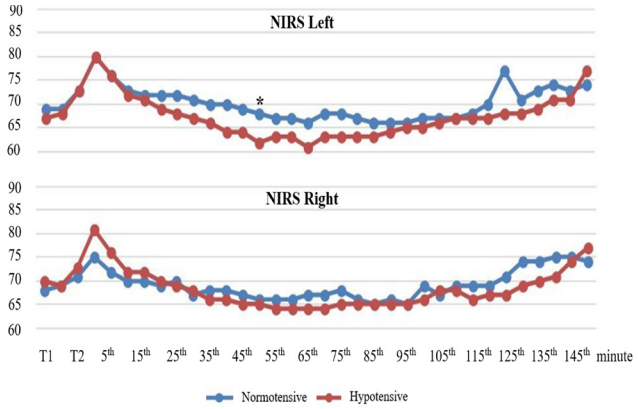

Figure 1. Left and right Near-infrared spectroscopy regional cerebral oxygen saturation values according to study groups *(p<0.05)

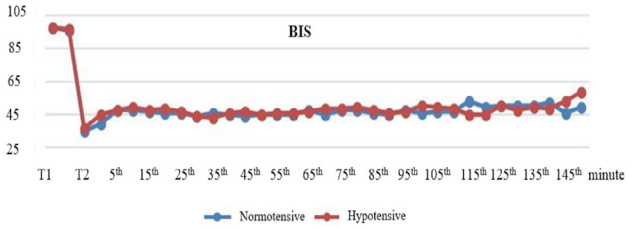

Figure 2. Bispectral index values according to study groups

Table 4. Comparison of postoperative blood pressure and heart rate values between groups

	Groups		
	Group-N	Group-H	p
	Mean±SD	Mean±SD	
MAP 5th min	89±11.2	99±14.0	0.004*
10th min	88±11.3	97±13.3	0.010*
15th min	87±10.3	96±12.3	0.004*
5th min [a]	99	99	0.115
10th min [a]	99	99	0.078
15th min [a]	99	99	0.055
HR 5th min	80±12.0	88±16.6	0.022*
10th min	78±11.5	88±16.2	0.008*
15th min	75±10.7	88±15.3	0.001*

Independent samples t-test, Mann-Whitney U test p<0.05. SD: Standard deviation, MAP: Mean arterial pressure

CH is widely used in middle ear surgery to reduce intraoperative bleeding. Lower arterial pressure improves surgical field visibility, may shorten operative time, and may reduce procedure-related complications.[8] In procedures such as plastic, maxillofacial, and otorhinolaryngologic surgery, where rapid normalization of blood pressure may cause reactive bleeding, moderate hypotension with gradual onset and recovery is preferred.[9]

Nevertheless, concerns remain regarding the potential for vital organ hypoperfusion during deliberate hypotension.[10-12] This risk is particularly relevant in patients with chronic hypertension, in whom altered vascular structure and autoregulatory shifts may predispose to cerebral hypoperfusion.

Hypertensive patients represent a substantial proportion of the surgical population, and hypertension is a well-established predictor of perioperative morbidity and mortality.[13] Even when blood pressure is medically controlled preoperatively, hypertensive patients are more prone to abrupt hemodynamic fluctuations during general anesthesia. Therefore, careful titration and hemodynamic stability are essential in this population. Ongoing investigations continue to evaluate whether maintaining MAP within a defined hypotensive range may predispose hypertensive patients to organ hypoperfusion.[14] Consequently, the application of CH in these patients raises concerns regarding potential adverse effects.

In normotensive individuals, cerebral blood flow is maintained independently of systemic pressure changes until MAP decreases to approximately 60 mmHg. In contrast, chronic hypertension leads to vascular remodeling, reduced arterial compliance, and a rightward shift of the cerebral autoregulatory curve, potentially impairing autoregulation.[15] Therefore, reducing MAP below a critical threshold in hypertensive patients may compromise cerebral perfusion.

CH is commonly defined as maintaining MAP between 50-65 mmHg. In a study by Erdem et al.,[16] MAP was maintained at 50-60 mmHg in 50 ASA I patients. Other studies have targeted MAP ranges of 60-70 mmHg during CH.[17,18] In a study by Nowak et al.,[19] 47 patients were stratified into mild (MAP >75 mmHg), moderate (65<MAP≤75 mmHg), and deep (MAP≤65 mmHg) hypotension groups.

In the present study, MAP was targeted at 65-70 mmHg. The desired MAP was achieved approximately 20 minutes after surgical incision. After reaching target levels, MAP values were comparable between groups. However, during the transition from baseline to steady-state hypotension, hypertensive patients exhibited significantly greater changes in MAP compared with normotensive patients.

Previous studies on controlled hypotensive anesthesia have primarily focused on intraoperative hemodynamic stability, postoperative recovery, and cognitive outcomes. Although several investigations have evaluated potential adverse effects of CH in hypertensive patients, we found no studies specifically assessing cerebral perfusion using NIRS in this population.

Kim et al.[15] investigated early hypertensive cerebrovascular changes in a rat model, examining cerebral morphological alterations and impaired vasodilation. Using magnetic resonance imaging techniques, they compared regional cerebrovascular characteristics between hypertensive and normotensive rats. Although global cerebral blood flow values were similar in both groups, hypertensive rats demonstrated lower regional cerebral arterial blood volume. In response to hypercapnia, hypertensive rats exhibited greater changes in cerebral blood flow, while arterial blood volume changes were comparable between groups.

In the present study, although greater reductions in blood pressure were required in hypertensive patients to achieve the same target MAP, no significant differences were observed in NIRS-derived rSO_2 values between groups. However, these findings should be interpreted with caution given the non-randomized design and potential confounding variables.

These findings suggest that within the MAP range maintained (65-70 mmHg), cerebral oxygenation was preserved despite larger hemodynamic fluctuations in hypertensive patients.

Potential impairment of organ perfusion-particularly cerebral perfusion-remains a major concern during controlled hypotension. Reduced rSO_2 reflects a mismatch between cerebral oxygen delivery and demand and may indicate compromised perfusion. NIRS is a noninvasive modality that provides continuous monitoring of regional cerebral tissue oxygenation and is considered a reliable surrogate of cerebral perfusion.

Rigamonti et al.[20] reported that decreases in rSO_2 correlated with electroencephalographic and clinical findings suggestive of cerebral ischemia, supporting NIRS as a simple and continuous noninvasive monitoring tool. Similarly, Kim et al.[21] demonstrated a correlation between rSO_2 and jugular venous oxygen saturation, a recognized reference method for assessing global cerebral oxygenation.

In a study by Erdem et al.,[16] CH in 50 ASA I normotensive patients resulted in reductions of more than 20% from baseline rSO_2 values; CH was discontinued when this threshold was reached. In contrast, Shear et al.[22] reported that maintaining MAP between 55-65 mmHg did not significantly affect NIRS values. Cox et al.,[23] evaluating patients undergoing shoulder arthroscopy in the beach-chair position, observed cerebral desaturation detected by NIRS and reported a correlation between intraoperative blood pressure and rSO_2 values.

In the present study, hypertensive patients required greater reductions in MAP from baseline to achieve the target range of 65-70 mmHg. This initially suggested a potential for greater impairment in cerebral perfusion and oxygenation compared with normotensive patients. However, despite larger blood pressure fluctuations, no significant differences in NIRS-derived rSO_2 values were observed between groups. These findings indicate that within the maintained MAP range, CH did not adversely affect regional cerebral oxygenation in hypertensive patients compared with normotensive individuals.

POCD is a potential concern associated with controlled hypotension. Murkin et al.[24] demonstrated that intraoperative cerebral desaturation was associated with postoperative cognitive decline, stroke, and prolonged hospital stay. Similarly, Li et al.[25] suggested that impaired rSO_2 may contribute to the development of POCD.

In a study by Nowak et al.,[19] cognitive function was assessed using the Stroop test, Trail Making Test, and verbal fluency test at baseline, postoperative 6 hours, and 30 hours. Although a decline in Stroop test performance was observed at 6 hours, this impairment resolved by 30 hours, and intraoperative hypotension did not appear to influence late psychometric outcomes. Choi et al.[26] evaluated cognitive function using the MMT preoperatively and at postoperative 1 week in 60 patients undergoing CH and found no evidence of postoperative cognitive decline.

Consistent with these findings, no cognitive impairment was detected in our study at postoperative 6 and 24 hours based on MMT scores. Moreover, no differences were observed between hypertensive and normotensive patients (postoperative 6 hours: 28±2.0 vs 28±1.8; postoperative 24 hours: 28±1.7 vs 28±1.8).

Various pharmacologic strategies may be used to achieve controlled hypotension. Choi et al.[26] compared nitroglycerin and nicardipine and reported no differences in cerebral oxygen saturation or postoperative cognitive outcomes between groups. Tirelli et al.[18] compared total intravenous anesthesia (TIVA) using propofol-remifentanil with inhalational anesthesia using isoflurane-fentanyl and concluded that both techniques were safe for controlled hypotension; however, TIVA provided reduced bleeding and improved surgical field conditions, likely due to the selective arterial vasodilatory effects of propofol. Similarly, Eberhart et al.[17] compared propofol-remifentanil with isoflurane-alfentanil and found no significant differences in blood pressure control; however, lower heart rates and improved surgical conditions were reported in the TIVA group.

In the present study, CH was achieved using sevoflurane combined with remifentanil infusion. Hypertensive patients required significantly higher remifentanil doses to reach target MAP (Group 1 vs Group 2: 1002±579.3 μg vs 1511±838.9 μg), yet no additional hypotensive agents were necessary.

Bradycardia is among the most common adverse effects associated with remifentanil.[27] Despite higher remifentanil consumption in hypertensive patients, no clinically significant bradycardia requiring intervention occurred.

Although increased opioid administration in hypertensive patients might theoretically delay emergence and extubation, no significant difference in extubation time was observed between groups (Group 1 vs Group 2: 6±2.5 min vs 5±1.9 min).

All patients received tramadol 1 mg/kg IV for postoperative analgesia. Additional analgesic requirements were comparable between groups (Group 1 vs Group 2: 80±16.5 mg vs 86±16.3 mg).

PONV is a common complication; however, no episodes were observed in our cohort. Prophylactic antiemetic therapy was administered to all patients due to tramadol use.

Limitations

Several limitations should be acknowledged. First, this study was not randomized, and patient allocation was based on baseline blood pressure status, which may introduce selection bias and limit the internal validity of the findings.

Second, operative and anesthesia durations were longer in hypertensive patients (surgical duration: 90±30.8 min vs 113±30.8 min; anesthesia duration: 102±31.1 min vs

126±29.9 min). This may be attributable to the additional time required to achieve target blood pressure and potentially reduced surgical visibility during this period. Although the mean difference was approximately 20 minutes, we do not believe this interval was clinically sufficient to influence intraoperative rSO_2 or postoperative cognitive outcomes.

Third, cognitive function was assessed using the MMT alone. Although widely used, the MMT may not detect subtle cognitive changes. The lack of a comprehensive neuropsychological test battery may have limited the sensitivity for detecting mild postoperative cognitive dysfunction.

Additionally, potential confounding factors such as differences in intraoperative anesthetic requirements, hemodynamic variability, and duration of surgery between groups may have influenced the outcomes and should be considered when interpreting the results.

CONCLUSION

In this study evaluating the effects of CH during tympanoplasty in hypertensive patients, no differences were observed compared with normotensive patients in terms of intraoperative rSO_2 or early and late postoperative cognitive function. Although hypertensive patients required greater reductions in blood pressure and higher remifentanil doses to achieve target MAP, no increase in clinically significant bradycardia, delayed extubation, or PONV was observed. Within a MAP range of 65-70 mmHg, CH did not result in decreased rSO_2 or cognitive dysfunction in hypertensive patients. However, these findings should be interpreted cautiously due to the methodological limitations of the study, including the non-randomized design and limited cognitive assessment. Therefore, while CH appears to be feasible in this patient population under careful monitoring, further randomized controlled studies with more comprehensive cognitive evaluation are needed to confirm these findings.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by the Clinical Researches Ethics Committee of İstanbul Medeniyet University Göztepe Training and Research Hospital (Date: 09.01.2018, Decision No: 2017/0410).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights-including the right to withdraw at any time without consequence. All participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept and Study Design: CYA, SG; Data Collection: CYA; Manuscript Drafting: CYA, HAS; Critical Revision of the Manuscript: SG; Final Approval of the Manuscript: All Authors

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Eurasian Journal of
Anesthesiology & Intensive Care

Case Report

doi
10.51271/EAJAIC-0053

Diagnostic challenges in distinguishing TRALI from COVID-19 ARDS

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Cite this article: Koç Z, Silay E, Gür A, Aydoğan S. Diagnostic challenges in distinguishing TRALI from COVID-19 ARDS. *Eur J Anesthesiol Intens Care*. 2026;3(2):49-52. doi:10.51271/EAJAIC-0053

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Received: 05/01/2026 ◆ **Accepted**: 20/02/2026 ◆ **Published**: 16/05/2026

ABSTRACT

Severe acute respiratory syndrome caused by the SARS-CoV-2 virus (COVID-19) presents with a wide spectrum of clinical manifestations, ranging from asymptomatic infection to acute respiratory distress syndrome (ARDS) and multiorgan failure. Transfusion-related acute lung injury (TRALI) is a serious complication of blood transfusion and is associated with significant morbidity and mortality. A 46-year-old male patient with known chronic kidney disease presented to the emergency department with dyspnea and cough. The patient oxygen saturation was 78%. Laboratory findings revealed elevated white blood cell count, C-reactive protein, and D-dimer levels, along with lymphopenia; hemoglobin level was 7.3 g/dl. Chest computed tomography demonstrated diffuse bilateral ground-glass opacities with focal areas of consolidation. The patient was monitored in the intensive care unit with nasal oxygen therapy. Following blood transfusion for anemia, respiratory status deteriorated at the sixth hour post-transfusion, necessitating endotracheal intubation and initiation of mechanical ventilation. The deterioration in the patient's respiratory status requiring mechanical ventilation was attributed to several differential diagnoses, including ARDS secondary to COVID-19 or other microorganisms, TRALI, and transfusion-associated circulatory overload (TACO). Bedside chest radiography demonstrated bilateral diffuse pulmonary opacities. Transthoracic echocardiography showed no evidence of left atrial hypertension (LAH), thereby excluding TACO. Accordingly, in our patient, who had risk factors for ARDS but a stable respiratory status for 36 hours before transfusion, the diagnosis of Type II TRALI was considered. The patient was successfully extubated on day 9. Repeated SARS-CoV-2 PCR tests and rapid antibody tests were negative, and blood and urine cultures showed no growth. The patient was discharged to the ward on day 20. TRALI may develop even in the presence of risk factors for ARDS. For the differential diagnosis of TRALI, TACO, and ARDS, a comprehensive evaluation using clinical findings, laboratory parameters, and objective criteria such as echocardiography is recommended.

Keywords: ARDS, COVID-19, TACO, TRALI

INTRODUCTION

On December 31, 2019, an outbreak characterized by severe respiratory illness emerged in Wuhan, China. The virus responsible for this outbreak was identified as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which causes Coronavirus Disease 2019 (COVID-19).[1-4] The outbreak rapidly spread worldwide, resulting in millions of cases and thousands of deaths.

COVID-19 ARDS is diagnosed when someone with confirmed COVID-19 infection meets the Berlin 2012 ARDS diagnostic. ARDS develops in 42% of patients presenting with COVID-19 pneumonia, and 61-81% of those requiring intensive care. COVID-19 ARDS causes the typical ARDS pathological changes of diffuse alveolar damage in the lung.[5] Hypoxemic respiratory failure in ARDS generally results from intrapulmonary ventilation-perfusion mismatch or shunt and usually requires mechanical ventilation (MV). It was reported that 69% of patients admitted to the intensive care unit required mechanical ventilation. The mortality rates for ICU patients and those receiving mechanical ventilation were 28.3% and 43%, respectively. As the onset time of COVID-19-related ARDS was 8-12 days, it suggested that the 1-week onset limit defined by ARDS Berlin criteria did not apply to COVID-19-related ARDS.[6-8]

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Transfusion-related acute lung injury (TRALI) is a serious complication of blood transfusion and is among the leading causes of transfusion-related morbidity and mortality in most developed countries. In 2019, an international expert consensus modified and redefined the original TRALI definition established in 2004, and proposed new terminology.[9] TRALI is defined by the development of acute hypoxemia accompanied by bilateral pulmonary edema occurring during blood product transfusion or within six hours following transfusion.[10] Epidemiological data suggest that the incidence of TRALI is approximately one case per 130,000 transfused blood products; however, this estimate may differ according to the diagnostic criteria applied and the sensitivity of surveillance and reporting systems.[10,11] The pathogenesis of TRALI is complex and multifactorial. According to the widely accepted two-hit model, an initial inflammatory or pre-existing pulmonary condition primes the pulmonary vasculature, followed by a second hit-typically donor-derived antibodies or bioactive lipids in transfused blood products- leading to neutrophil activation, endothelial injury, and pulmonary edema.[12]

In this case report, we evaluated the deterioration in respiratory status that developed after blood transfusion in a 46-year-old patient hospitalized with suspected COVID-19 pneumonia according to the TRALI diagnostic criteria newly defined in 2019. The aim of this report is to highlight the possible association between COVID-19-related inflammatory status and TRALI, to discuss the diagnostic challenges in differentiating TRALI from other causes of acute respiratory deterioration, and to increase awareness of this rare but serious transfusion complication in the post-COVID-19 era.

CASE

A 46-year-old male smoker with known chronic kidney disease not requiring dialysis presented to the emergency department with dyspnea and cough. On admission, he was tachycardic, hypertensive, and tachypneic, with bilateral basal crackles on lung auscultation. He was afebrile, and oxygen saturation (SpO_2) was 78% on room air.

Non-contrast chest computed tomography revealed diffuse bilateral ground-glass opacities involving both upper and lower lobes, with focal areas of consolidation (Figure 1). Given the presentation during the COVID-19 pandemic, findings were considered consistent with COVID-19 pneumonia. Laboratory evaluation demonstrated elevated white blood cell count, C-reactive protein, and D-dimer levels, along with lymphopenia. Hemoglobin was 7.3 g/dl, creatinine 2.38 mg/dl, pH 7.21, and pCO_2 37.3 mmHg. Supplementary investigations obtained during the hospital course are summarized in Table.

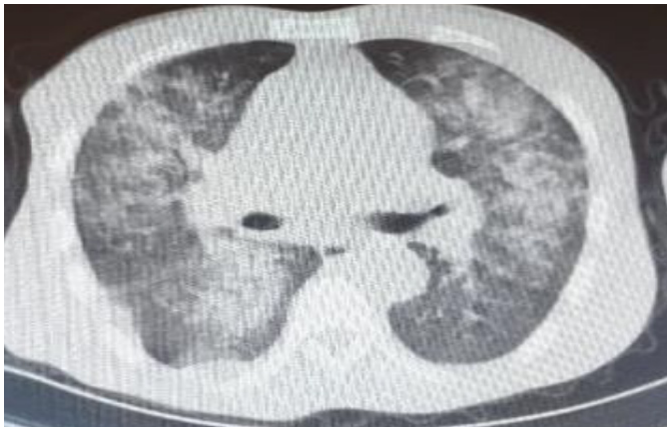

Figure 1. Chest computed tomography at emergency department admission

The patient was admitted to the intensive care unit. During the intensive care unit stay, the patient's vital signs were as follows: body temperature 36.2°C, blood pressure 176/101 mmHg, heart rate 115 beats/min, respiratory rate 28 breaths/min, and oxygen saturation 95% with nasal oxygen supplementation, decreasing to 88% on room air. Empirical treatment with favipiravir and azithromycin was initiated for suspected COVID-19, along with piperacillin-tazobactam and levofloxacin for possible bacterial infection. SARS-CoV-2 PCR testing was negative. Prior to transfusion, blood samples were collected for peripheral blood smear analysis and assessment of vitamin B12 and folate levels. Due to anemia, the patient received 2 units of packed red blood cells (PRBCs). The peripheral smear demonstrated hypochromic microcytic anemia. Serum vitamin B12 and folate levels were within

Table. Values of selected laboratory parameters

	Normal	Day 1	Day 2	Day 3	Day 5	Day 7	Day 9	Day 12	Day 18
WBC count 10^3/mcL	4-10	28.73	8.35	29.52	8.49	13.89	7.23	9.92	3.24
Lymphocyte count 10^3/mcL	0.8-3.2	0.94	0.74	0.8	0.23	0.29	0.26	0.36	0.67
Hemoglobin g/dl	13-16	7.3	7.2	10.8	8.3	8.4	7.7	8.5	7.9
Hematocrit (%)	37-47	27.5	25.8	35.5	28.1	28.7	22.6	28.1	26.3
CRP mg/L	0-5	40.3	62.3	55.9	67.7	30.8	32.6	35.5	69.7
ESR mm/h	0-15	29	19	17	29	12	42	49	22
D-dimer mcg/L	0-500	1730	720	2050	1630	1290	1090	1000	840
Fibrinogen (mg/dl)	2000-4000	4000		4720	4060	3650	3970	4190	3770
LDH (U/L)	140-280	278		391	300	290	235	263	
Creatinine (mg/dl)	0.6-1.3	2.38	2.52	2.34	2.83	2.73	2.05	2.39	1.65
Ferritin mcg/L	15-400	51	61	67	97	89	86	171	254
Procalcitonin mcg/L	0-0.5	0.35	12.68	10.39	46.20	19.65	6.4	1.14	0.25

WBC: White blood cell, CRP: C-reactive protein, LDH: Lactate dehydrogenase, ESR: Erythrocyte sedimentation rate

normal ranges. Despite the presence of acute inflammation, the low ferritin level was attributed to iron deficiency anemia and anemia of chronic disease. On day 2, hemoglobin remained low (7.2 g/dl), with normal bilirubin levels and negative direct and indirect Coombs tests; therefore, hemolytic anemia was excluded, and 3 additional units of PRBCs were transfused. The patient had a stable respiratory status for at least 36 hours prior to transfusion. Prior to blood transfusion, the patient had an oxygen saturation of 97-100% with nasal oxygen supplementation and 91-93% on room air, with a respiratory rate of 18-23 breaths/min. Six hours after transfusion, the patient developed tachypnea (38 breaths/min), tachycardia (134 beats/min), hypertension (174/112 mmHg), and hemoptysis, without fever. Oxygen saturation decreased to 50%, and the patient was subsequently intubated and placed on mechanical ventilation. The ventilator was set to pressure-regulated volume control (PRVC) ventilation mode with an FiO_2 of 70%, a respiratory rate of 12 breaths/min, a tidal volume of 490 ml, and a PEEP of 10 cmH_2O.

With a PaO_2/FiO_2 ratio of 221, the clinical picture was consistent with mild acute respiratory distress syndrome (ARDS). The deterioration in the patient's respiratory status requiring mechanical ventilation was attributed to several differential diagnoses, including ARDS secondary to COVID-19 or other microorganisms, secondary bacterial infection, TRALI, and transfusion-associated circulatory overload (TACO). Treatment targeting other potential microorganisms was expanded with the addition of trimethoprim-sulfamethoxazole, piperacillin-tazobactam was discontinued, and meropenem was initiated. Central venous pressure was 8 mmHg, and bedside chest radiography demonstrated bilateral diffuse pulmonary opacities (Figure 2). Transthoracic echocardiography showed no evidence of left atrial hypertension (LAH), thereby excluding TACO. Based on the presence of ARDS risk factors without established ARDS and respiratory deterioration within 6 hours of transfusion, the patient was diagnosed with TRALI Type II.

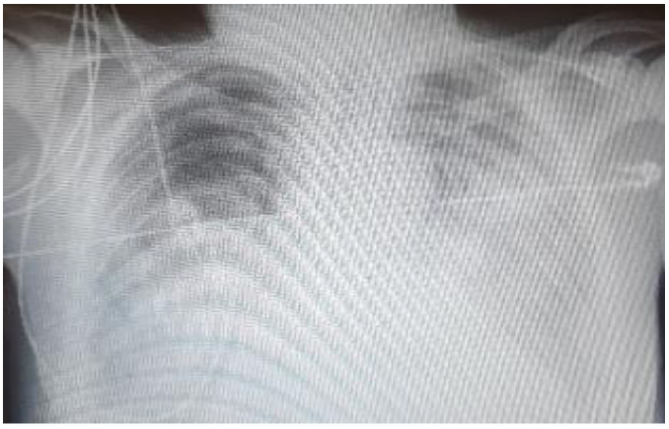

Figure 2. Post-transfusion chest X-ray

On day 3, vancomycin was added to the treatment regimen due to an elevated white blood cell count of $29.5\times10^3/\mu L$ and a procalcitonin level of 10.33 ng/ml.The patient was successfully extubated on day 9. Repeated SARS-CoV-2 PCR tests and rapid antibody tests were negative, and blood and urine cultures showed no growth. The patient was discharged to the ward on day 20.

DISCUSSION

The most common symptoms of COVID-19 include fever, dry cough, dyspnea, fatigue, and myalgia.[13] COVID-19-related ARDS develops in approximately 17% of patients as a result of alveolar damage, typically occurring 8-14 days after symptom onset.[6] Laboratory findings associated with COVID-19 include elevated white blood cell count, AST, ALT, high-sensitivity C-reactive protein (hs-CRP), and D-dimer levels, as well as lymphopenia and hypoalbuminemia.[14,15] Characteristic chest computed tomography findings consist of ground-glass opacities, often accompanied by areas of consolidation.[16] In the present case, the patient's clinical presentation, laboratory results, and radiological findings were consistent with COVID-19. Furthermore, ARDS developed approximately two weeks after symptom onset, which is in accordance with the typical disease course reported in the literature.

TRALI was redefined in 2019 and reclassified into TRALI Type I and TRALI Type II, with the term "possible TRALI" being eliminated. TRALI Type I is defined as an acute onset condition in patients without risk factors for ARDS, characterized by hypoxemia (SpO_2<90% on room air or a PaO_2/FiO_2 [P/F] ratio<300), bilateral pulmonary edema demonstrated by ultrasound, radiography, or computed tomography, absence of LAH, and symptom onset during or within 6 hours of transfusion. TRALI Type II includes the criteria of Type I and additionally applies to patients with risk factors for ARDS who do not meet ARDS diagnostic criteria or who have mild ARDS (P/F ratio 200-300), but whose respiratory status deteriorates following transfusion, with a stable respiratory status during the 12 hours preceding transfusion.[9]

For the differential diagnosis of TACO, it is recommended that pulmonary edema and LAH be objectively assessed using echocardiography and pulmonary imaging, rather than relying solely on indirect clinical signs such as hypertension or tachycardia.[6] In some cases, measuring brain natriuretic peptide (BNP) levels at the onset of pulmonary symptoms may be helpful, with BNP levels <300 pg/ml suggesting exclusion of TACO. Additionally, a pretransfusion-to-posttransfusion BNP ratio >1.5 has been reported to further rule out TACO.[9] In our case, BNP levels were not measured because there was no known history of cardiac disease and volume overload was assessed by echocardiography.

In our patient, who was initially managed for pneumonia, risk factors for ARDS were present, but respiratory status remained stable for at least 36 hours prior to transfusion. Respiratory deterioration occurred within 6 hours after transfusion, and post-transfusion chest radiography demonstrated bilateral pulmonary opacities. LAH was not detected on echocardiography, thereby excluding TACO. Accordingly, the patient was diagnosed with TRALI Type II. Accordingly, the patient was diagnosed with TRALI Type II.

The mainstay of treatment for TRALI is supportive care. If a blood transfusion is ongoing, it should be immediately discontinued. While a conservative approach with supplemental oxygen is sufficient in mild cases, mechanical

ventilation and intensive care support are required in more severe cases. The use of corticosteroids and diuretics is not recommended.[17] In some patients, recovery may take longer than one week.[18]

CONCLUSION

TRALI may develop even in the presence of ARDS risk factors, such as pneumonia, making the diagnosis challenging. For the differential diagnosis of TRALI, TACO, and ARDS, a comprehensive clinical assessment supported by laboratory findings and objective tools such as echocardiography is recommended. The occurrence of any transfusion-related complication should be reported to the respective blood transfusion services.We believe that as clinicians publish their cases, differential diagnoses related to this condition will be better established and overall awareness will increase.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient(s) included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Concept: ZK, ES, AG, SA; Design: ZK, ES; Control ZK, ES; Resources: ZK, ES, AG, SA; Materials: ZK, ES, AG, SA; Data Collection and/or Processing: ZK, ES, AG, SA; Analysis and/or Interpretation: ZK, ES, AG, SA; Literature Review: ZK, ES, AG, SA; Article Writing: ZK, ES; Critical Review: ZK, ES.

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MHA MEDIHEALTH ACADEMY

Eurasian Journal of Anesthesiology & Intensive Care

Case Report

doi 10.51271/EAJAIC-0054

Spinal epidural hematoma presenting with hypercapnic respiratory failure in diabetic ketoacidosis

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Cite this article: Duman Aydın P, Küçükosman G, Kongur E, Sağıroğlu AC. Spinal epidural hematoma presenting with hypercapnic respiratory failure in diabetic ketoacidosis. *Eur J Anesthesiol Intens Care.* 2026;3(2):53-55. doi:10.51271/EAJAIC-0054

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Received: 06/01/2026 ◆ **Accepted**: 20/03/2026 ◆ **Published**: 16/05/2026

ABSTRACT

Spinal epidural hematoma (SEH) is a rare but potentially devastating condition that may lead to severe neurological deficits if not recognized early. Diagnosis may be challenging when SEH occurs in patients with concurrent metabolic or infectious disorders. We report a case of SEH in a 70-year-old male patient who presented with diabetic ketoacidosis (DKA) and pneumosepsis. The patient was admitted to the intensive care unit and received treatment for DKA and infection. After metabolic stabilization, sedation was discontinued and the patient was extubated. Shortly after extubation, he developed acute respiratory failure accompanied by tetraplegia. Emergency contrast-enhanced cervicothoracic magnetic resonance imaging revealed an epidural hematoma extending from the C1-2 level to T6, causing significant spinal cord compression. Surgical intervention was not performed because of delayed diagnosis and lesion localization. This case highlights the importance of detailed neurological evaluation in patients with complex clinical presentations. Central causes such as SEH should be considered in cases of unexplained respiratory failure or sudden neurological deterioration.

Keywords: Spinal epidural hematoma, diabetic ketoacidosis, hypercapnia, tetraplegia

INTRODUCTION

Spinal epidural hematoma (SEH) is a rare but potentially devastating clinical entity with high morbidity.[1] It is estimated to occur in approximately 1 per 100,000 to 1 per 1,000,000 individuals per year in the general population.[2] SEH typically presents with sudden onset neck or back pain and may rapidly progress to neurological deficits such as motor weakness or paralysis.[3,4] Delayed diagnosis may lead to irreversible outcomes including permanent tetraplegia or death.[4] When SEH occurs concurrently with metabolic conditions such as diabetic ketoacidosis (DKA) and infectious processes such as pneumonia, diagnostic recognition becomes even more challenging.

This case of cervicothoracic SEH in a patient hospitalized for DKA and pneumosepsis illustrates the difficulty of timely diagnosis in patients with overlapping metabolic and infectious disorders.

CASE

Written informed consent was obtained from the patient for publication. A 70-year-old male with a history of hypertension, diabetes mellitus, and benign prostatic hyperplasia presented to the emergency department with generalized weakness and deterioration in overall condition, reporting persistent neck and back pain for one week without a history of trauma. The patient was conscious, oriented, and cooperative, with moderately impaired general condition.

Initial laboratory tests revealed hyperglycemia (blood glucose 385 mg/dl), positive urinary ketones (++), elevated C-reactive protein (365 mg/L), procalcitonin (1.7 ng/ml), and leukocytosis (white blood cell count 37,000/μL). Arterial blood gas analysis showed combined metabolic and respiratory acidosis (pH 7.1, pCO_2 56 mmHg, HCO_3 14 mmol/L). Peripheral oxygen saturation on room air was below 90%, accompanied by respiratory distress. Thoracic computed tomography demonstrated areas of consolidation, consistent with pneumosepsis. The patient was admitted to the intensive care unit (ICU) for management of concurrent DKA and pneumosepsis. Within approximately one hour, respiratory distress progressed, accompanied by carbon dioxide retention (pCO_2 85-86 mmHg) and impaired

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consciousness, necessitating endotracheal intubation. Neurological assessment was not feasible due to sedation and analgesia initiated after intubation. Arterial blood gas analysis demonstrated combined metabolic and respiratory acidosis, and treatment for DKA was initiated according to endocrinology recommendations. At the 16th hour of ICU follow-up, blood glucose levels normalized and ketonemia resolved, allowing sedation to be discontinued. Extubation was performed at the 20th hour; however, within minutes, the patient developed acute respiratory failure and complete motor paralysis, necessitating immediate re-intubation. Emergency cervical magnetic resonance imaging (MRI) identified a fluid-containing collection at the C1-2 level, and contrast-enhanced cervicothoracic MRI demonstrated an epidural hemorrhagic collection extending from C1-2 to T6, causing anterior spinal cord compression (**Figure**). Neurosurgical consultation concluded that surgical intervention was not feasible due to craniospinal localization and delayed diagnosis.

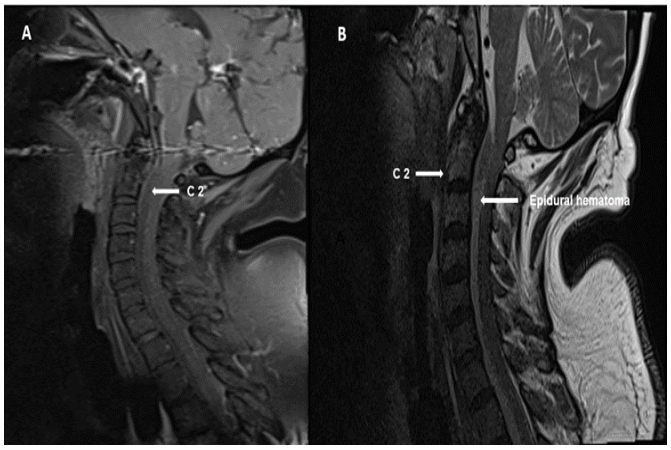

Figure. Contrast-enhanced cervicothoracic magnetic resonance imaging demonstrating an epidural hematoma extending from the C1-2 level to the upper thoracic spine, causing anterior compression of the spinal cord.
(A) Upper cervical level demonstrating the epidural hematoma. (B) Spinal cord compression caused by the epidural hematoma

Mechanical ventilation was maintained for six days, after which tracheostomy and percutaneous endoscopic gastrostomy were performed in the same session. Following improvement of pneumonia, the patient was transferred to a palliative care unit on the 25th day of hospitalization with home-type mechanical ventilation support.

DISCUSSION

SEH typically presents with acute neck or back pain and may progress rapidly to motor deficits or paralysis.[2-5] In this case, neck and back pain was the initial symptom, providing an important clue for spinal epidural pathology. However, the simultaneous presence of DKA and pneumonia contributed to a delay in diagnosis.

In DKA, compensatory hyperventilation and resultant hypocapnia are typically expected as a physiological response to metabolic acidosis.[6,7] In contrast, this patient exhibited hypercapnia (pCO_2 56 mmHg), which could not be explained by DKA alone and suggested involvement of the respiratory muscles. High cervical spinal cord compression can impair respiratory function by affecting the phrenic nerve, which primarily originates from the C3-C5 segments and is essential for diaphragmatic function.[8] Although the hematoma was located at the C1-2 level, its mass effect extended along the cervical spinal cord, disrupting diaphragmatic and intercostal muscle innervation and resulting in hypercapnic respiratory failure.

The exact mechanism underlying spontaneous SEH remains unclear. Reported risk factors include anticoagulant or antiplatelet therapy, hypertension, anemia, diabetes, hypothyroidism, pregnancy, and activities such as deep diving.[10] In our patient, there was no history of trauma or use of anticoagulant medication, and no evidence of coagulopathy was identified. Nevertheless, the presence of diabetes and metabolic disturbances associated with DKA may have contributed to vascular fragility and spontaneous hemorrhage.

There are currently no established guidelines for the management of spontaneous SEH. Early surgical decompression is generally preferred, but conservative management may be considered in selected patients under close neurological monitoring.[5] Optimal neurological outcomes are associated with decompression performed within the first 24 hours after symptom onset.[9] In this case, however, the cranioservical location of the lesion, delayed diagnosis, and high surgical risk precluded operative intervention.

Clinicians should maintain a high index of suspicion for spinal pathology in patients with multifactorial conditions who develop unexplained hypercapnia or acute neurological deterioration.

CONCLUSION

This case highlights the importance of thorough history taking and neurological examination in diagnosing SEH. Unexplained hypercapnia or sudden neurological deterioration in patients with multifactorial conditions should prompt consideration of spinal pathology.

ETHICAL DECLARATIONS

Informed Consent

Written informed consent was obtained from the patient(s) included in this report. Signed consent forms are retained by the authors and are available upon request.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Concept: PDA, ACS; Design: PDA, GK, ACS; Control: ACS, EK; Resources: PDA, GK; Materials: PDA, GK; Data Collection and/or Processing: PDA, GK, EK; Analysis and/or Interpretation:

PDA, ACS, EK; Literature Review: PDA, GK, ACS; Article Writing: PDA, GK, EK, ACS; Critical Review: ACS, EK.

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