

EVALUATION OF EARLY POSTOPERATIVE KIDNEY DYSFUNCTION IN PATIENTS UNDERGOING ELECTIVE OPEN ABDOMINAL AORTIC ANEURYSM REPAIR WITH INTRAOPERATIVE HYPOTENSION AND CONTINUOUS VASOPRESSOR SUPPORT

Trposka Poposka A.^{1,2}, Kuzmanoska B.^{1,2}, Jovanovski Srceva M.^{1,2}, Naumovski F.^{1,2}, Leshi A.^{1,2}, Stavrik K.^{3,2}.

¹University Clinic for Traumatology, Orthopedics, Anesthesia, Reanimation, Intensive Care and Emergency Department – Skopje; Department for Anesthesia, Reanimation and Intensive Care – Skopje; University Clinical Center “Mother Theresa” – Skopje;

²Faculty of Medicine, Ss. Cyril and Methodius University in Skopje, Skopje, North Macedonia;

³Center for Family Medicine, Faculty of Medicine, Ss. Cyril and Methodius University in Skopje, Skopje, North Macedonia.

Abstract

Background: Development of postoperative kidney dysfunction is one of the most common and serious perioperative complications in patients undergoing open abdominal aortic aneurysm (AAA) repair resulting from several factors. In this context, intraoperative hypotension and the subsequent need for vasopressor support are associated with postoperative kidney dysfunction, although their individual contributions are difficult to distinguish.

Objectives: To evaluate early postoperative renal-function changes in patients who required continuous intraoperative norepinephrine infusion to treat hypotension.

Methods: A prospective observational cohort study included 20 patients undergoing elective open abdominal aortic aneurysm repair. Patients were divided into two groups according to the intraoperative need for continuous norepinephrine infusion. Intraoperative hemodynamic parameters, acid-base status, and postoperative renal function markers were analyzed and compared between the groups.

Results: Patients receiving intraoperative norepinephrine infusion had significantly lower minimum mean arterial pressure values compared with patients without vasopressor support (61.5 vs. 75.75 mmHg, $p < 0.005$). Significantly higher serum creatinine levels (124.5 vs. 79.88 $\mu\text{mol/L}$, $p = 0.03$) and significantly lower 24-hour urine output (1700 vs. 2450 mL, $p = 0.002$) were observed in the norepinephrine group 48 hours following surgery.

Conclusion: Patients requiring intraoperative vasopressor support during open abdominal aneurysm repair experience higher level of hemodynamic instability, more severe hypotension, and less favorable early postoperative kidney dysfunction markers. However, these findings cannot establish direct adverse renal dysfunction effects.

Keywords: *acute kidney dysfunction; abdominal aortic aneurysm; intraoperative hypotension; vasopressor support.*

Introduction

Open surgical repair of an abdominal aortic aneurysm (AAA) is a major vascular procedure associated with significant hemodynamic fluctuations, aortic cross-clamping, potential blood loss, and ischemia-reperfusion injury. These perioperative challenges may compromise renal perfusion and contribute to the development of early postoperative renal dysfunction, ranging from transient alterations in renal function to clinically significant acute kidney injury (AKI) (1,2).

Therefore, postoperative kidney dysfunction is an important concern in these patients. Renal dysfunction after open AAA repair rarely has a single cause. Reduced perfusion pressure, ischemia-reperfusion injury, and perioperative inflammation may interact with patient-related factors such as older age, hypertension, diabetes, and pre-existing renal impairment (2–4). The frequency of postoperative renal dysfunction varies across studies, partly due to differences in case mix and diagnostic criteria (1,2).

Intraoperative hypotension represents an important potentially modifiable factor associated with postoperative renal dysfunction. Observational studies have demonstrated that both the severity and duration of intraoperative hypotension are associated with postoperative kidney dysfunction (5–7). Contemporary perioperative recommendations emphasize maintaining adequate mean arterial pressure (MAP), generally at least 60–65 mmHg, to reduce the risk of hypotension-associated organ injury. Nevertheless, optimal blood pressure targets should be individualized according to baseline cardiovascular status, comorbidities, and clinical condition (8).

To maintain adequate systemic perfusion, vasopressors such as norepinephrine are frequently administered when hypotension persists despite appropriate hemodynamic optimization. Norepinephrine increases systemic vascular resistance and arterial pressure primarily through alpha-adrenergic receptor stimulation. Although norepinephrine may improve systemic and renal perfusion pressure, its relationship with postoperative renal outcomes is complex. Vasopressor requirement may reflect the severity of underlying hemodynamic instability rather than a direct adverse effect of the drug itself (9,10).

Recent observational evidence suggests that both intraoperative hypotension and cumulative norepinephrine dose are associated with postoperative renal dysfunction in patients undergoing noncardiac surgery. However, these findings do not establish a causal relationship between norepinephrine administration and renal injury (10). This distinction is particularly relevant during major vascular surgery, where hemodynamic instability, aortic cross-clamping, and reperfusion may simultaneously affect renal perfusion and contribute to postoperative renal dysfunction or AKI.

Acute kidney injury is a clinically significant form of acute renal dysfunction characterized by a rapid deterioration in kidney function. According to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, AKI is diagnosed when serum creatinine increases by ≥ 0.3 mg/dL (≥ 26.5 μ mol/L) within 48 hours, rises to ≥ 1.5 times baseline within the preceding seven days, or when urine output decreases below 0.5 mL/kg/h for at least six hours (13). These criteria provide a standardized frame-

work for diagnosing and classifying AKI in patients undergoing major surgical procedures.

However, early postoperative deterioration in renal function may occur without meeting the criteria to establish a AKI formal diagnosis. Changes in serum creatinine and urine output may provide clinically relevant information during the early postoperative period, even when formal patient-level KDIGO classification has not been performed.

Early recognition of postoperative renal dysfunction is clinically important, as deterioration in renal function, particularly when progressing to AKI, may be associated with increased morbidity, prolonged hospitalization, higher healthcare costs, and unfavorable long-term renal and cardiovascular outcomes (3,4,12).

Despite increasing recognition of the relationship between intraoperative hypotension, vasopressor administration, and postoperative renal complications, the association between hemodynamic instability, norepinephrine requirement, and early postoperative changes in renal function following open AAA repair warrants further investigation.

Therefore, the present study aimed to evaluate the association between intraoperative hypotension, the requirement for continuous norepinephrine infusion, and early postoperative renal dysfunction in patients undergoing elective open abdominal aortic aneurysm repair. Early postoperative renal function was assessed using serum creatinine levels and urine output within the first 48 hours after surgery, without formal classification of AKI according to KDIGO criteria.

Objective

The primary objective was to compare early postoperative renal function markers in patients who did and did not require continuous intraoperative norepinephrine during elective open infrarenal AAA repair.

The secondary objectives were to describe differences in minimum intraoperative MAP, acid-base parameters, perioperative fluid administration, aortic cross-clamp duration, urine output, and length of hospital stay. The study was exploratory and was not designed to establish causality.

Materials and Methods

This study was conducted as an observational prospective cohort pilot study that included patients undergoing elective surgical treatment for infrarenal abdominal aortic aneurysm repair. The research was conducted over a six-month period at the University Clinic for Anesthesia, Reanimation, and Intensive Care (KARIL) in Skopje, in collaboration with the University Clinic for Thoracic and Vascular Surgery. The study cohort was hospitalized and treated at the University Clinic for Thoracic and Vascular Surgery in Skopje. Prior to enrollment in the study, patients provided written informed consent.

All patients aged 60-80 undergoing infrarenal surgical AAA repair were included in the study, while patients with pre-existing renal disease, emergency cases, patients with confirmed chronic kidney disease, patients with impaired left ventricular systolic function ($EF < 45\%$), patients scheduled for endovascular treatment, and those who did not sign an informed consent form were not included.

To meet the study objectives, enrolled patients were categorized into two groups after surgery based on their need for intraoperative vasopressor support. The first group (n=10) included patients who received continuous vasopressor (norepinephrine) infusion only during the intraoperative period based on MAP (according to the protocol as POQI;2024; Saugel et al.(10)); while the second group (n=10) included patients in whom vasopressor support was not used.

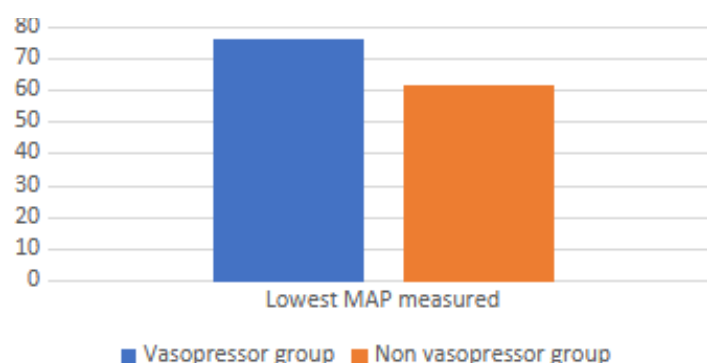
Data obtained from all patients' data sheets were evaluated and analyzed for demographic data, hemodynamic profile, the duration of hypotension, and the intraoperative administration of crystalloids, colloids, blood, and blood products (measured in milliliters). With a focus on the intraoperative period, and postoperative values of generally accepted markers of renal injury were also analyzed, as well as the time for cross-clamping of the aorta, acid-base status, duration of the procedure, and postoperative outcomes. In the postoperative period, 48 hours after the intervention, levels of renal injury markers were analyzed and compared between the two study groups, the cumulative dose of vasopressor use in the first group and the time of vasopressor usage.

Standard descriptive statistical analysis was used in the study to summarize data including mean and median values. Depending on the distribution of numerical values, comparison between the two groups were performed using the Student's t-test or the Mann-Whitney U test, as appropriate.

Results

A total of 20 patients were included in the study, 10 (50%) received a continuous vasopressor (norepinephrine) infusion during the intraoperative period, while the remaining patients did not receive vasopressor support. Gender distribution was not a statistically significant differentiator between the two groups, and most of the patients in both groups were male. The mean age was not statistically different between the two groups (69.13 (SD 6.58) vs 67.63 (SD 7.21) (Student's t-test: $t=0.43$, $p=0.33$).

Group of patients receiving norepinephrine infusion during the intraoperative period, showed statistically significantly lower values of the lowest mean arterial pressure with a mean value of 61.5 mmHg, compared to 75.75 mmHg in patients without intraoperative vasopressor support (Student's t-test: $t=2.97$, $p < 0.005$) (Graph 1). Duration of vasopressor infusion in this group was $56.9 \text{ min} \pm 23.5 \text{ min}$ (mean $\pm$ SD). Patients in this group received a continuous infusion of norepinephrine with mean dose of $0.215 \pm 0.078 \text{ } \mu\text{g/kg/min}$ and cumulative dose of $940.9 \pm 623.7 \text{ } \mu\text{g}$ in total.



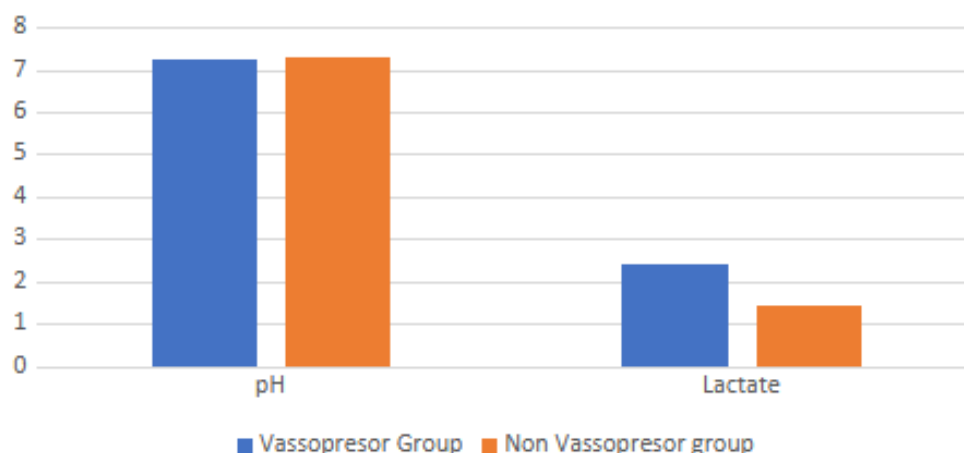
Graph No. 1: Graphical representation of the mean lowest MAP in both study groups.

Table No. 1: Doses of norepinephrine received in the vasopressor group

Variable	Norepinephrine group (n=10)
Norepinephrine dose ($\mu\text{g/kg/min}$)	0.215 ± 0.078
Cumulative norepinephrine dose (μg)	940.9 ± 623.7
Duration of norepinephrine infusion (min)	56.9 ± 23.5

No statistically significant difference regarding intraoperative crystalloid infusion volumes was detected; the mean crystalloid administration was 4,000 mL in the norepinephrine group and 3,500 mL in the no-norepinephrine group ($p=0.23$).

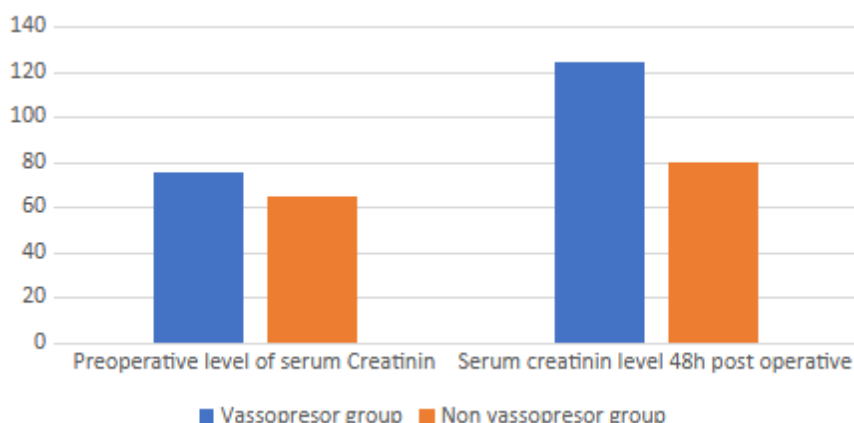
Equal amounts of colloids were infused in both groups during the intraoperative period. Furthermore, no statistically significant differences were observed regarding cross-clamp duration: 77.88 minutes for patients receiving continuous norepinephrine infusion versus 70.25 minutes for those not receiving norepinephrine infusion (Student's t-test: $t=1.14$; $p=0.13$). In the group of patients treated with norepinephrine infusion, non-significantly lower pH values were measured with a median of 7.25 compared to 7.34 in patients treated without vasopressor support (Mann-Whitney U test: $U=21$; $z=-1.1$; $p=0.123$) (Graph 2) along with non-significantly higher lactate levels in patients receiving vasopressor support (median 2.41 vs. 1.43 in those not requiring vasopressors; Mann-Whitney U test: $U=18$; $z=-1.4$; $p=0.07$) (Graph 2).

**Graph No. 2:** Graphical representation of pH and lactate levels in both study groups.

In patients receiving continuous vasopressor support intraoperatively, higher preoperative serum creatinine levels were measured with a median of 75.63 compared to 65.07 in those who did not require continuous norepinephrine infusion (Mann-Whitney U Test: $U=21$; $z=-1.1$, $p=0.123$) (Graph No. 3). The difference was not statistically significant.

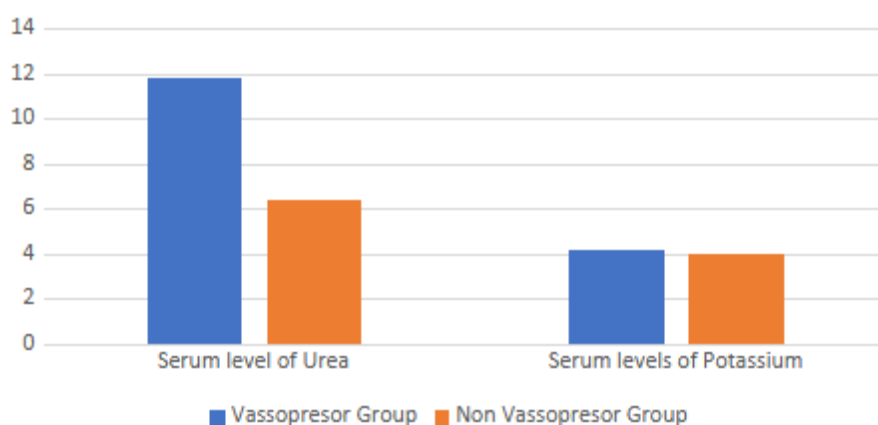
Forty-eight hours after the intervention, statistically significantly higher serum creatinine levels were measured, with a mean value of $124.5 \mu\text{mol/L}$ in patients who received intraoperative norepinephrine infusion, compared to $79.88 \mu\text{mol/L}$ in patients who did not require continuous vasopressor support

(Student's t-test: $t=1.96$; $p=0.03$) (Graph No. 3).



Graph No. 3: Preoperative creatinine level and creatinine level at 48 hours after surgery in patients with and without the need for vasopressor support.

Furthermore, in patients requiring intraoperative vasopressor support, measurements taken 48 hours after the intervention showed, albeit statistically nonsignificant, higher urea levels (mean: 11.83 vs. 6.38 in patients not requiring vasopressors; Student's t-test: $t=1.29$, $p=0.1$; Graph 4) and potassium levels (median: 4.23 vs. 3.99; Mann-Whitney U test: $U=29$, $z=-0.3$, $p=0.37$; Graph 4).



Graph No. 4: Urea and potassium levels 48 hours after the intervention in patients with and without the need for intraoperative vasopressor support.

In patients receiving intraoperative norepinephrine infusion, insignificantly lower urine output values were measured with a median of 500 mL, compared to 700 mL in patients who did not receive a vasopressor (Mann-Whitney U Test: $U=30$; $z=-0.2$; $p=0.41$).

Forty-eight hours after the procedure, patients who had received intraoperative vasopressor support showed statistically significantly lower values for 24-hour urine output (median: 1700 mL), compared to the group that did not receive continuous intraoperative vasopressor support (median: 2450 mL) (Mann-Whitney U Test: $U=5.50$; $z=-2.7$; $p=0.002$). Renal markers are shown in Table 3.

No statistically significant difference was found in relation to the length of hospital stay between the two analyzed groups; the mean duration was 8.5 days for patients receiving vasopressor infusion versus 8 days for those who did not receive a vasopressor intraoperatively (Student's t-test: $t=0.31$; $p=0.37$).

Table No. 2: Perioperative characteristics according to intraoperative norepinephrine requirement

Variable	Norepinephrine (n=10)	No norepinephrine (n=10)	p value
Age, years	69.13 $\pm$ 6.58	67.63 $\pm$ 7.21	0.33
Minimum MAP, mmHg	61.5	75.75	<0.005
Crystalloids, mL	4000	3500	0.23
Aortic cross-clamp duration, min	77.88	70.25	0.13
pH	7.25	7.34	0.123
Lactate, mmol/L	2.41	1.43	0.07
Intraoperative urine output, mL	500	700	0.41

Table No. 3: Renal function markers and postoperative outcome

Variable	Norepinephrine (n=10)	No norepinephrine (n=10)	p value
Baseline serum creatinine, μ mol/L	75.63	65.07	0.123
Serum creatinine at 48 h, μ mol/L	124.5	79.88	0.03
Urea at 48 h	11.83	6.38	0.10
Potassium at 48 h, mmol/L	4.23	3.99	0.37
24-h postoperative urine output, mL	1700	2450	0.002
Length of hospital stay, days	8.5	8.0	0.37

Values are presented as mean $\pm$ SD or as the reported central value from the source dataset, according to the original statistical analysis. MAP, mean arterial pressure.

Discussion

Acute kidney dysfunction is one of the most serious complications following open repair of an abdominal aortic aneurysm. Open repair of an abdominal aortic aneurysm (AAA) is a complex and highly invasive vascular surgical procedure associated with profound systemic physiological stress and significant intraoperative hemodynamic fluctuations. The combination of manipulation of major blood vessels, aortic clamping, potential massive blood loss, and subsequent reperfusion imposes a complex pathophysiological burden on organ systems, particularly the kidneys (1-3).

Among numerous potentially modifiable factors, intraoperative hypotension is considered one of the most important risk factors for postoperative renal dysfunction. Numerous studies have shown that even brief periods of reduced mean arterial pressure are associated with an increased risk of postoperative organ dysfunction, specifically acute kidney injury and complications (5–7). The kidneys are particularly sensitive to changes in systemic perfusion, and prolonged or severe hypotension can lead to impaired renal blood flow, reduced glomerular filtration, and ischemic injury (1,4,7).

The PeriOperative Quality Initiative (POQI) international consensus statement recommends avoiding sustained intraoperative hypotension and generally maintaining MAP at or above 60–65 mmHg, with adjustments based on individual patient needs and clinical conditions (10). In patients undergoing open AAA repair, maintaining adequate perfusion pressure is particularly important because aortic cross-clamping, unclamping, blood loss, and anesthesia-related vasodilation may produce substantial changes in systemic and regional hemodynamics.

In modern anesthetic practice, norepinephrine is the first-choice vasopressor for the treatment of intraoperative arterial hypotension. Its action is mediated primarily through alpha-adrenergic stimulation, resulting in vasoconstriction, increased systemic vascular resistance, and the maintenance of mean arterial pressure. It enables the maintenance of systemic arterial pressure and adequate organ perfusion during hemodynamic instability, particularly in patients undergoing major surgical procedures associated with significant hemodynamic fluctuations. In patients undergoing open repair of an abdominal aortic aneurysm, norepinephrine is frequently used to correct hemodynamic instability caused by aortic clamping and reperfusion phenomena, changes in afterload, blood loss, and anesthesia-induced vasodilation. Consequently, the need for intraoperative norepinephrine infusion can be considered an indirect indicator of a more complicated intraoperative course and more pronounced hemodynamic instability (1,7,10-13).

In the present prospective observational study, patients requiring continuous intraoperative norepinephrine infusion exhibited significantly lower minimum mean arterial pressure (MAP) values, compared with patients who did not receive vasopressor support (61.5 vs. 75.75 mmHg; $p < 0.005$). The mean minimum MAP in the norepinephrine group was below 65 mmHg - a threshold commonly associated with an increased risk of postoperative organ injury. This finding is particularly significant because the average lowest MAP value in this group fell below the 65 mmHg threshold, which is currently considered a critical limit below which the risk of organ hypoperfusion and postoperative acute kidney dysfunction and injury increases substantially. Previous studies by Walsh et al. and Salmasi et al. demonstrated associations between intraoperative hypotension and postoperative renal complications; while the systematic review by Wesselink et al. emphasized the importance of both the severity and duration of hypotensive episodes (5–7).

In our study, patients receiving norepinephrine had significantly lower minimum MAP values, suggesting greater intraoperative hemodynamic instability. Therefore, the observed association between norepinephrine administration and postoperative renal dysfunction should not be interpreted as evidence of a direct effect of norepinephrine. Rather, vasopressor requirement may represent a marker of more pronounced circulatory instability and underlying disturbances in tissue perfusion. This distinction is particularly important because norepinephrine was administered based on clinical need rather than randomized allocation.

Serum creatinine is a routinely used marker for evaluating changes in renal function and is incorporated into the Kidney Disease: Improving Global Outcomes (KDIGO) diagnostic criteria for AKI (13). In the present study, preoperative serum creatinine levels did not differ significantly between the groups. However, 48 hours after surgery, patients requiring intraoperative norepinephrine support exhibited significantly higher serum creatinine concentrations than patients who did not receive vasopressors (124.5 vs. 79.88 $\mu\text{mol/L}$; $p = 0.03$).

These findings indicate an association between intraoperative vasopressor requirement and impaired early postoperative renal function. Nevertheless, the absence of a statistically significant difference in baseline creatinine does not establish equivalence of preoperative renal function, particularly in a small study population. Furthermore, although serum creatinine increased in the norepinephrine group, formal patient-level KDIGO classification was not performed. Consequently, the present findings should be interpreted as changes in early postoperative renal function rather than as a confirmed difference in AKI incidence.

In addition to serum creatinine, urine output represents an important clinical parameter for assessing renal function and is included in the KDIGO diagnostic criteria for AKI (11-14). In our study, intraoperative urine output did not differ significantly between the groups. However, postoperative 24-hour urine output was significantly lower in patients requiring norepinephrine support compared with those who did not receive vasopressors (1700 vs. 2450 mL; $p=0.002$).

The lower postoperative urine output observed in the norepinephrine group, together with higher postoperative serum creatinine concentrations, suggests less favorable early renal functional outcomes in patients experiencing greater intraoperative hemodynamic instability. Nevertheless, total 24-hour urine volume alone is insufficient for diagnosing oliguria according to KDIGO criteria, based on which the urine output is evaluated relative to body weight and duration. Moreover, postoperative urine output may be influenced by fluid balance, diuretic administration, neurohormonal responses, and other perioperative factors. In the present study, no statistically significant differences were observed between the groups in the volume of administered crystalloids and colloids or the duration of aortic cross-clamping. Although these findings reduce the likelihood that the measured differences in these variables alone explain the observed renal outcomes, residual confounding cannot be excluded. The relatively small sample size also limits the ability to identify clinically relevant differences in other perioperative factors.

Additional indicators of tissue perfusion, including arterial pH and serum lactate, were evaluated. Patients requiring norepinephrine support showed numerically lower pH values (7.25 vs. 7.34) and higher serum lactate concentrations (2.41 vs. 1.43 mmol/L). However, these differences did not reach statistical significance. Therefore, they should be interpreted as descriptive findings rather than evidence of a confirmed difference in systemic tissue hypoperfusion. Nevertheless, their direction is compatible with the greater hemodynamic instability observed in patients requiring vasopressor support.

Taken together, our findings suggest that patients requiring continuous intraoperative norepinephrine infusion during elective open AAA repair experience greater hemodynamic instability and less favorable early postoperative renal functional parameters. These observations are consistent with previous studies demonstrating associations between intraoperative hypotension, vasopressor exposure, and postoperative kidney injury (5–7,9,10).

Limitations and Further Suggestions

The present study has several limitations. First, the sample size only 20 patients limited statistical power and the generalizability of the findings. Second, the observational design and clinically determined administration of norepinephrine introduce potential confounding by indication. Third,

although minimum MAP values were recorded, the cumulative duration and severity of hypotension were not quantified in the present analysis. Fourth, renal function was evaluated using serum creatinine and urine output without formal patient-level KDIGO classification, preventing reliable determination of AKI incidence and severity.

Despite these limitations, the study provides preliminary clinical evidence supporting the importance of careful intraoperative hemodynamic monitoring and early postoperative assessment of renal function in patients undergoing open AAA repair.

Conclusion

According to our study, continuous intraoperative norepinephrine requirement was associated with lower minimum MAP values, higher serum creatinine concentrations at 48 hours after surgery, and reduced postoperative urine output following elective open AAA repair. These findings suggest that vasopressor requirement may identify patients at increased risk of early postoperative renal dysfunction. However, the observational design does not allow the establishment of a causal relationship between norepinephrine administration and renal impairment. Larger prospective studies incorporating detailed hypotension exposure, vasopressor dosing, and standardized KDIGO assessment are required to clarify these associations.

Declarations

Ethics approval and consent to participate: This study was approved by the Ethics Committee of the Faculty of Medicine, Ss. Cyril and Methodius University in Skopje (Approval No. 02-2848/15, dated 7 May 2026).

Informed consent: Written informed consent was obtained from all participants prior to inclusion in the study.

Conflict of interest: The authors declare no conflict of interest.

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Availability of data and materials: Data are available from the corresponding author upon reasonable request.

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