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CASE SERIES

Perioperative Anesthetic Management of Ruptured Aortic Aneurysms: A Case Series from a Tertiary Care Hospital

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Abstract

A ruptured aortic aneurysm is an ASA physical class 5 emergency associated with high perioperative morbidity and mortality, necessitating rapid multidisciplinary decision-making and intervention. The ruptured aortic aneurysm cases must be centralized to high-volume centers capable of performing both open and endovascular repair to minimize morbidity and mortality. During intervention, the priorities include goal-directed fluid resuscitation, implementing permissive hypotension without compromising end-organ perfusion, and managing massive transfusion while counteracting the lethal triad of acidosis, hypothermia, and coagulopathy. Based on the patient's status and procedural management, anesthesia will be tailored to general or local/monitored anesthesia. This case series highlights outcomes and discharge dispositions of various procedural interventions for ruptured aortic aneurysms.

Keywords: Aortic aneurysm, massive blood transfusion, coagulopathy, aortic cross-clamping

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Introduction

An aneurysm is an abnormal, localized widening of an artery. An abdominal aortic aneurysm (AAA) is typically defined as a permanent dilation of the abdominal aorta to at least 30 mm or more than 50% of its original diameter at the level of the renal arteries, most often involving the infrarenal segment. Risk factors include smoking, male sex, older age, systemic hypertension, hypercholesterolemia, a history of peripheral and coronary artery disease, connective tissue disorders, and a family history of aneurysm [1].

Population-based studies have estimated that the prevalence of AAA is 3–5% in elderly men aged 65 years or older, whereas it has doubled to 10% in those aged 80 years or older. The dreaded complication of any AAA is rupture, whether contained or free, and the formation of an intraperitoneal or retroperitoneal hematoma. Nearly 50% of the patients with ruptured aortic aneurysm die before reaching the hospital, and another one-third of the patients die during interventions or postoperatively [2]. The Society for Vascular Surgery (SVS) recommended that the “door to intervention” time be 90 minutes within a

30/30/30 framework for diagnosis, transfer, anesthesia preparation, and surgical intervention [3].

We report a case series of five patients who underwent emergency aortic repair (three open surgical repairs [OSR] for ruptured abdominal aortic aneurysms and two endovascular repairs [EVAR]) for ruptured thoracoabdominal aortic aneurysms. This case series highlights the role of goal-directed fluid resuscitation, advanced hemodynamic monitoring, massive blood transfusion, and permissive hypotension in managing ASA class 4 and 5 of ruptured aortic aneurysms.

Case 1: open surgical repair of ruptured infrarenal AAA

An elderly man in his sixties presented with sudden acute abdominal pain, profuse sweating, and agitation. On examination, he revealed a pulsatile mass with relative hemodynamic instability, requiring rapid-sequence intubation (RSI), and was started on vasopressor and inotropic support in ED. Computed Tomography angiography (CTA) imaging showed a 10 x 7 cm infrarenal ruptured aortic aneurysm with retroperitoneal hematoma (Figure 1).

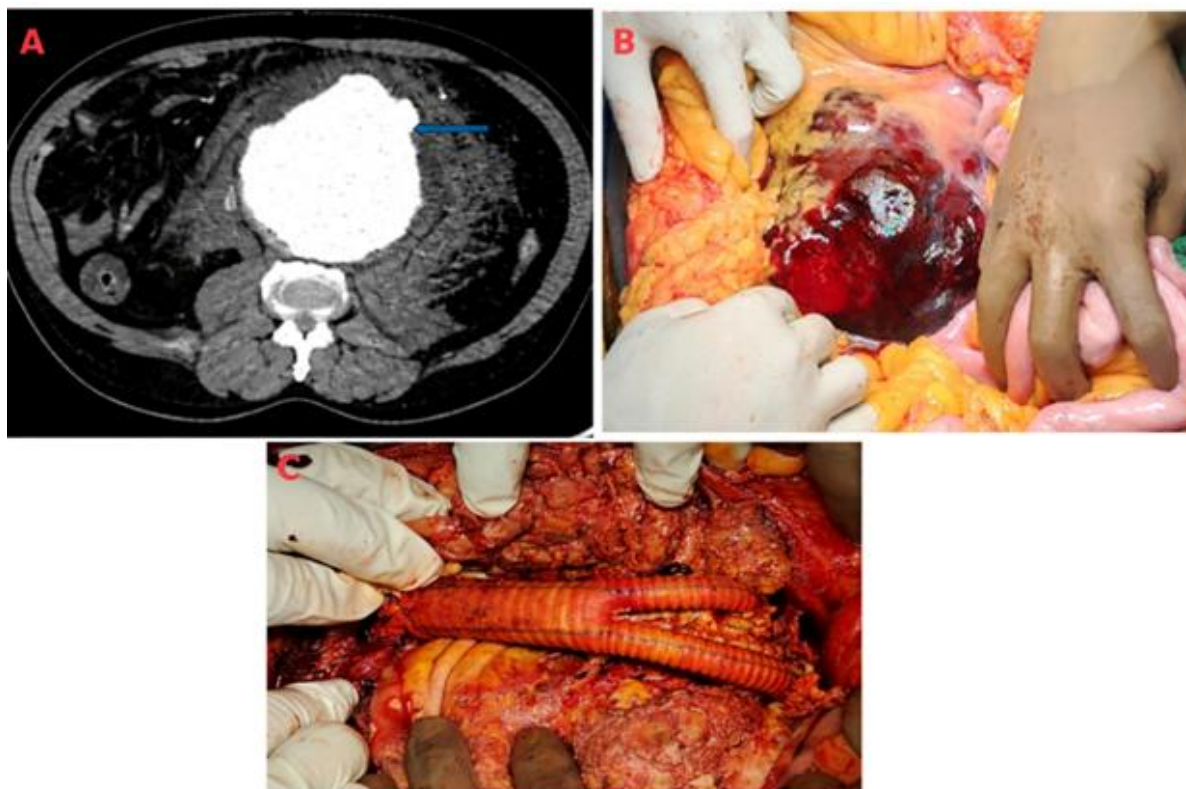


Figure 1. A: CT angiogram showing an infrarenal abdominal aortic aneurysm measuring 10×7 cm (blue arrow), B: intraoperative visualization of posterior wall rupture, and C: surgical repair with placement of a Dacron bifurcated graft (16×8 mm).

In the Operating Room (OR), an Invasive left brachial arterial line (peripheral pulses feeble) and a left Internal jugular vein (IJV) central line (Right external jugular vein venous access was started in the ED) were placed for an OSR. Aortic cross-clamping caused hypertension, managed with glyceryl trinitrate (GTN). Acidosis and reperfusion injury were treated with sodium bicarbonate and mannitol. Tranexamic acid infusion was started to reduce bleeding. Estimated blood loss was 4,500 mL with massive transfusion. Postoperatively, he was shifted and mechanically ventilated in the ICU, extubated after 48 hours, and discharged stable on postoperative day 10.

Case 2 – Open rAAA Repair with Axillobifemoral Bypass

An elderly man in his sixties presented with sudden abdominal and back pain along with fever. His examination revealed a pulsatile abdominal mass measuring 7×7 cm with absent distal pulses. In the ED, he experienced syncope and hypotension and was started on norepinephrine. Due to hemodynamic instability and suspected rupture, emergency laparotomy and aneurysm repair with axillobifemoral bypass were undertaken. The decision to proceed with axillobifemoral bypass was made in view of an infected mycotic aneurysm. In the operating theatre, he was intubated with RSI with a MAP of 80 mmHg using a video laryngoscope. An invasive right radial arterial line and a right IJV central line were

placed, along with advanced monitoring of FloTrac, in view of LV dysfunction with RWMA.

A 9 × 8.5 cm infrarenal aneurysm with posterior rupture and 2,000 mL hemoperitoneum was found. Stepwise supraceliac followed by infrarenal clamping-induced hypertension was managed with GTN. To mitigate acidosis and reperfusion injury, sodium bicarbonate infusion was given. The estimated blood loss was 7500 ml, which was replaced with albumin and blood products. He was shifted to the ICU and ventilated mechanically for 24 hours and discharged on day 8.

Case 3 – Open ruptured infrarenal AAA repair

An elderly man in his sixties presented with abdominal pain radiating to the back for 10 days. Examination revealed a 10 × 8 cm pulsatile mass in the abdomen, and a CTA scan confirmed a ruptured abdominal aortic aneurysm. Upon arrival at the operating theatre, his SBP was 90 mmHg, and he was intubated by RSI. A right radial arterial line and right IJV were inserted under local anesthesia before induction. Advanced FloTrac cardiac output monitoring was connected. During surgery, a 14 × 10 cm ruptured infrarenal aneurysm was discovered. Stepwise supraceliac then infrarenal clamping was performed. Clamp-induced hypertension was managed with GTN, while hypotension from declamping was treated with Norepinephrine and vasopressin. To reduce reperfusion injury and acidosis, sodium bicarbonate was administered. Approximately 3500 ml of blood loss was replaced with albumin and blood products, with the cell saver recovering about 1650 ml of blood. Postoperatively, he was transferred to the ICU, mechanically

ventilated for 24 hours, and moved to the ward on day 5 and was discharged on day 15.

Case 4 – Ruptured Thoracoabdominal Aneurysm Managed with TEVAR [thoracic endovascular aortic repair]

A female in her fifties presented with severe abdominal pain, fever, and vomiting. Her CTA showed a descending thoracic aortic aneurysm at the T12 level in a size of 4 x5 cm with contained rupture planned for TEVAR. In OR, TEVAR was done under monitored anesthesia care with an invasive arterial line. Intraoperatively, there was no hemodynamic instability, and the patient was not on any vasopressors. Post-procedure aortogram showed no endoleak. She was shifted to HDU for monitoring and discharged on day 5.

Case 5 – Ruptured Thoracoabdominal Aneurysm Managed with TEVAR

An elderly man in his fifties presented with back pain, fever, and features of hemorrhagic shock of SBP 90 mmHg and respiratory rate of 40/min with decreased left lung air entry to the ED. He was rushed to the theater after CTA, where a thoracoabdominal aortic aneurysm type 5 was intubated by RSI, and the right radial artery and right IJV were inserted and started on vasopressors. After TEVAR, his right lower limb pulses were absent, and he was immediately taken up for right femoral artery thrombectomy and left Intercostal drain [ICD] placement for hemothorax. He lost around 300 mL of blood and has received one unit of packed cell transfusion. He was transferred to the ICU for mechanical ventilation, extubated after 48 hours, and discharged on the 3rd week.

Discussion

Ruptured abdominal aortic aneurysm (rAAA) is defined as rupture of the AAA with bleeding from outside the true aortic wall with an intra- or retroperitoneal hematoma. Ruptured aortic aneurysms (RAA) are associated with high prehospital and in-hospital mortality. The key challenge in reducing mortality is diagnosing rAAA early in the ED/ward and shifting to the interventional place. Earlier, rAAA was diagnosed with STS (standard triad signs), including abdominal pain, hypotension, and a pulsatile mass. The classical triad is absent in 70% of rAAA due to the absence of palpable pulsatile mass and unreliable atypical symptoms. The newer score, MARS [Modified Abdominal Aortic Rupture Signs], was introduced, which encompasses 1) the registered pain-associated symptoms or

signs, 2) all hypovolemic-associated signs, and 3) pulsatile abdominal mass and/or ultrasound finding[4]. Risk stratification is crucial for predicting outcomes and making decisions for patients with ruptured aortic aneurysms. The Hardman Index and the Glasgow Aneurysm Score (GAS) are among the most used tools for estimating mortality risk associated with a given procedure. The Hardman Index considers five variables: age >76 years, loss of consciousness, hemoglobin <9 g/dL, serum creatinine >190 µmol/L, and ischemic changes on ECG, while GAS evaluates age, shock, myocardial disease, cerebrovascular disease, and renal disease. In our case series, we noted that GAS scores were low and Hardman Index scores ranged from 0 to 1 (Table 1), which may have contributed to the good outcomes given the emergency nature of the situation.

Table 1. Demographic, clinical, and perioperative characteristics of patients with ruptured aortic aneurysm

Variable	Case 1	Case 2	Case 3	Case 4	Case 5
Age (years)	69	63	65	59	51
Smoking status	Yes	Yes	Yes	No	No
Hardman index score	1	1	1	0	0
Glasgow Aneurysm Score (GAS)	86	72	79	59	68
Aneurysm size (cm)	10 × 7	9 × 8.5	14 × 10	4 × 5	8.5 × 6
Haemoglobin (g/dL)	13.8	9.6	9.3	10.8	13.6
Creatinine (mg/dL)	1.29	1.08	2.02	0.43	1.01
Initial arterial pH	7.24	7.40	7.31	NA	7.38
Base deficit (mEq/L)	-11.1	-3.9	-8.7	NA	-6.6
Initial lactate (mmol/L)	8.7	1.1	1.6	NA	2.6
Advanced monitoring	FloTrac	FloTrac	FloTrac	None	NIRS
Aortic clamp site	Supraceliac	Supraceliac	Supraceliac	None	None
Tranexamic acid used	Yes	Yes	Yes	No	No
Packed red blood cells (units)	6	9	4	None	1
Estimated blood loss (mL)	4500	7500	3500	150	300
Fresh frozen plasma / Cryoprecipitate (units)	4 / 3	10 / 16	0	None	None

Lowest intraoperative pH	7.12	7.40	7.17	NA	7.37
Peak intraoperative lactate (mmol/L)	7.1	8.7	5.7	NA	2.6
Outcome	Good	Good	Good	Good	Good

The potential for rupture is related to the aneurysm's size. The aneurysm of size 5 – 6 cm has a 3-15% risk of rupture, whereas the 6-7 cm aneurysm has a 10 – 20%, and the 7-8 cm aneurysm has a 20-40% risk of rupture, and more than 8 cm has a 50% risk of rupture [5]. In our series, all patients have an aneurysm >7 cm, except case 4, which had a smaller aneurysm with contained rupture, indicating that rupture does not depend solely on aneurysm size. The aneurysm diameter threshold for elective repair is 55 mm in men and 50 mm in women. To consider elective repair, an ultrasonogram was the initial choice to determine the diameter, whereas CTA is used for treatment planning. In an emergency setting, a patient with hemodynamically stable conditions should consider EVAR as the best option, even for a hemodynamically unstable patient; if possible, so the patient can undergo CTA and EVAR as per the 2024 European guidelines [6]. In a meta-analysis study, the patients with hemodynamically unstable conditions who underwent EVAR have an in-hospital mortality of 37%, whereas for OSR, it is 62% [7]. The IMPROVE trial supports an endovascular-first strategy in ruptured AAA, demonstrating comparable mortality with potential advantages in recovery and discharge outcomes [8].

Often, OSR requires general anesthesia for a ruptured aortic aneurysm, whereas EVAR can be performed under local anesthesia in a hemodynamically stable patient. The intervention for the RAA depends on multiple factors,

including age, comorbidities, suitability of the aortic anatomy, infected versus non-infected aorta, the center's expertise, the patient's hemodynamic status, and the cost of the procedure. For RAA, the more invasive the procedure, the greater the need for advanced monitoring, such as invasive lines, cardiac output monitoring, and TEG/ROTEM. Multiple studies have shown that incorporating viscoelastic assays into RAA management is a powerful tool to guide safe transfusion, as it reflects both hypercoagulability and fibrinolysis, thereby reducing unnecessary transfusions that can further increase morbidity and mortality [9]. We used FloTrac for real-time monitoring in 3 cases, TEG/ROTEM to guide transfusion in 4 cases, and level 1 and 2 warming devices for open repair cases and to maintain normothermia. In EVAR, warming must be limited to the upper body; the lower limbs must be spared due to increased burn risk, as there is no circulation during graft deployment.

There is substantial evidence that permissive hypotension is advantageous for RAA outcomes by allowing stable clot formation, preventing blown-out clots, decreasing bleeding, and preventing coagulopathy, whereas normotensive resuscitation might exacerbate bleeding and compromise outcomes. The target systolic BP for RAA is 70-90 mmHg until the surgeon achieves proximal aortic control [10]. The ROSE concept defines the Resuscitation phase in the ED as restrictive rather than aggressive fluid management to maintain higher SBP. The Optimization

phase occurs in the OR, where optimization should be with blood and blood products rather than crystalloids, in a ratio of FFP: Blood 1:1. The stabilization phase starts once the repair has been completed, by avoiding excessive fluid administration and giving only maintenance fluids. The evacuation phase is the de-escalation phase, during which excess fluids are removed from the body [11]. The ESVS [European Society for Vascular Surgery] has recommended the intraoperative use of cell salvage to reduce intraoperative bleeding and the need for allogenic transfusion. Our cases have emphasized this permissive hypotension, maintaining systolic BP at 70-90 mmHg while preserving end-organ perfusion and restricting aggressive fluid management through early use of blood and blood products, thereby mitigating the lethal triad of acidosis, hypothermia, and coagulopathy by incorporating the ROSE concept. We have used cell salvage [1690 ml blood] in our case 3.

Aortic aneurysm during repair often requires cross-clamping. The level and duration of aortic cross-clamping determine the extent of postoperative complications, driven by hypoperfusion followed by ischemia-reperfusion injury. Cross-clamping increases preload proximal to the clamp and cuts off the blood supply distal to it. At which level to be cross-clamped is often decided by the extent of the aneurysm and the diseased aortic wall. Suprarenal and supraceliac aortic cross-clamping will increase the workload to the heart and cause proximal arterial hypertension, which has to be treated with vasodilators like sodium nitroprusside or glyceryl trinitrate. Once the clamp has been released, a declamping shock will occur due to mediators' release, which must be treated with vasopressors, inotropes, and sodium bicarbonate. Both

clamping and declamping should be performed stepwise to avoid severe arterial hypertension and sudden bursts of massive mediators [6,12]. In all 3 OSRs, cross-clamping at the supraceliac level is associated with greater hemodynamic instability during clamping and declamping, which was proactively managed with pharmacological agents and advanced monitoring. In case 5, an ICD for hemothorax was placed after TEVAR. Intercostal drainage is typically deferred until after endovascular exclusion of the rupture, as premature pleural decompression may abolish any potential containment effect and precipitate uncontrolled hemorrhage.

Aortic aneurysm repair can lead to a multitude of complications, such as myocardial infarction [MI], arrhythmia, Acute Kidney Injury and Acute Tubular Necrosis [ischemia and contrast dye], ACS (abdominal compartment syndrome), including paralytic ileus, bowel ischemia and perforation, and rarely lower limb ischemia. Lower limb pulses have to be verified in all cases of aortic repair [13]. None of our cases had MI and colonic ischemia, and 3 of our OSR cases had AKI, which resolved before discharge. In case 5, the patient had an absent right lower limb pulse and underwent immediate femoral thrombectomy. EVAR patients were discharged in the 1st week, whereas the first 2 OSRs were discharged in the 2nd week, and the 3rd was discharged in the 3rd week. These differences should be viewed with caution, as the decision between EVAR and open surgical repair was influenced by factors such as anatomical suitability, hemodynamic status, comorbidities, and institutional expertise, which may introduce selection bias.

Conclusion

To manage a ruptured aortic aneurysm effectively, a rapid multidisciplinary, integrated action is important. Timely intervention, ideally within 90 minutes within a 30/30/30 framework for diagnosis, transfer, anesthesia preparation, and procedural intervention, is crucial for improving outcomes. The perioperative management of these complex cases was guided by the patient's hemodynamic stability, comorbidities, and planned intervention. The open approach may have a longer postoperative stay than EVAR. In rAAA cases, better outcomes result from regionalization to high-volume centers of excellence that offer both surgical and endovascular options, thereby reducing morbidity and mortality.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

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Informed Consent

Informed consent was obtained from the patient's next of kin, and anonymity will be maintained.

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