

Anesthetic Management for Abdominal Aortic Aneurysm Rupture

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ABSTRACT

Background: Aortic aneurysm is the thirteenth leading cause of death in the United States. Around 15,000 people died each year because of aortic aneurysm rupture. The mortality rate for this case is still high at around 90%. Patient diagnosed with acute aortic rupture will need an urgent surgery.

Case: A 36-year-old man came with complaints of pain in the waist to back area since 1 week before entering the hospital and worsening 2 days before entering the hospital. Pain is felt intermittent. The pain is sharp and severe when the patient strains. On physical examination found high blood pressure (160/104 mmHg), chest X-ray showed widening of the mediastinum suspected descending aortic aneurysm, CT angiography showed a fusiform type descending aortic aneurysm pars thoracolumbar, ruptured abdominal aortic aneurysm of juxtarenal fusiform type to the terminal abdominal aorta and hemoperitoneum. The patient underwent repair of an abdominal aortic aneurysm under general anesthesia. After surgery the patient was admitted to the ICU for clinical and hemodynamic monitoring, but the patient's condition in the ICU worsened. The patient experienced acute kidney failure and liver failure, then the patient died on the 10th day after surgery.

Discussion: Surgery for ruptured abdominal aortic aneurysm is associated with high mortality. Even patients who survive the initial procedure are at high risk of complications (such as renal, cardiac, respiratory, hematological, or gastrointestinal failure). The main goal of anesthesia is to maintain anesthesia with cardiovascular stability and normothermia for as long as possible. Minimum standards of monitoring for surgery include electrocardiogram, CVP, arterial line, temperature, and urine output. This operation uses a cell saver machine which functions to collect lost blood, clean the blood and return it to the patient.

Conclusion: Surgery for patients with acute aortic rupture requires complicated and complex anesthetic techniques. This operation requires collaboration and good communication between the surgeon and the anesthesiologist.

Keywords: abdominal aorta; aneurysm; cell saver machine; general anesthesia; mediastinum

INTRODUCTION

Aortic aneurysm ranks as the 13th most common cause of death in the United States. About 15,000 people die each year due to aortic aneurysm rupture. In research, it was concluded that 1–2% of the population had signs of an aneurysm in the aorta, but it was not detected until rupture occurred, and the death rate from aortic aneurysm rupture was 90%.¹

The prevalence of abdominal aortic aneurysms located in the infra-renal area is three times greater than that of aortic aneurysms located in the thorax. Thoracic aortic aneurysms occur in 50% of the ascending aorta, 10% in the aortic arch, and 40% in the descending aorta. Twenty-five percent of patients who experience a thoracic aortic aneurysm also experience an abdominal aortic aneurysm.¹

Abdominal aortic aneurysm (AAA) occurs generally in male smokers over 65 years of age. Chronic smoking is the single most important risk factor for both the development and progression of AAA. The prevalence of AAA (aortic diameter >30 mm) in chronic smokers is four times greater than in non-smokers, and the average rate of aneurysm growth in smokers is 2.8 mm per year versus 2.5 mm per year in non-smokers. The most common cause of AAA is atherosclerosis; rare causes include Marfan syndrome, salmonella, brucellosis, tuberculosis, and Takayasu disease.²

AAA is usually asymptomatic but progresses over time, and the risk of spontaneous rupture increases. Morbidity and mortality rates are lower after elective surgery than after emergency surgery. So, a community early detection program is being intensively launched. The Multicentre

Aneurysm Screening Study proved that there was a 53% reduction in mortality in British men aged 65–74 who were examined by regular ultrasound screening and underwent elective surgery when the aneurysm diameter reached 5.5 cm. In contrast, the UK Small Aneurysm Trial recognized no benefit in elective surgery for very small AAAs (<5.5 cm diameter).²

Surgical procedures in patients with acute aortic rupture require complicated and complex anesthetic techniques to maintain other organ systems during the operation. This operation requires collaboration and good communication between the surgeon and the anesthetist. Thus, we submit a case report regarding anesthetic management in abdominal aortic aneurysm repair surgery.

CASE

A 36-year-old male patient, weight 60 kg and height 165 cm, came to the emergency room at Sardjito Hospital with complaints of pain in the waist to back area since one week before admission to the hospital, the patient has been experiencing intermittent sharp pain. The pain worsens when the patient pushes. Previously, the patient had visited Purbowangi Gombong Hospital and was admitted for 2 days, receiving analgesic medications. The pain improved, and the patient was discharged. Three days before entering the hospital, the patient complained of hip and back pain, but the pain felt worse. The patient went to PKU Gombong Hospital and had an abdominal ultrasound examination, and it was said that there was dilation of the aorta and that he was referred to Sardjito Hospital.

The patient has had a history of high blood pressure since 1 year ago, with the highest systolic blood pressure of 200 mmHg and an average systolic blood pressure of 150 mmHg. He has received 10 mg of amlodipine therapy, but the patient does not take medication regularly. The patient has had a history of smoking since the age of 17. The history of other diseases such as diabetes mellitus, heart disease, stroke, and liver dysfunction was stated to have never been known because they were not routinely checked. The history of drug allergies and alcohol consumption was denied.

A physical examination was carried out on July 26, 2022, and it found complete consciousness with a blood pressure of 160/104 mmHg, a heart rate of 102 x/minute, a respiratory rate of 20 times/minute, an axillary temperature of 36.5 °C, an oxygen saturation of 97% in room air, and a VAS scale of 8. From the head examination, it was found that the pupil isochore was 2mm/2mm with a light reflex (+/+), and anemic conjunctiva was found. On examination of the neck, there were no lesions, and the JVP was not increased. On chest examination, both inspection, palpation,

percussion, and auscultation were within normal limits.

On inspection of the abdomen, it was found that the epigastric area of the abdomen was pulsing according to the heart rate, and there was no distension or injury to the abdomen. On auscultation, bowel sounds were normal; tympanic percussion and abdominal palpation revealed a supple abdomen; tenderness in the epigastric area; no muscular defecations; and no palpable thrill. On examination of the extremities, the acral was warm, the pulse was strong, and the capillary refill time was <2 seconds.

In the emergency room, the patient received amlodipine 1x10 mg therapy with a target blood pressure of <140/90 mmHg, ramipril 1x5 mg, bisoprolol 1x2.5 mg with a target HR <60 times per minute, atorvastatin 1x40 mg, ranitidine injection 50 mg/12 hours intravenously, and ketorolac injection 30 mg/8 hours intravenously. A chest X-ray (Figure 1) examination was carried out, with the conclusion that the pulmonary system was normal, the widening of the mediastinum was suspicious for a descending aortic aneurysm, and the cor size was normal.

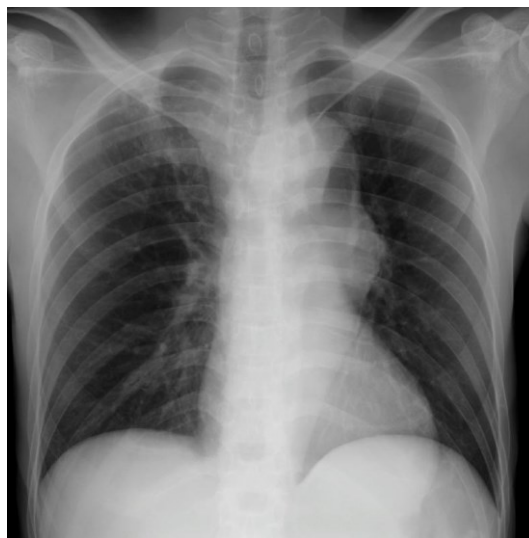


Figure 1. Chest X-ray

An ECG examination was carried out with results of sinus rhythm, HR 98 x/min, normoaxis. A CT angiography examination of the thoracic and abdominal aorta (Figure 2) was carried out and showed the following impression: fusiform descending aortic

aneurysm, rupture of the juxtarenal fusiform abdominal aortic aneurysm to the terminal abdominal aorta, with hematoma around it, and hemoperitoneum were found.

A complete blood count revealed:

Table 1. Laboratory Result

Parametric	Result	Unit	Normal Value
Haemoglobin	7.5	g/dl	12 – 15
Erythrocyte	2.95	million/ μ L	4.00 – 5.40
Hematocrit	24	%	35 - 49
MCH	25.4	pg	26.0 – 32.0
MCHC	31.3	g/dL	32.0 – 36.0
Diff count leukocyte			
Neutrofil	80.0	%	50.0 – 70.0
Limfosit	14.5	%	18.0 – 42.0
Eosinofil	0.2	%	1 - 3
Basofil	0.0	%	0.0 – 2.0
PPT	14.8	sec	9.4 – 12.5
INR	1.35		0.9 – 1.1
PPT control	11.00	sec	
APTT	31.9	sec	25.1 – 36.5
APTT control	31.20	sec	
Albumin	2.20	g/dL	3.97 – 4.94
Sodium	133	mmol/L	136 - 145

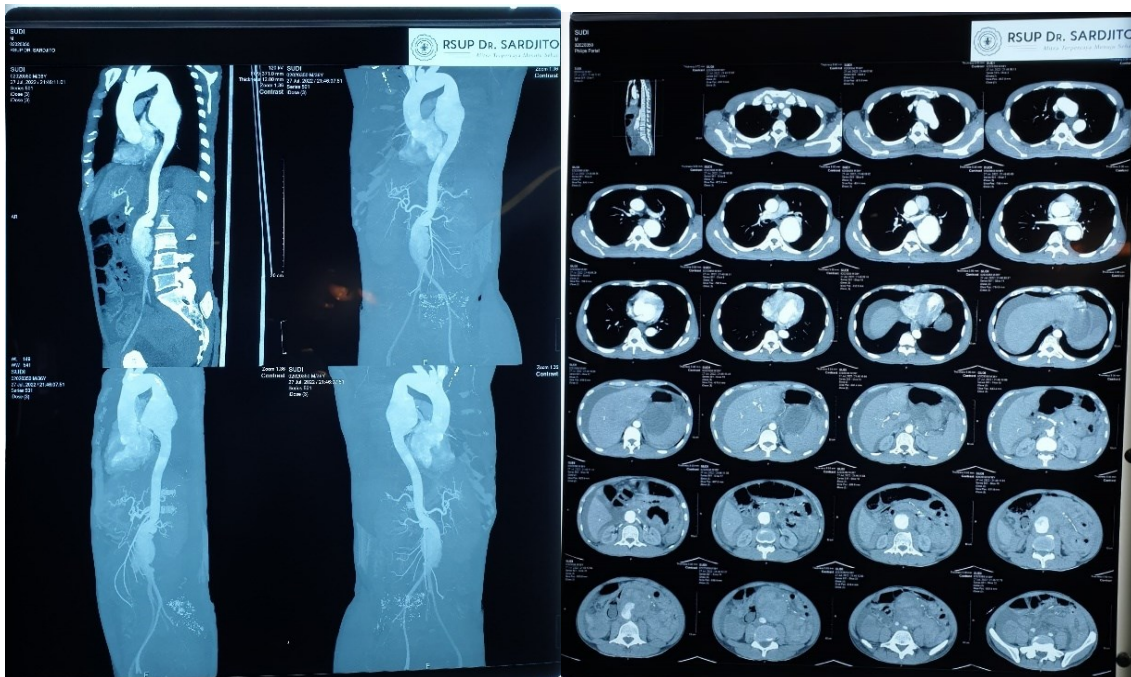


Figure 2. CT angiography

Anesthesia Management

The patient entered the operating room with a stable hemodynamic condition, blood pressure 130/80 mmHg, sinus rhythmic ECG, HR 98x/m, SpO₂ 97%, and nasal cannula oxygen 3 liters/minute. A peripheral infusion was installed using an 18G IV catheter in the dorsum of the right and left manus, and an arterial line was installed in the right radial artery using a 20G IV catheter. Then the patient was induced with the intravenous anesthetic drug midazolam (5 mg), followed by propofol (30 mg), fentanyl 200 mg, and muscle relaxants using rocuronium 50 mg. The airway was controlled with a mask, then the patient was intubated with ETT no. 7.5, depth 20 cm, and for respiratory function, a ventilator is used with volume control mode FiO₂ 50%, RR: 14 x/m, tidal volume 450 ml, I:E ratio 1:2. After that, a central venous catheter was placed in the left subclavian vein using a 7-Fr central venous catheter, a side port was installed in the right internal jugular vein using an 8-Fr venous catheter, and a second arterial line was placed in the right femoral artery. Then a temperature probe is installed in the nasopharyngeal cavity.

Maintenance of anesthesia is achieved using:

Maintenance: O₂-Water = 50: 50, 1 liter/minute + Sevoflurane 2 vol%, continuous rocuronium 5 mcg/kgbb/minute, and continuous fentanyl 5 mcg/kgbb. The patient received the vasopressor drug norepinephrine 0.05–0.2 mcg/kg/minute and the inotropic dobutamine 5–10 mcg/kg/minute. The fluid balance obtained was: Ringer Lactate 1000 ml, Gelafusal 2000 ml, Ringerfundin 500 ml, Thrombocyte Concentrate 459 ml, Fresh Frozen Plasma 1359 ml, Packed Red Cell 2268 ml, and Cell Saver 950 ml,

with a total input of 8,536 ml. Obtained bleeding of 7500 ml and urine of 850 ml with a total output of 8,350 ml, so fluid balance: 186 ml.

After the induction of anesthesia is complete, the hemodynamic condition is relatively stable, with a pulse ranging from 60–80 x/minute, systolic blood pressure ranging from 90–120 mmHg, diastolic blood pressure ranging from 40–60 mmHg, MAP 55–80 mmHg, and CVP 6–8 mmHg. The surgery was carried out for 7 hours by making an incision in the chest cavity to reduce intrathoracic pressure, followed by an incision in the abdomen. Heparin was given at 150 IU/kgbb, then aortic cross-clamping was performed, and abdominal aortic aneurysm repair began. After the abdominal aortic aneurysm repair is complete, the cross-clamp is removed. Severe hypotension occurs but can be overcome by providing volume to the circulation and the use of vasoconstrictors and inotropes.

After the surgical procedure was completed, the hemodynamic condition, pulse ranged from 101 x/minute, systolic blood pressure ranged from 80–100 mmHg, and diastolic blood pressure ranged from 40–50 mmHg, with the support of norepinephrine 0.2 mcg/kg/minute and dobutamine 10 mcg/kg/minute. The patient was transferred to the ICU and treated in the ICU for post-surgical treatment, but the patient's condition in the ICU worsened; urine production on the second post-surgical treatment day was only less than 0.5 ml/kgBW/hour, and the patient's serum creatinine increased by more than 0.5 g/hour. dL, so it was decided to do continuous venous hemodialysis. The patient's condition continued to worsen, accompanied by an increase in SGOT-SSGPT of three times the value before

surgery. The patient experienced acute kidney failure and liver failure, then died on the 10th day after surgery.

DISCUSSION

A 36-year-old male patient, weight 60 kg and height 165 cm, came to the emergency room at Sardjito Hospital with complaints of pain in the waist to back area since one week before admission to the hospital, the patient has been experiencing intermittent sharp pain. The pain worsens when the patient pushes. Three days before entering the hospital, the patient complained of hip and back pain, but the pain felt worse. In abdominal ultrasound it was said that there was dilation of the aorta and that he was referred to Sardjito Hospital.

The patient has had a history of high blood pressure since 1 year ago, with the highest systolic blood pressure of 200 mmHg and an average systolic blood pressure of 150 mmHg. He has received 10 mg of amlodipine therapy, but the patient does not take medication regularly. The patient has had a history of smoking since the age of 17. The history of other diseases such as diabetes mellitus, heart disease, stroke, and liver dysfunction was stated to have never been known because they were not routinely checked. The history of drug allergies and alcohol consumption was denied.

The development of AAA results from changes in the connective tissue of the aortic wall. Elastin and collagen fibers provide the majority of the tensile strength of the aortic wall. Degradation of elastin fibers seems to be an early feature of aneurysm formation, while collagen disruption is the main cause of rupture. Normal collagen/elastin homeostasis is maintained by a fine balance between matrix

metalloproteinases (MMP) and their tissue inhibitors; if disturbed, proteolysis occurs, causing aneurysm formation. Other factors involved in aneurysm formation include chronic inflammatory infiltrate, smooth muscle cell apoptosis, and increased production of proinflammatory cytokines.³

AAA rupture is a surgical emergency and prompt preoperative evaluation is required. There are some situations when surgery is not appropriate, such as in patients who have experienced cardiac arrest or patients with terminal illnesses. In the past, patients with severe cardiorespiratory disease could be denied elective surgery, but with the increasing availability of endovascular techniques, many of these patients can undergo surgery.⁴

Based on physical examination, the patient found in a complete consciousness with a blood pressure of 160/104 mmHg, a heart rate of 102 x/minute, a respiratory rate of 20 times/minute, an axillary temperature of 36.5 °C, an oxygen saturation of 97% in room air, and a VAS scale of 8. From the head examination, it was found that the pupil isochore was 2mm/2mm with a light reflex (+/+), and anemic conjunctiva.

On inspection of the abdomen, it was found that the epigastric area of the abdomen was pulsing according to the heart rate, and there was no distension or injury to the abdomen. On auscultation, bowel sounds were normal; tympanic percussion and abdominal palpation revealed a supple abdomen; tenderness in the epigastric area; no muscular defecations; and no palpable thrill. On examination of the extremities, the acral was warm, the pulse was strong, and the capillary refill time was <2 seconds.

Many patients have significant coronary artery disease that is not always apparent on history and examination. Diabetes, hypertension, and renal disorders are also common. Blood pressure should be checked noninvasively in both arms because brachiocephalic and subclavian artery stenosis may be present. If there is a difference in readings, the higher reading should be used. If this occurs, the second anesthetist should organize the preparation of equipment and theater drugs (OK room), ensuring an adequate supply of blood and coagulation products. We recommend a minimum of 10 units of red blood cells, as well as platelets, fresh frozen plasma, and cryoprecipitate. Hospitals should have systems in place to prepare these blood products without delay (such as not waiting for laboratory coagulation results); close testing of the patient may also have a role.⁴

A chest X-ray examination was carried out, with the conclusion that the pulmonary system was normal, the widening of the mediastinum was suspicious for a descending aortic aneurysm, and the cor size was normal. A CT angiography examination of the thoracic and abdominal aorta was carried out and showed the following impression: fusiform descending aortic aneurysm, rupture of the juxtarenal fusiform abdominal aortic aneurysm to the terminal abdominal aorta, with hematoma around it, and hemoperitoneum were found.

The first response of many anesthetists faced with a ruptured AAA patient is to administer intravenous fluids quickly to restore blood pressure to near-normal values. However, administering excessive fluids before clamping the aorta will increase bleeding through detachment of the thrombus and dilution

of clotting factors. It is reasonable to avoid any kind of red blood cell transfusion before surgery unless the patient is unconscious or showing signs of cardiac ischemia. If pain is severe, a small amount of additional intravenous morphine may be given while surgical plans are being made. It may be considered to place an epidural catheter before surgery in patients with plasma leakage with satisfactory coagulation results and who are hemodynamically stable. The advantage of this is that epidural analgesia may facilitate common postoperative coagulopathy weaning and may contraindicate epidural insertion within 48–72 hours.⁴

Induction of anesthesia in patients with ruptured AAA may be associated with cardiovascular failure due to: (1) cardiodepressant effects of intravenous and inhalation agents; (2) relaxation of the abdominal muscles, thereby reducing the tamponade effect; (3) intermittent positive pressure ventilation, which reduces venous return; and (4) reduced sympathetic tone. Therefore, induction of anesthesia should be performed with the patient on the operating table, fully prepared for surgery, with blood for transfusion readily available.⁵

In this case, cardiac function of the patient was normal based on ECG and X-ray. Then the patient was induced with the intravenous anesthetic drug midazolam (5 mg), followed by propofol (30 mg), fentanyl 200 mg, and muscle relaxants using rocuronium 50 mg. The airway was controlled with a mask, then the patient was intubated with ETT no. 7.5, depth 20 cm, and for respiratory function, a ventilator is used with volume control mode FiO₂ 50%, RR: 14 x/m, tidal volume 450 ml, I:E ratio 1:2. After induction, hemodynamic status was stable.

Anesthesia is usually maintained with a balance of techniques using volatile agents, opioids, and neuromuscular blockade. Nitrous oxide will reduce arterial pressure in patients who have reduced cardiac contractility or increased sympathoadrenal activity, both of which are frequently found in patients with AAA rupture. For this reason, some anesthetists avoid its use. High doses of opioids (such as fentanyl, 5–20 µg/kg) are often used.⁵

In this case, maintenance of anesthesia is achieved using: O₂-Water = 50: 50, 1 liter/minute + Sevoflurane 2 vol%, continuous rocuronium 5 mcg/kgbb/minute, and continuous fentanyl 5 mcg/kgbb. The patient received the vasopressor drug norepinephrine 0.05–0.2 mcg/kg/minute and the inotropic dobutamine 5–10 mcg/kg/minute. The fluid balance obtained was: Ringer Lactate 1000 ml, Gelafusal 2000 ml, Ringerfundin 500 ml, Thrombocyte Concentrate 459 ml, Fresh Frozen Plasma 1359 ml, Packed Red Cell 2268 ml, and Cell Saver 950 ml, with a total input of 8,536 ml. Obtained bleeding of 7500 ml and urine of 850 ml with a total output of 8,350 ml, so fluid balance: 186 ml.

In the absence of suitable blood, group O or specific blood can be used. Large volumes of intravenous fluids may be required rapidly; therefore, heating equipment should be installed with suitable fluids and/or blood, if possible, using a rapid infusion device. Direct arterial pressure monitoring should be performed before induction of anesthesia, but central venous access can be deferred at this time unless no venous access is available.⁵

No specific technique or anesthetic agent has been shown to significantly improve outcomes; the primary goal is to maintain anesthesia with cardiovascular stability and normothermia for as long as possible. A modified rapid induction sequence is required using careful dose titration of the induction agent, followed by succinylcholine. In an attempt to reduce the required dose of the induction agent, opioids (such as fentanyl and alfentanil) may be used.⁵

The physiologic response to aortic cross-clamping depends on several variables, including preoperative left ventricular function, collateral circulation, and level of cross-clamp. When the aorta is cross-clamped, increased afterload can cause hypertension in the area proximal to the clamp. This can be reduced by increasing the depth of anesthesia or administering vasodilators (such as GTN). This also provides an intravascular fluid influx in preparation for clamp removal.⁶

Restoration of circulation on cross-clamp release is accompanied by a sudden decrease in afterload and severe ischemia-reperfusion injury. This can cause severe hypotension, lactic acidemia, myocardial ischemia, and cardiovascular collapse. This can be reduced by maintaining mean arterial pressure and increasing circulating volume, which is facilitated by the administration of vasodilators during cross-clamp application. However, hypotension normally occurs, and vasoconstrictors and/or inotropic drugs are usually required.⁶

The minimum recommended monitoring standards for AAA rupture repair include ECG (CM 5 configuration), CVP, arterial line, body temperature, and urine output. Pulmonary-artery flotation catheters are rarely used. Each hospital should have a protocol for administering blood products in these cases because it is impractical to wait for coagulation test results before requesting them. Because of the limitations of standard coagulation tests, many centers are assessing the role of thromboelastography (TEG) in emergency vascular surgery. TEG reliably shows hypercoagulability and fibrinolysis, both of which are often considered low by conventional coagulation tests; it is potentially useful in any situation where there is a rapid change in hemostatic profile and is widely used to determine transfusion practices for liver and cardiac surgery. Perioperative hypothermia occurs frequently due to the open abdomen, patient exposure, blood loss, and the large volume of intravascular fluid transfused. Every effort should be made to maintain the patient's temperature during surgery by using warming bags and warm fluids.^{7,8}

After the surgical procedure was completed, the hemodynamic condition, pulse ranged from 101 x/minute, systolic blood pressure ranged from 80–100 mmHg, and diastolic blood pressure ranged from 40–50 mmHg, with the support of norepinephrine 0.2 mcg/kg/minute and dobutamine 10 mcg/kg/minute.

The patient was transferred to the ICU and treated in the ICU for post-surgical treatment, but the patient's condition in the ICU worsened; urine production on the second post-surgical treatment day was only less than 0.5 ml/kgBW/hour, and the patient's serum creatinine

increased by more than 0.5 g/hour. dL, so it was decided to do continuous venous hemodialysis. The patient's condition continued to worsen, accompanied by an increase in SGOT-SSGPT of three times the value before surgery. The patient experienced acute kidney failure and liver failure, then died on the 10th day after surgery.

Patients are at risk of developing renal impairment due to: preoperative hypotension and hypovolemia; aortic clamping causing direct renal ischemia; a large embolic load; and postoperative blood loss. To prevent postoperative renal impairment, every effort should be made to maintain adequate perfusion pressure and limit the duration of suprarenal clamping. Many anesthetists administer drugs such as mannitol, furosemide, or dopamine to prevent kidney failure, but there is no convincing evidence that these improve outcomes. The main priority is to maintain adequate extracellular fluid volume both during and after surgery.⁹

All patients should be sent to the postoperative ICU, where there is supportive care, including optimization and maintenance of circulating volume. Rewarming will continue until a normal body temperature is achieved, and respiratory support is usually required for a minimum of 24 hours and often several days. Kidney function, coagulation, hemoglobin, and acid-base balance are closely monitored. Renal replacement therapy is required in a significant proportion of patients, and those with coagulopathy may require continuous blood product transfusions. Other important problems include analgesia and the anticipated prolonged ileus.¹⁰

There are several mechanisms that can lead to AKI after EVAR, including microembolization, suprarenal fixation, accessory renal artery occlusion, and CIN, as well as the inflammatory and ischemic responses associated with the intervention.¹⁶

Liver enzyme levels acutely increased in 1.5% of patients after elective infrarenal aortic aneurysm repair with infrarenal cross-clamping. In patients with moderately elevated serum liver enzyme levels, postoperative recovery was relatively uncomplicated, whereas all three patients with AIH developed acute renal failure and had a more complicated postoperative course. Those with postoperative liver dysfunction had a longer duration of intraoperative hypotension and more pronounced metabolic acidosis.¹⁷

CONCLUSION

Abdominal aortic aneurysms are usually asymptomatic but progress over time, and the risk of spontaneous rupture increases. Surgical procedures in patients with acute aortic rupture require complicated and complex anesthetic techniques to maintain other organ systems during the operation. This operation requires collaboration and good communication between the surgeon and the anesthetist. The anesthesia management and the reasons for postoperative complications in this case need to be further evaluated.

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