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Review Article

Haemodynamic management in ruptured abdominal aortic aneurysm

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Summary: Ruptured abdominal aortic aneurysm currently accounts for about 1 in 200 deaths and is a critical surgical emergency with an average hospital mortality of 50%. The combination of acute massive haemorrhage in an elderly patient with pre-existing medical disease is highly lethal and a major challenge for any health care system. This article outlines the general principles of management and discusses the problems of haemodynamic assessment and preclamping fluid resuscitation.

Introduction

The first abdominal aortic aneurysm was repaired in the mid-1950s, and operative mortality rates for elective repair have gradually fallen to approximately 3%.[1,2] However, survival from ruptured abdominal aortic aneurysm (RAAA) has shown no real improvement and remains at 50%.[3] More screening and elective surgery are now performed to reduce overall mortality, but this can not eliminate rupture altogether.

Isolated improvements in mortality have been attributed to earlier diagnosis, aggressive resuscitation and postoperative care, early transport to theatre, and involvement of specialist surgeons.[4] The construction of management guidelines for the health care team may therefore play an important role in optimizing the chances of survival from RAAA.

Some authors consider that no patient should be denied surgery as significant survival rates have been reported even in patients over 80 years old and those presenting with cardiac arrest,[5] but this may not represent the consensus view. Patients surviving RAAA by 2 months have mortality rates and quality of life comparable to those undergoing elective abdominal aortic aneurysm (AAA) repair,[6] life expectancy being related to the severity of associated diseases.

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Pathophysiology

A total of 50-60% of AAAs will rupture within 5 years, and the larger the aneurysm the more likely it is to rupture. Between 33 and 62% will die before they reach hospital. Clot formation, retroperitoneal tamponade, increased abdominal muscle tone and hypotension are thought to play a critical role in limiting haemorrhage. The site and severity of rupture varies. Eighty-eight per cent rupture into the retroperitoneal space and 12% into the peritoneal cavity.[7] Free rupture into the peritoneum carries a mortality of 60-97%.[8,9] Occasionally rupture can occur into the inferior vena cava[10] or the duodenum.[11] A total of 98% occur infra-renally.[7] Mortality is closely linked to a wide variety of factors[4,8,12-20] (Table I), notably the degree of hypotension on admission.

The normal compensatory response to haemorrhage may be attenuated in the patient with RAAA who is generally elderly and has pre-existing medical illness[8,12,15-17,19,21] (Table II). Widespread atheroma further promotes early failure of supply-dependent organs and cardiac complications account for over 50% of deaths. The duration of shock, extent of tissue hypoxia, premorbid state, and the ability to compensate by increasing oxygen flux, are major determinants of outcome in shock states.[22] Some retrospective studies show that patients who fail to increase blood pressure in response to fluids and inotropes have the highest mortality.[15,18]

Presentation and diagnosis

Patients may present with a broad range of symptoms and physiological states, and only 50% will

Table I Factors associated with increased mortality in RAAA*

Acute	*Premorbid*
Hypotension†	Increasing age[8,13,18] > 80 years†
Massive blood transfusion†	Ischaemic heart disease
Low haematocrit	
Myocardial impairment	
Cardiac arrest†	
Low urine output	
Delays in diagnosis	
Prolonged surgery	
Inadequate surgical experience	

*Derived from the following papers (refs. 4,8,12–20); †associated with mortality rates of > 75%.

Table II Incidence of co-existing disease in RAAA*

Coronary artery disease or cardiac failure	50%
Pre-existing hypertension	30–50%
Obstructive airways disease	30–40%
Chronic renal disease	5%
Cerebrovascular disease	6%

*Derived from the following papers (refs. 8, 12, 15–17, 19, 21).

present with the classical triad of abdominal or back pain, pulsatile mass and shock. The diagnosis can usually be made on clinical grounds,[13] but ultrasound is an important diagnostic tool and can confirm the presence of an aneurysm in 90% of patients within 5 minutes. Other diagnostic radiological techniques may occasionally be required such as computed tomographic (CT) scan and aortography. An electrocardiogram is useful to exclude myocardial infarction but other routine investigations do not influence management, cause unnecessary delays, and risk sudden deterioration even in the patient who is 'stable'. Early diagnosis is therefore important and may reduce mortality by 50%.

Haemodynamic assessment

Haemodynamic assessment in RAAA may be difficult for two reasons:

1. Obtaining accurate haemodynamic data is difficult in the shocked patient. Indirect blood pressure (BP) monitoring may be unreliable and insertion of an arterial line, essential for beat to beat BP monitoring, is not always technically possible, although a hand-held Doppler probe may be useful in measuring systolic pressure. The haematocrit is inaccurate as an assessment of acute blood loss as compensatory haemodilution has yet to occur.
2. The interpretation of any measured haemodynamic variables may be misleading. There is poor correlation between the degree of hypovolaemia, blood pressure and central venous pressure in haemorrhagic shock due to the variable effects of preload, afterload and stroke volume.[23] Blood gases and serum lactate may correlate better with the degree of shock. Hypotension may also be compounded by myocardial ischaemia, or vasoactive cardiac depressant factors released from hypoxic tissues.

Despite the problems of haemodynamic assessment, it is important to document data as any acute changes may act as a subsequent guide to fluid and inotrope therapy.

Early management

Ideally, the patient should be taken directly to theatre for investigation and management if RAAA is suspected. Management priorities are dictated chiefly by the clinical state of the patient at presentation. The systematic approach to management, as used in major trauma, may be preferable to the traditional approach of history, examination, diagnosis and treatment. This enables investigation and therapy to occur synchronously and reduces the likelihood of omission (Table III). Aggressive surgical management and prompt aortic clamping are the key management priorities.

Preoperative fluid resuscitation

There is some recent controversy over the efficacy of preclamping fluid resuscitation in RAAA. One view is that fluid resuscitation to restore blood pressure should be avoided since it may invite further haemorrhage leading to dilutional coagulopathy, massive blood transfusion and technical surgical difficulties.[4,13] The opposing view is that fluid resuscitation should be started immediately since the longer the period of shock the greater the risk of developing cardiac complications and multisystem organ failure.[18,24] There have been no prospective studies evaluating different preoperative fluid regimes in RAAA. There is evidence supporting both viewpoints. The case against fluid resuscitation is supported by several retrospective studies and indirect evidence from trauma patients; however, numerous retrospective studies have failed to confirm this.

It has recently been suggested that resuscitation should only be commenced when the systolic blood pressure falls below 50–70 mmHg and that vital

Table III Immediate management of the patient with suspected RAAA

Treatment	*Diagnosis*
O_2 via facemask (to keep $SaO_2 > 95\%$)	Clinical assessment
2 × 14G venous cannulae	12 lead ECG
Blood for cross-match	Ultrasound scan
Order fresh-frozen plasma and platelets	Rarely–CT/aortography
Monitoring–ECG, BP, SaO_2	
Intravenous fluids–per policy	
Contact anaesthetist/surgeon	
Alert theatre	
Arterial line	

ECG = electrocardiogram; BP = blood pressure; CT = computed tomography; SaO_2 = arterial oxygen saturation.

organ function is maintained at this level.[4] However, a systolic blood pressure of 70–80 mmHg might be a more reasonable figure.[8,13]

On the basis of current evidence, it seems that a policy of minimal fluid resuscitation may be compatible with higher patient survival, provided that other guidelines are adhered to. These include rapid surgical management, clamping at the diaphragmatic hiatus and meticulous control of bleeding. If a policy of minimal fluid resuscitation is followed, hypotension should still be treated if there is any evidence of vital organ failure.

Table IV Objectives of anaesthetic management

Ongoing assessment of haemodynamic status
Establishment of monitoring in time available
Prevention of awareness
Maintenance of intravascular volume
Vigorous treatment of any coagulopathy
Maintenance of optimal cardiac output and tissue oxygenation
Control of sudden haemodynamic changes
Intensive postoperative care

Table V Objectives of surgical management

Rapid transfer to theatre
Immediate aortic cross clamping
Via laparotomy
Via thoracotomy (rarely)
Other manoeuvres to gain control of the aorta
Digital manual occlusion
Intra-aortic balloon catheter
22G Fogarty catheter via left brachial artery
Proximal anastamosis
Distal anastamosis
Careful control of surgical bleeding

Theatre management

RAAA is a major challenge for both anaesthetist (Table IV) and surgeon (Table V). The haemodynamic status on reaching theatre is variable, but under-resuscitation is likely if current surgical policies on fluid restriction and rapid clamping are followed. Induction of anaesthesia may cause a catastrophic fall in blood pressure when sympatho-adrenal stimulation is obtunded and the tamponading effect of the tight abdominal musculature is lost. A G-suit may be useful, particularly in the moribund patient.

Crystalloid or colloid can be used for urgent plasma expansion but dilutional coagulopathy is a potential hazard with large volumes. There may be a place for small volume hypertonic saline which has been shown to improve mean arterial pressure, oxygen flux and diuresis over isotonic solutions in traumatic shock. However, hypertonic saline can also lead to cerebral oedema, increased blood loss and hyperkalaemia.[25] Blood loss is best replaced with fresh whole blood and autotransfusion may be useful. Clotting factors should be given as required. Administration of bicarbonate should probably be avoided as it may worsen lactic acidosis.

Control of the aorta should ideally be achieved shortly after induction and allows fluid resuscitation to commence without fear of further aggravating haemorrhage. Aortic cross-clamping results in a dramatic reduction in haemorrhage, allowing effective restoration of filling pressures with intravenous fluids. Stroke volume and cardiac output fall by 15–35% and the systemic vascular resistance rises by 40%, which is some patients may lead to acute cardiac failure. A glyceryl trinitrate infusion may reduce ischaemic changes. Dopamine, frusemide and mannitol are sometimes

used to maintain renal function, although their value in this context is unknown.

Declamping of the aorta may result in a varying degree of hypotension, secondary to cardiac failure and vasodilatation. Declamping shock is caused by a combination of factors including a profound lactic acid and potassium load, hypoxic vasodilatation of the lower extremities, blood loss from the anastomosis, and release of toxic and vasoactive byproducts of anaerobic metabolism. The severity of reperfusion injury will be related to the duration of clamping, the degree of shock prior to clamping, the speed with which the clamps are released and the patient's cardiovascular reserve. In many patients reperfusion injury may lead to cardiac failure and pulmonary complications. Slow release of clamps and cardiovascular optimization prior to clamp release may reduce the extent of reperfusion injury.

Postoperative management

All patients will require postoperative intensive care management. Close monitoring of cardiovascular, respiratory and renal function, graft patency and coagulation status will be required. Patients surviving the perioperative period have a high incidence of complications secondary to hypotension and massive blood transfusion. Fielding *et al.* found that most postoperative deaths occurred in patients who had two or more complications and that renal or respiratory impairment were the most frequent and most lethal[26] (Table VI). Postoperative bleeding is related to surgical trauma and coagulopathy and may precipitate re-operation.

Table VI Incidence of postoperative complications with RAAA

Myocardial impairment	30–50%
Respiratory failure	30–50%
Renal failure	10–40%
Bleeding	10–20%
Ischaemic colitis	5–20%
Stroke	5%
Lower limb ischaemia	4%
Paraplegia	2%

Conclusion

It is generally considered that most advances in the management of RAAA will come from rapid diagnosis, vigorous surgical management and meticulous attention to perioperative haemodynamics and haemostasis. Areas of management requiring emphasis include: early involvement of anaesthetist and surgeon, rapid establishment of i.v. access and direct arterial pressure monitoring, rapid transfer to theatre, appropriate fluid resuscitation based on agreed guidelines and vigorous management of coagulopathy.

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