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Ruptured Abdominal Aortic Aneurysms

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1. Introduction

Abdominal aortic aneurysm (AAA) rupture (RAAA) is defined as bleeding outside the adventitia of a dilated aortic wall. Ruptures are classified as free when they flow into the peritoneal cavity causing massive blood loss, usually associated with a high mortality rate, and as retroperitoneal which are characterized by minor blood loss since retroperitoneal tissues contain the hematoma. In a minority of cases AAAs rupture into the intestinal tract (frequently in the last portion of the duodenum) creating an aortic enteric fistula or into the cava vein. Differentiation between symptomatic and ruptured aneurysm is essential. Like patients with RAAA, individuals with symptomatic AAAs may present with a variable symptomatology ranging from mild tenderness to severe pain, but there is no blood outside the aortic wall at computed tomography angiography (CTA) or intraoperatively (Fig. 1).

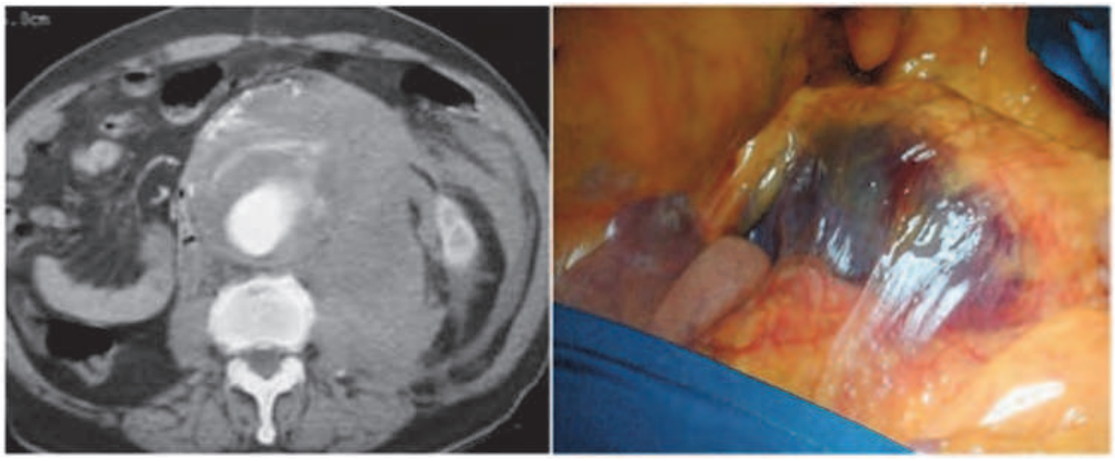

Fig. 1. Preoperative angio-CT showing a RAAA (panel A), and the intraoperative finding (panel B).

Pain is related to the aneurysm itself as a consequence of an acute expansion of the wall, intraluminal hemorrhage or bleeding into the thrombus, or it can be caused by other factors: cholecystitis, diverticulitis, pancreatitis. Patients who undergo elective AAA repair have the best prognosis, followed by patients with symptomatic aneurysms, who do not have hypotension, followed by patients with RAAA. The second of these must not be included in data concerning RAAA as they would artificially improve the outcome results.

The incidence of RAAA ranges between 5.6 and 17.5 per 100,000 person-years in the Western Hemisphere (Moll et al., 2006).

2. Diagnosis, transfer and preoperative care

The mortality rate associated with RAAA is especially high during transfer to the emergency room (ER) or other emergency settings and later in the operating theatre (OT). Most ruptures occur outside of the hospital and the time between onset of symptoms and arrival at the OT has been found to be critical in determining the final outcome. Funds have been provided by national and regional health organizations to plan specialized pre-hospital management and transportation, including the use of helicopters.

RAAA should be suspected in all patients over 50 complaining of abdominal or back pain, hypotension, and presenting with a pulsatile mass (the classic triad presentation). The mass may be obscured in patients with a large abdominal circumference. An episode of syncope can be indicative of orthostatic hypotension. An emergency Doppler ultrasound (DUS) should be performed to confirm the diagnosis in those patients. The patient should be transferred to the nearest high-volume hospital where an expert vascular/endovascular surgical team is available. Patients needing elective and RAAA repair whose surgeons have high annual procedure volumes are more likely to survive after surgery. A volume-outcome relationship has already been demonstrated for elective AAA repair as well as for other operations (Cho et al., 2008; Heikkinen et al., 2002; Dueck et al., 2004). It is unclear if surgeons with high annual procedure volumes become more expert and therefore produce superior outcomes or if better surgeons producing superior outcomes garner more referrals and thus become high-volume surgeons. Vascular surgeons, in any case, have better outcomes than general surgeons. RAAA patients of high-volume surgeons trained in vascular surgery have an adjusted 30-day mortality rate of 40.5%, compared with 43.9% for all other surgeons, with an absolute risk difference of 3.4%. Although the absolute difference in adjusted 30-day survival between high-volume vascular surgeons (3.5%) and all other surgeons (5.0%) was smaller for elective repairs, the number of elective operations is higher with respect to the number of RAAA repairs (Cho et al., 2008; Harris et al., 1991).

The statistics concerning surgeon volume and mortality suggest that it may be beneficial to regionalize RAAA repair to high-volume vascular surgeons. But the argument can also be made that regionalization can lengthen the time between the onset of symptoms and surgery while patients are being transferred from one hospital to another. Informed policy decisions concerning regionalization cannot be made until the time effect is compared with the benefit of being operated on by high-volume vascular surgeons. Patients may, however, prefer to undergo surgery closer to home, and this could nullify the small survival advantage for elective AAA repair.

This controversy seems to have been partially resolved by Endovascular Ruptured Aneurysm Repair (EVRAR). In a recent study by McPhee et al. who analyzed data gathered in the USA between 2001-2006, EVRAR was found to be independently associated with a lower postoperative mortality risk than was open surgical repair (odds risk 1.56), with the highest advantage found in high-volume centers for elective EVAR (McPhee et al., 2009).

When a RAAA is suspected a blood sample should be taken for analysis and cross matched (a minimum 8 units of blood cells and 4 units of blood plasma should be prepared) and a large intravenous access and a Foley catheter should be placed simultaneously.

Optimal management guidelines for preoperative crystalloid infusion in patients with severe hypotension have not been established. Aggressive resuscitation during which large quantities of fluids are administered can elevate systolic blood pressure (SBP) causing rupture of the temporary aortic seal that forms leading to further blood loss and worsening of hypotension (Johansen et al., 1991). The optimal strategy, termed permissive hypotension, seems to be providing minimal fluid infusion and the pharmacological resuscitation necessary to maintain consciousness with a SBP of about 80 mm Hg (50-90mm Hg) (Crawford 1991; Alric et al., 2003). When possible, red blood cells should always be used during resuscitation. There are no randomized trials comparing different types of resuscitation for RAAA. In a series of patients presenting with severe hypotension (SBP<90 mm Hg) survival was improved in those who received minimal fluid infusion and in whom permissive hypotension was maintained until they arrived in the OT (Bickell et al., 1994).

An emergency ultrasound examination should be performed by an expert operator to confirm the diagnosis once the patient has arrived in the ER and, if the patient is hemodynamically stable (consciousness and SBP >90 mmHg), a CTA should subsequently be performed to examine the aneurysm anatomy, to identify the rupture site, to plan aortic cross-clamping (supra or infra-renal), and to analyze the feasibility of endovascular repair. The unstable patient (unconsciousness and SBP<90 mmHg) should be taken directly to the OT (Fig. 2).

3. Open RAAA repair

Once in the OT, the patient must be quickly prepared and draped so that the operation can begin after induction of anesthesia that can itself cause severe hypotension if curare is used. An intra-arterial line to monitor blood pressure, a central vein catheter, and a nasogastric tube should be placed.

Conventional open surgical repair (OR), involving surgical exposure of the aorta and replacement of the aneurysm with a synthetic tube graft if possible, is the most common treatment for AAA. Its advantages include shorter operating times and limited overall systemic physiologic insult. A small (2-3 cm) iliac aneurysm or a moderate stenosis can be repaired at a later date. Systemic heparinization, which reduces bleeding complications, can be avoided although retrograde iliac flushing and injection of a heparinized flush solution into the iliac arteries is necessary to remove any soft clots and to reduce distal thrombosis. Once RAAA repair has been completed and before the aorta has been unclamped, which is always associated with significant hypotension, the anesthesiologist must verify that appropriate fluids are being administrated and that pressor agents and bicarbonate are prepared for use if required. Once the repair has been performed in about 25-30% of cases the abdomen cannot be closed without significant tension from a swollen bowel or a massive retroperitoneal hematoma or both that could lead to postoperative abdominal compartment syndrome secondary to an increase in intra-abdominal pressure. To avoid that complication an early or delayed closure of the abdomen using a non-absorbable mesh covered with polyurethane is indicated rather than a decompressive laparotomy, as it seems to reduce graft infection and multiple organ failure development (Rasmussen et al., 2002). Open AAA repair is a complex, major operation with high morbidity and mortality due to the combined effects of surgical exposure, hemorrhage, and aortic clamping which may induce lower torso ischaemia-reperfusion injury.

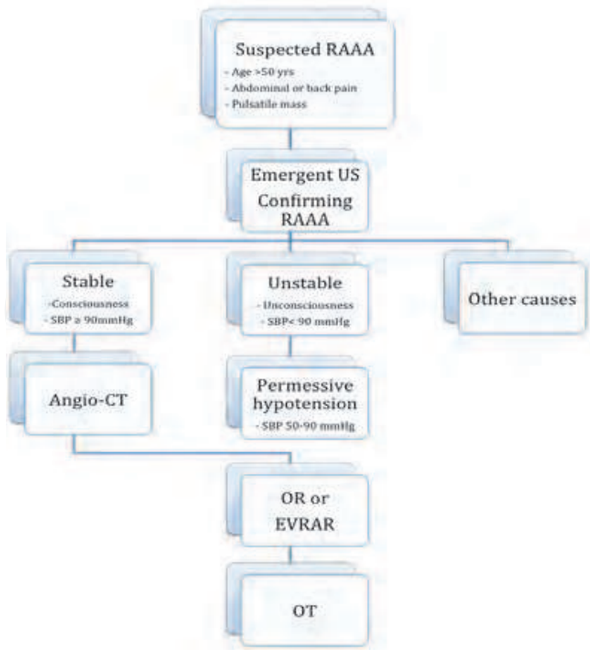

Fig. 2. Flow-chart showing a simplified protocol for the management of RAAA. *(OR: open repair, OT: operating theatre).*

4. Operative strategies for RAAA

The main goal of surgery to repair RAAA is to obtain a safe, rapid, and effective proximal cross-clamping of the aorta to interrupt hemorrhage. Different intraoperative approaches and strategies with regard to surgical and anesthetic aspects can be used to obtain aortic control and to improve the postoperative outcome.

4.1 The transperitoneal approach

The most common open approach is the transperitoneal one through a midline abdominal incision. Most surgeons prefer this technique because they are more confident with it and because it provides wide exposure of the infrarenal as well of the suprarenal aorta. It is possible, in fact, to rapidly dissect the infradiaphragmatic aorta and to cross-clamp the aorta at this point. To gain aortic control the left lobe of the liver must then be retracted to the right and the gastrohepathic omentum is opened allowing entry into the lesser sac. A nasogastric tube is usually used to identify the esophagus, which is retracted to the left. It is then possible to expose the aorta between the diaphragmatic crura, which is dissected with an electrocautery to facilitate a rapid, safe clamp placement. Once aortic control is obtained the clamp on the subdiaphragmatic aorta should be placed on the infrarenal neck if possible. The supraceliac aortic cross-clamping must be as short as possible to reduce visceral and renal ischemia. Some surgeons prefer to perform supraceliac aortic cross-clamping routinely to control hemorrhage readily and to avoid damage to the left renal vein and/or the gonadic veins which may take place during an infrarenal neck dissection when there is a large hematoma in that area. Some concerns have been voiced with regard to this method because supraceliac cross-clamping can worsen the visceral and renal ischemia induced by hemorrhagic shock, which in turn could contribute to often fatal multisystem organ failure.[32]

4.1.1 The left retroperitoneal approach

Some surgeons prefer the left retroperitoneal approach to the abdominal aorta because of the advantages that it offers with respect to the standard transabdominal access. It has, in fact, been associated with less severe intraoperative hypothermia, fluid loss, lower postoperative ventilatory deficit, and a shorter period of paralytic ileus (Chang et al. 1990). This exposure is, moreover, considered the approach of choice for patients with a so-called "hostile abdomen", those who have undergone multiple intraabdominal procedures or pelvic irradiation. It can also be useful in obese patients and in cases of horseshoe kidney (Wahlgren et al., 2007).

The approach provides wide access to the infradiaphragmatic aorta as a 10th intercostal incision is used allowing rapid control of the supraceliac aorta before the aneurysm is exposed through the hematoma. Some surgeons in fact, advocate its routine use for RAAA repair. A retrospective analysis (Chang et al., 1990) on RAAA treated via a retroperitoneal extended approach (10th intercostal incision, 25 pts) as opposed to a standard transperitoneal procedure (38 pts) revealed that there was a lower 30-day mortality (12% vs 34.%; $p<0.05$), less need for ventilatory support in the OT, and a shorter duration of paralytic ileus when the former was used. But a prospective randomized study (Cambria et al., 1990) analyzing the transperitoneal approach (59 pts) as compared to the retroperitoneal procedure (54 pts) reported finding no advantage to the former.

Some concerns have been voiced with regard to this approach and especially concerning the difficulty in controlling the right renal artery, and, if the aneurysm is large, exposure of the right iliac artery could be technically demanding. It is important then that the surgeon be familiar with a procedure which, at best, is difficult. The retroperitoneal approach may, moreover, be contraindicated in patients undergoing active cardiac massage for resuscitation or with a questionable diagnosis.

4.1.1.1 Balloon occlusion

According to some surgeons safe aortic control can be attained using an aortic occlusion balloon (AOB) placed in the visceral aorta under fluoroscopic guidance enabling laparotomy while avoiding the risk of rupturing the aneurysm and causing massive hemorrhage. Lieutenant Colonel Carl W. Huges first introduced the technique to repair traumatic rupture of the abdominal aorta in 1950 during the Korean war (Arthis et al., 2006). Many advancements have been made since then especially during the last decade. Newer techniques and materials are now used to obtain aortic control using a compliant AOB (Assar et al., 2009). The most common approach is to insert the AOB through a percutaneous transfemoral access using a 11 to 16 F sheath placed in the suprarenal or pararenal aorta over a stiff guidewire (Arthurs et al., 2006; Malina et al., 2005; Mayer et al., 2009). A compliant AOB is then passed through the sheath and inflated proximal to the RAAA. The sheath is advanced to support the inflated balloon from below to avoid distal dislocation. The sheath should be fixed firmly to prevent any displacement that can occur if blood pressure rises. The entire procedure should be performed under fluoroscopic guidance, but when this is unavailable confirmation of occlusion is generally associated to the loss of a palpable femoral pulse and an increase in blood pressure.

A transbrachial approach has also been described for RAAA (Matsuda et al., 2003). In 1964 Heimbecker first described remote transaxillary aortic control using AOB in RAAA (Heimbecker 1964). The advantages of transbrachial or axillary AOB placement is that the AOB inflated at the infrarenal aorta level does not interrupt the blood supply to the celiac,

supramesenteric, and renal arteries. Important concerns have, however, been advanced with regard to this approach, and, in fact, it does seem to present some disadvantages. This technique may, in fact, involve a cutdown procedure to access the brachial artery and at times this may require a second operating team and dissection of the median nerve, potentially increasing the time for AOB placement. Most AOBs are large, bare, stiff, and difficult to pass in a blind fashion across the arch of the aorta. Oblique imaging may be necessary to traverse the arch into the descending thoracic aorta, while placement of a large sheath in the brachial artery may cause additional ischemic complications in the upper extremity.

Risks associated with AOB, the most catastrophic of which being that of provoking a free rupture in a contained RAAA, need be considered. A wire or catheter could feasibly exit through the site of the aortic rupture or the weakened aortic wall aggravating hemorrhage. Transfemoral AOB should then always be performed under fluoroscopic guidance to avoid this complication. Another potential obstacle could be percutaneously locating the common femoral artery in a hypotensive patient. Low et al (1986) reported a 58% success rate in the percutaneous cannulation of the common femoral artery to place an AOB in cases of traumatic exsanguinating hemorrhage. When the attempt fails, a femoral cutdown procedure is required and ultrasound-guided access techniques may be useful. The risk of visceral, spinal cord, and lower extremity ischemia in an already moribund patient is associated to any technique of proximal control depending on the extent of the preexisting shock and collateral flow. Aortic occlusion times vary and some surgeons limit balloon inflation time to 10 minutes with a variable period of deflation for reperfusion (Assar et al., 2009) because intermittent aortic occlusion is better tolerated. This technique can also be applied to gain control of the iliac arteries once the aneurismal sac is opened to avoid iliac vein lesions if there is a large hematoma.

4.1.1.2 Intraoperative Auto Transfusion (IAT) and red cell salvage

IAT is a method of blood conservation that reduces the need for allogeneic blood transfusions during AAA surgery (Alric et al., 2003; Takagi H et al., 2007). In a systematic review including 2 small randomized controlled trials, Alvarez et al (2004) did not find evidence that IAT decreases exposure to allogeneic blood transfusions (ABT) during infrarenal AAA surgery. Since the time that review was written, the results of two large randomized controlled trials have been published. In a recent meta-analysis of randomized controlled trials published before 2007, Takagi et al reported that IAT reduces the risk of ABT in elective AAA surgery, and its use has been recommended even for RAA (Markovic et al., 2009). Once IAT becomes routine in all AAA surgeries and all operators have become familiar with it, its use may become commonplace during RAAA repair protocols.

IAT has obvious advantages as blood volume and normothermia are maintained because of the rapidity with which blood is salvaged and returned to the patient. Two basic blood salvage and replacement methods are possible: washed and unwashed (whole blood) autotransfusion. Cell washing devices can theoretically remove potentially toxic byproducts of injured red blood cells, such as activated clotting proteins and activated complement, but they also eliminate beneficial blood components such as platelets and clotting proteins. Although heparin is added by the autotransfusion system, it is removed when the blood is washed by an autotransfusion device. Unwashed autotransfusions return not only platelets and proteins but also unwanted free hemoglobin and activated coagulation factors known to increase the frequency of transfusion-related coagulopathy or nephropathy and its use

has thus been entirely abandoned. Dilutional coagulopathy is considered the major adverse event related to the use of cell washing systems. Administration of fresh frozen plasma and platelets is recommended when more than 8 units of autologous blood have been returned (Marty-Ane et al., 1995). IAT is particularly useful in the treatment of massive hemorrhage during which rapid reinfusion of large volumes of blood can be lifesaving and it is most effective when the salvage and reinfusion of shed blood can be accomplished at flow rates compatible with the degree of hemorrhage. Postoperative coagulation disorders and hypothermia-induced coagulopathies are prevented to a large extent by autotransfusion which returns warmer blood products (Marty-Ane et al., 1995).

4.1.1.2.1 Renal function

The incidence (ranging between 26 and 42%) of renal failure after RAAA is remarkably high. Of patients with renal failure, 11-40% require postoperative dialysis with an associated mortality rate of 76-89% (Harris et al., 1991; Magee et al., 1992). Impairment in renal function has been associated with suprarenal cross-clamping, duration of cross clamping >30 minutes, preexisting conditions of renal dysfunction (serum creatinine > 2 mg/dl), shock, and age over 80 (Bonventre 2007; Nicholson et al., 1996). The etiology of renal dysfunction is multifactorial and many who develop it have sustained major insult to other organs. Ischaemic renal injury affects the tubules at the level of the outer medullar, which primarily include the thick ascending loop of Henle and the S3 portion of the proximal convoluted tubule. Tubular cell death is now considered characterized by both necrosis and apoptosis. Necrosis results from profound cellular adenosine triphosphate (ATP) depletion and is characterized by a sequence of events that begins with loss of cell polarity and of the epithelial brush border, followed by the loss of the integrity of tight junctions and the appearance of integrins such as intercellular adhesion molecule 1 on the cell surface. These interact with leukocyte adhesion molecules to mediate an inflammatory response with the release of cytotoxic mediators. Cells then slough into the tubular lumen and further impair already compromised filtrate flow. Tubular cell apoptosis is also triggered by ischaemia through as yet uncharacterized mechanisms, also resulting in cell loss, but there is no inflammatory component, and the resultant apoptotic bodies are phagocytosed by macrophages or surviving epithelial cells. Apoptosis is also observed in the recovery phase during epithelial proliferation and probably has a role in restoring a normal tubular structure. Clinically, the initial observation is that of a loss of urinary concentrating ability as the medullary gradient dissipates, followed by a decline in urine output as tubules become obstructed and denuded (Bonventre 2007).

Some intraoperative strategies concerning anesthetic and surgical aspects can be utilized to protect the renal parenchyma. Use of mannitol, an osmotic diuretic and the earliest pharmacological agent utilized to protect renal function following vascular surgery, continues to be controversial. In a randomized controlled study, Nicholson et al reported that it reduces renal injury following infrarenal AAA, but in a recent review Hersey et al. (2008) concluded that there is no evidence that mannitol preserves renal function during aortic surgery. Mannitol infusion, nevertheless, is the most commonly used method to protect renal parenchyma from ischemic damage and from ischemic-reperfusion injury. Its use has been shown to protect renal function during elective surgery when suprarenal aortic cross-clamping does not exceed 60 minutes (Deriu et al., 2001). The dose commonly used is a bolus of 25 mg IV administered before or immediately after aortic cross-clamping. Mannitol

seems to increase tubular fluid volume through its osmotic effect and by reducing tubular cell swelling and hence resistance to flow. Since it is a hydroxyl radical scavenger, it is effective in an ischaemia-reperfusion injury setting (Hersey & Poullis 2008).

Another pharmacological therapy that can be begun before or during supraceliac aortic cross-clamping is fenoldopam mesylate, a dopamine-1 agonist receptor which improves plasmatic renal blood flow to the cortex and medullary regions. The dose used to obtain renal effects is 0.05/0.003 μg per kilogram per minute, is well below that usually needed to lower systemic blood pressure (Halpenny et al., 2002; Gilbert et al., 2001). There is no data on its usefulness during RAAA surgery, but those effects on renal function noted during aortic surgery probably could presumably apply to RAAA as well.

Other drugs such as dopamine, dopexamine, calcium channel antagonists and natriuretic peptide have been proposed, but their use is still limited and further studies are needed.

Several intra-operative surgical strategies are applicable to protect renal tissue from prolonged cross-clamping ischemia. Many surgeons utilise the local cooling of the kidneys to protect the tissue from protracted cross-clamping ischemia by cyclic or continuous perfusion of renal arteries with cold solutions (Ringer's lactate or saline) (Allen et al., 1993; Kashyap et al., 1997). Ice slushes can be applied to the kidney surface to further ensure hypothermia, if exposure is retroperitoneal. But hypothermia of the renal parenchyma remains a problem when the kidney remains *in situ*, and the results found using this method are controversial with regard to the prevention of post-operative renal failure (Allen et al., 1993). In the light of our research studies and preliminary clinical reports, we utilize short-term kidney arterial blood reperfusion whenever clamping ischemia exceeds 30 min.

Reperfusion is achieved by re-establishing pulsatile normothermic blood flow either through the repaired renal artery or, in the majority of operative procedures, through the Pruitt–Inahara shunt (500-50-9F. Ideas for MedicineTM, Cryo-Life® Comp, St. Petersburg, FL, U.S.A.).

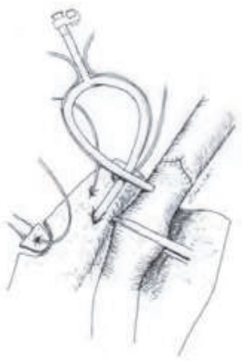

Fig. 3. The temporary renal reperfusion is achieved by re-establishing pulsatile normothermic blood flow through a Pruitt–Inahara shunt (500-50-9F. Ideas for MedicineTM, Cryo-Life® Comp, St. Petersburg, FL, U.S.A.) when the procedure requires more than 30 min of renal artery cross clamping. The proximal end of the shunt is inserted into the tubegraft and its distal end into the renal artery. After 3 min of blood the aorta and the renal artery are clamped, the shunt is promptly removed and the renal artery reconstruction completed. The reperfusion is repeated every 30 min if necessary.

This technique can protect renal parenchyma from prolonged clamping ischemia (up to 100 min) (Deriu et al., 2001).

5. Postoperative complications

The most frequent complications after surgery for RAAA are listed in Table 1.

	Incidence	Associated mortality
Postoperative bleeding	12-14%	-
Colonic ischemia	3-13%	73-100%
Respiratory failure	26-47%	34-68%
Renal dysfunction	26-42%	-
Postoperative dialysis	3-18%	76-89%
Myocardial infarction	14-24%	19-66%
Major arrhythmia	19-23%	40-48%
Congestive heart failure	18-21%	39-42%
Multisystem organ failure	59-66%	65-71%
Paraplegia and paraparesis	1.1-2.3%	45-53%

Table 1. Major postoperative complications following open repair for RAAA.

Uncontrollable haemorrhage during RAAA repair is a major cause of perioperative mortality (Davies et al., 1993; Milne et al., 1994). Patients with coagulopathic haemorrhage must undergo timely administration of fresh frozen plasma, platelets, cryoprecipitate and antifibrinolytic agents. But in patients arriving in the OT with signs of severe haemorrhagic shock, these measures may be insufficient to restore normal haemostatic function in the face of persistent hypoperfusion, acidosis and hypothermia by the end of the operation. In this clinical situation, intra-abdominal packing is recommended to control further bleeding, to optimize organ perfusion, and to correct metabolic disturbances in the intensive therapy unit (van Herwaarden et al., 2001). Concerns have been advanced with regard to this technique and the possibility of secondary intra-abdominal infective complications noted in 13% in an early postoperative period and in 18% in a later phase and as a result the technique is not commonly used in open RAAA repair (Johnston 1994).

Colon ischemia can develop as a single mucosal lesion or as necrosis involving the full-thickness of the colon with or without perforation. Several factors are thought to be responsible for colonic ischemia: severe hypotension, preoperative inferior mesenteric artery (IMA) patency, the presence of a collateral supply from the superior mesenteric, and the site of hematoma. The patency of at least one hypogastric artery is generally considered acceptable to ensure adequate intestinal perfusion. After RAAA repair the colon must be carefully inspected and in case of doubt the IMA should be reimplanted and/or the hypogastric arteries should be inspected by ultrasound examination to verify their patency. If colonic ischemia is suspected during the postoperative period, a colonscopy should be performed as it is considered diagnostic, and colon resection, associated with a high mortality rate (50%), may be necessary (Harris et al., 1991; Johnston 1994).

Myocardial infarction, associated with an high mortality rate (20-65%), develops as a result of excessive cardiac workload due to blood loss, aggressive resuscitation efforts, clamping and declamping in patients often with pre-existing history of ischemic coronary artery disease %).[61] Rapid diagnosis and treatment of this fatal complication is imperative.

Multiple-organ failure (MOF) is the major cause of death in the intensive care unit after successful RAAA repair.[62] Some factors such as suprarenal aortic cross-clamping and longer

operative duration of aortic clamping seem to be involved in its development (Bauer et al., 1993). Infection and shock are the two most common clinical predisposing factors and processes such as severe tissue injury or pancreatitis that induce a major inflammatory response seem to be capable of initiating a cascade of events that culminates in MOF (Deitch 1992; Haveman et al., 2008). Regardless of the cause, MOF generally follows a predictable course, beginning with the lungs and followed by hepatic, intestinal, and renal failure, in that order. Hematologic and myocardial failure are usually later manifestations of MOF, while the onset of alterations involving central nervous system alterations can occur either early or late. This classical sequential pattern of organ failure can be modified, however, by pre-existing disease or by a precipitating clinical event. Renal failure, for example, may precede hepatic or even pulmonary failure in subjects with intrinsic renal disease or in patients who have sustained prolonged periods of shock, while hepatic or myocardial failure may be an early or even the initial manifestation of this syndrome in a patient with cirrhosis or myocardial damage. These clinical exceptions illustrate the biologic principle that, although systemic responses are similar in patients developing MOF, the exact sequence of organ failure can be influenced by the individual's acute disease processes or physiologic reserve (Deitch 1992).

One hypothesis concerning the development of MOF after RAAA surgery is based on the "two hit" theory: the rupture and repair of an AAA is a combination of two ischemic-reperfusion injuries (Lindsay et al., 1999). The first ischemic event is hemorrhagic shock, which sets off an inflammatory response, followed by resuscitation causing the first reperfusion injury. The second ischemic event is aortic cross-clamping followed by the aortic declamping causing the second reperfusion injury.

This theory supports the microcirculatory hypothesis of MOF according to which organ injury is related to ischemia or vascular endothelial injury. In the macrophage hypothesis, instead, prolonged activation of macrophages results in excessive production, surface expression, and liberation of cytokines and other products, which through a cascade effect involving humoral and cellular effector systems exert deleterious local and systemic effects. The former hypothesis includes several distinct but to some extent overlapping potential mechanisms of injury, including inadequate tissue and cellular oxygen delivery, the ischemia-reperfusion phenomenon, and tissue injury due to endothelial-leukocyte interactions (Reilly et al., 1991; Granger et al., 1988). There are, thus, many points in which the microcirculatory and macrophage hypotheses of organ failure overlap and interact (Deitch 1992; Pober & Cotran, 1990). Clinical and experimental observations have in fact clearly demonstrated that systemic inflammation adversely affects the microcirculation, while ischemia can exaggerate the host's inflammatory response to subsequent stimuli by activating neutrophils and priming macrophages (Deitch 1992).

Prognosis appears to be directly related to both the number of organs that fail and the length of time the patient is in organ failure, but our ability to predict outcome in individual cases is not so precise as to supersede clinical judgment in determining when further treatment is futile.

The first cause of death in the surgical intensive care unit is MOF, and the best treatment is prevention. In spite of the development of new-generation antibiotics and ever more sophisticated techniques of organ support, our ability to save patients once MOF has set in has not appreciably improved over the last two decades (Deitch 1992). New therapeutic

strategies aiming to prevent and/or limit the development of the physiologic abnormalities inducing organ failure are needed to improve survival in these patients.

6. Results after RAAA repair

The mortality rate of RAAA remains exceedingly high; in fact with the exception of only a few series (Crawford 1991, Lawrie et al., 1980), it ranges between 30 and more than 70% in most reports (Mureebe et al., 2008; Bown et al., 2002; Johansen et al., 1991; Bauer et al., 1993). If these statistics include patients who died at home or during transportation to the hospital, the mortality rate approaches 90% (Dueck et al., 2004). Poor survival rates have been reported throughout the last few decades despite advancements in specialized pre-hospital management and transportation, rapid emergency diagnostic evaluation and aneurysm repair by high-volume vascular surgery teams, and sophisticated post-operative intensive care units (Darling et al., 1996; Bown et al., 2002; Alric et al., 2003; Bauer et al., 1993; Dueck et al., 2004; Antonello et al., 2009).

In the light of high mortality rates and high hospital costs, some attempts have been made to identify pre-operative variables that might be used to select patients for surgery (Antonello et al., 2009; Boyle et al., 2003; Calderwood et al., 2004; Tambyraja et al., 2008a, 2008b). While this approach may be justified in our cost-effective age, its ethical and legal implications need to be pondered. The decision to deny operative treatment to a patient with a RAAA can be made only on an individual basis and can be justified only in those cases characterized by poor quality of life due to a precarious general clinical status or mental condition. Variables and scoring systems (Tab. 2), have recently been developed to identify patients with a prohibitive operative risk for RAAA repair.

Among these, the Glasgow Aneurysm Score (GAS) seems to be the most simple and accurate in identifying those patients unfit for emergency repair surgery (Antonello et al., 2007; Korhonen et al., 2004). Sufficient data are still lacking in the literature with regard to these systems, GAS included, and large prospective studies are certainly warranted.

The best approach to RAAA remains, in any case, prevention. DUS screening programs for AAA in men over 65 have recently been attempted to reduce mortality (Scott et al., 1995, 2002; Lindholt et al.). The rationale behind these programs is that ultrasound is a valid, fast, safe method to detect AAAs, with an estimated sensitivity and specificity of 98 and 99%, respectively, and that perioperative mortality for elective AAA is lower than 5% in most centers (Lindholt et al., 1999). The Multicenter Aneurysm Screening Study (MASS) results published in 2002 in the Lancet journal showed that ultrasound screening performed over a 29 month period in men between 65 and 74 reduced the AAA mortality risk by 42% (Ashton et al., 2002). Two other reviews on this topic were later published by Cochrane (Cosford et al., 2007) and Lindholt et al. who confirmed these findings and reported a significant mid-term reduction in AAA-related and overall mortality after 3-5 yrs, data that has been verified by long-term analysis. There is, instead, insufficient evidence demonstrating usefulness in women. While ultrasound screening is relatively inexpensive, further analysis is needed, and all the latest findings concerning this subject must be examined carefully whenever health care administrators are considering population-based screening programs.

	Odds ratio (95%CI)
Age >80 year	1.5-3
Female sex	1.5-2
Hypotension < 80mmhg	2.5-3.5
Renal impairment	2-3.5
Pulmonary disease	1.5-2.2
Haemoglobin <8 g/l	1.8-2
Hematocrit <30%	1.8-2.5
Diabetes	1.3-1.8
Coronary artery disease	1.7-2.2
Suprarenal cross clamping	1.8-2.5
	Area under the roc curve (95% CI)
Hardman Index	0.69
GAS°	0.64
ERAS§	0.72
Possum	0.69

* Physiological and Operative Score for Enumeration of mortality and Morbidity
° Glasgow Aneurysm Score
§ Edinburgh ruptured aneurysm score

Table 2. Principle risk factors and Scoring Systems developed to predict survival after open repair for RAAA.

7. Quality of life after RAAA

The long-term impact on quality of life after AAA repair is debatable. Health, of course, cannot be defined simply as the absence of disease and infirmity, but implies physical, mental, and social well-being. Traditional outcome measures with regard to vascular surgery, such as graft patency and complication rates, are certainly important. These measures tend, nevertheless, to reflect a technical point of view towards the procedure rather than concern for the impact that the event has had on the patient as a whole. Measuring quality of life (QL) before and after surgery is one way to evaluate the patient's perception of health and well-being. In extreme situations, a technically successful operation might leave a patient significantly debilitated by morbidity associated with the surgery. This seems to be a very real possibility, in fact, for survivors of AAA repair, given the high operative morbidity and mortality rates. The most frequently used questionnaires are the Rosser index (Korhonen et al., 2003; Joseph et al., 2002) (a measure of physical disability and psychological distress) and the Medical Outcomes Study Short Form-36 Health Survey (McDaniel et al., 2000; Reemtsma & Morgan, 1997; Chetter et al., 1997) (Sf-36; a generic QL measurement tool) promoted by the American Society for Vascular Surgery and validated for use in patients with vascular disease.
In studies utilizing the Rosser index, not widely validated for vascular patients, a significant reduction was found in the level of functional capacity in the RAAA group with respect to

patients who underwent elective repair (Magee et al., 1992; Hennessy et al., 1998). Biases emerged in these reports as the studies were small and the patients were not matched for comorbidities, so it is difficult to ascertain if the populations (elective vs emergency) were similar.

Larger studies with matched populations (elective vs emergency) were performed using the SF-36 by Tambyraja et al. (2005) and Hill et al. (2007) who reported that there were no significant differences in QL in the two groups. These studies also suggested that long-term RAAA survivors had the same QL as those who underwent open elective repair surgery.

8. Endovascular repair

Endovascular aortic repair is a new catheter-based, imaging guided procedure that has the potential to redefine the traditional approach to the treatment of AAA. Since it was first described by Parodi in 1991 (Parodi et al., 1991), its role in RAAA repair continues to be debated. In fact, while most published data drawn from non-randomised studies (Mayer et al., 2009) suggest that EVRAR is feasible in selected patients and in institutions specialized in endovascular techniques, the only published randomized control study by Hinchliffe et al. (2002) showed no benefit in terms of mortality or complications. But since that trial included symptomatic but non-ruptured AAA patients it is difficult to interpret these results. Long-term findings are needed to assess if EVRAR effectively treats endoleaks, prevents late ruptures, and ensures stent-graft integrity. The potential advantages of EVRAR with respect to open repair, such as reduced blood loss, less need for transfusions, and shorter stays in the ICU seem to be due to a decreased physiological insult. As EVRAR obviates the need for laparotomy, direct surgical exposure, handling of abdominal contents, and aorto-iliac clamping, it is an attractive alternative and potentially helpful in reducing the mortality rate of RAAA (Ten Bosh et al., 2010; Harkin et al., 2007; Sadat et al., 2008; Dillon et al., 2007).

Different strategies are employed to treat RAAA using endovascular therapy.

Proximal aortic control during EVRAR is obtained using a balloon placed in the visceral aorta via the brachial or the femoral accesses. The use of a balloon occlusion device is however associated to the risk of renal and splanchnic ischemia and distal embolization does not prevent on-going blood loss from ilio-femoral arteries.

The most common procedure is aorto-unifemoral graft. The advantage of this method is that the aneurysm is quickly excluded from the circulation since introduction and deployment is rapid, and the contralateral gate does not need to be engaged as opposed to the bifurcated endograft which can be time consuming in some situations. Stent grafts may offer wider applicability but can be used only when unilateral iliac anatomy is suitable, but a femoro-femoral crossover is nevertheless required (Fig. 4).

Thanks to the recent diffusion of the endovascular technique most RAAAs are treated using aorta-bi-iliac endografting, and no differences in outcomes have been found in the two types of endografts (Moll et al., 2011).

Abdominal compartment syndrome is a major cause of death after EVRAR and increases short-term mortality up to 5 times compared to that in patients with normal intra-abdominal pressure. All EVRAR patients should be monitored for this syndrome by frequent bladder pressure readings and open surgery must be considered when bladder pressure rises over 20 mm Hg (Djavani et al., 2011). The major concerns regarding

endovascular treatment in emergency situations are the patient's hemodynamic condition and time delays during the preoperative work up. The Montefiore RAAA Management Protocol (Harkin et al., 2007) seems to provide solutions and foresees using the endovascular approach in all patients with presumed ruptured aortoiliac aneurysms. Recent studies on EVRAR outline the procedure's many benefits observed in selected groups of patients with an anatomical suitability for EVAR, commonly reported at 60% (range 18-83%) (Ten Bosh et al., 2010, 2011). In those cases there was a reduction in the mortality rate, in the length of time spent in intensive care, and in the total time spent in the hospital traditionally associated with open repair. But due to heterogeneity and biases these results should be interpreted with caution (Sadat et al., 2008; Dillon et al., 2007). Large multicenter randomized controlled trials will establish the efficacy of EVRAR.

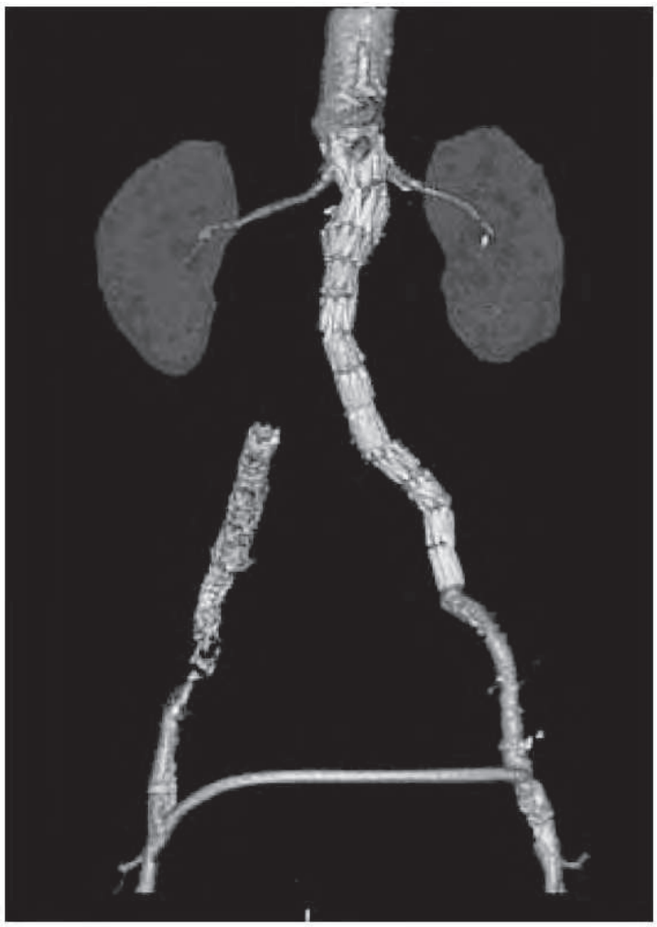

Fig. 4. Post-operative angio-CT showing an aorto-unifemoral endograft with a femoro-femoral cross-over performed for a RAAA

A pilot study recently outlined the difficulty in carrying out that kind of study (Hinchliffe et al., 2006). Some of the problems included: obtaining informed consent from patients with hemodynamic instability, inclusion of patients unsuitable for EVAR as well as traditional repair, immediate availability of CTA instrumentation, expert endovascular teams, and endograft devices.

9. Conclusions

Despite an increase in the volume of patients undergoing elective AAA repair over recent decades, the number of patients with RAAA has not fallen significantly (Johansson et al., 1994; Acosta et al., 2006; Wanhainen et al., 2008). Although the surgical mortality rate for elective AAA repair has steadily improved due to advancements in operative techniques and perioperative care and has fallen to less than 2% in specialized centres and 5% in less-specialized hospitals, the mortality rate of RAAA has not significantly changed over the past three decades and still ranges between 30 and 70% according to recent reports (Heller et al., 2000; Marty-Ane et al., 1995; Mureebe et al., 2008; Cho et al., 2008; Darling et al., 1996). The high mortality seems to be related to a combination of hemorrhagic shock and lower torso ischaemia followed by reperfusion injury despite successful revascularization (Harris et al., 1991; Bown et al., 2002)

Given the poor outcomes after open RAAA repair and in the light of lower perioperative morbidity and mortality after EVAR, some centers have adopted EVAR protocols for RAAA repair. Excellent results have been reported by those centers where the mortality rate has fallen to as low as 30%(Ten Bosch et al., 2010; Visser et al., 2007). But despite these results this technique cannot be offered to all patients with RAAA due to anatomic considerations (short neck, poor iliac access) and it has been estimated that only about 60% are anatomically suitable. (Visser et al., 2007; Ten Bosch et al., 2010, 2011) Additional factors reducing the applicability of EVRAR are institutional limitations, such as the lack of trained endovascular surgeons and/or endovascular equipment. But beyond the inherent difficulties and limitations connected to its use, EVRAR seems to present very important benefits confirming its usefulness and value as a modern surgical option for RAAA repair (Veith et al., 2003).

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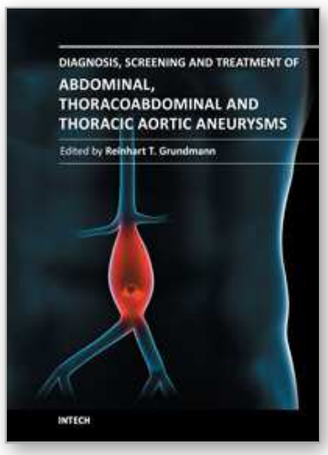

Diagnosis, Screening and Treatment of Abdominal, Thoracoabdominal and Thoracic Aortic Aneurysms

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This book considers mainly diagnosis, screening, surveillance and treatment of abdominal, thoracoabdominal and thoracic aortic aneurysms. It addresses vascular and cardiothoracic surgeons and interventional radiologists, but also anyone engaged in vascular medicine. The high mortality of ruptured aneurysms certainly favors the recommendation of prophylactic repair of asymptomatic aortic aneurysms (AA) and therewith a generous screening. However, the comorbidities of these patients and their age have to be kept in mind if the efficacy and cost effectiveness of screening and prophylactic surgery should not be overestimated. The treatment recommendations which will be outlined here, have to regard on the one hand the natural course of the disease, the risk of rupture, and the life expectancy of the patient, and on the other hand the morbidity and mortality of the prophylactic surgical intervention. The book describes perioperative mortality after endovascular and open repair of AA, long-term outcome after repair, and the cost-effectiveness of treatment.

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