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Treatment of Ruptured Abdominal Aortic Aneurysms

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1. Introduction

The incidence of Abdominal Aortic Aneurysms (AAA) has persistently increased over the past decades (Best et al., 2003). This is partly attributed to increased ageing of the population, improved diagnostic tools and the introduction of screening programmes (Sakalihasan et al., 2005). To date, AAAs are responsible for 1.3% of all deaths among men aged between 65-85 years in developed countries (Sakalihasan et al., 2005). This percentage is probably even higher due to underestimation of AAA related mortality, since AAAs generally exist without symptoms (Acosta et al., 2006).

In patients with an identified AAA and abdominal and/or back pain in combination with pain at palpation of the aneurysm (a so called symptomatic AAA), pending rupture of the AAA is assumed. However, evidence for a symptomatic AAA representing pending rupture is lacking (Scott et al., 2005). When rupture occurs, the mortality rate is as high as 80% (Semmens et al., 2000; Veith et al., 2003; Gorham et al., 2004). Forty percent of the patients with a ruptured AAA do not reach the hospital alive (Semmens et al., 2000) and in patients reaching the hospital and undergoing surgery, the mortality rate is approximately 50% (Sayers et al., 1997). Despite progression in surgical techniques, anaesthetical management, vascular prostheses and perioperative care, there is only a gradual decline in operative mortality rate over the past decades (Heller et al., 2000; Bown et al., 2002).

In 1991, a new minimally invasive technique was described by Parodi et al. to treat AAA, endovascular aneurysm repair (EVAR) (Parodi et al., 1991). In the elective setting, EVAR showed an absolute and relative mortality risk reduction of approximately 3 and 75%, respectively (Prinssen et al., 2004; EVAR-trial-participants 2005). In the acute setting, emergency EVAR (eEVAR) is a strategy that might allow for improvement in above mentioned poor prognosis. Since 1994 an increasing amount of publications of eEVAR to treat acute AAAs is published. Currently, eEVAR has become an accepted treatment option which is increasingly being performed to treat acute AAA. However, the potential reduction in peri-operative mortality of eEVAR compared to conventional open repair in patients with an acute AAA is still open to debate.

in this chapter, we will discuss the role of endovascular AAA repair in patients with a ruptured AAA.

2. Treatment options

In patients presenting with a ruptured AAA, a choice can be made whether or not to offer treatment at all (selective treatment policy). When decided to perform an intervention, two treatment options are available; conventional "open" AAA repair or the minimally invasive EndoVascular Aneurysm Repair (EVAR).

2.1 No intervention

In order to identify patients with an unrealistic expectation of a successful outcome after surgery, operative risk predictors, comorbidities and estimated quality of life can be assessed. However, excluding selected patients from treatment is an awkward consideration (Hardman et al., 1996; Tambyraja et al., 2008), which is signified by the number of prediction models generated for risk stratification to support improvement of patient selection for surgical intervention (Samy et al., 1994; Hardman et al., 1996; Prytherch et al., 2001). The 'Hardman Index' and 'Glasgow Aneurysm Score' are the most commonly used prognostic scoring systems. The Hardman Index identifies five independent preoperative factors associated with mortality; age, blood creatinine level, loss of consciousness after arrival, blood haemoglobin level and electrocardiographic ischemia (Hardman et al., 1996). The Glasgow Aneurysm Score uses the following factors: age, shock, myocardial disease, cerebrovascular disease and renal disease (Samy et al., 1994). The validity of both scoring systems has been assessed using 82 patients in the study of Tambyraja et al. from the year 2005 (Tambyraja et al., 2005). Unfortunately, both scoring systems seemed to be poor predictors for postoperative mortality in patients with a ruptured AAA. Two years later, Tambyraja et al. identified three risk factors which might form the basis of a new scoring system to predict the outcome of rAAA, the 'Edinburgh Ruptured Aneurysm Score' (Tambyraja et al., 2007). Risk factors were: blood haemoglobin level, blood pressure, and Glasgow Coma Scale. Until this moment validation studies are still needed in order to assess its predictive value and clinical applicability.

Due to modest validity and clinical applicability of present prognostic scoring systems, selecting patients for intervention remains a subjective consideration. Whenever possible, patients' and families' opinion as well as the opinion of the responsible medical doctor has to be included in the decision.

2.2 Conventional 'open' ruptured AAA repair

Conventional open repair of an AAA was performed for the first time in 1951, replacing the abdominal aortic aneurysm by a homograft (Dubost et al., 1951). Two years later, open repair was performed using synthetic grafts (DeBakey & Cooley 1953). The open procedure to treat ruptured as well as unruptured AAA has almost been consistent over time and known as being an invasive, but generally durable procedure. In patients who are often suffering from considerable hypovolemic shock, a laparotomy is performed immediately after induction of general anaesthesia. Subsequently, the aorta and/or iliacal arteries are clamped proximally and distally from the aneurysm. After clamping, the aneurysm is opened in order to provide access for placement of a polyester tube or bifurcated graft. The aneurysm sac is left in situ and secured around the graft in order to cover it.

This major operation carries a significant mortality and morbidity, due to the combined effects of general anaesthesia, surgical exposure, haemorrhage, and aortic clamping with related lower torso ischaemia-reperfusion injury (Dillon et al., 2007). General anaesthesia is required which might lead to acute haemodynamical changes as a result of associated

inhibition of sympathetic arterial tone. The hypotension and subsequent inadequate oxygenation might induce or accelerate cerebral en cardiac ischemia, resulting in a poor clinical prognosis. Furthermore, loss of abdominal muscle tone can occur during the induction of general anaesthesia which might cause free rupture of the retroperitoneal haematoma with related haemodynamical consequences (Lachat et al., 2002). During surgical exposure, blood loss is generally extensive (Sadat et al., 2008). Hypotension and subsequent inadequate oxygenation might induce or accelerate cerebral en cardiac ischemia, resulting in poor clinical prognosis. Furthermore, after removing the clamps, considerable ischemia-reperfusion injury of the lower extremities and the intra-abdominal organs might occur (Bown et al., 2003).

2.3 Minimally invasive endovascular ruptured AAA repair

In 1991, Parodi et al described a less invasive alternative to conventional 'open' aneurysm repair for the treatment of AAA, Endovascular Aneurysm Repair (EVAR) (Parodi et al., 1991). EVAR involves groin incisions in order to expose the femoral arteries. Using a catheter and guidewire a synthetic stentgraft is fed through the artery up to the AAA neck until positioned correctly just below the renal arteries and subsequently unfolded, excluding the aneurysm sac from blood flow and pressure. Control angiography is performed to assure correct placement of the endovascular stentgraft. Aorto-uni-iliac stentgrafts, reaching one of the common iliac arteries as well as bifurcated stentgrafts, reaching both iliac arteries are available. In case of aorto-uni-iliac stentgrafting, femoro-femoral bypass graft surgery has to be performed in order to restore blood flow to the contralateral leg. A controlateral endovascular occluder is used to stop retrograde bleeding up into the iliac artery into the aneurysm sac. Due to increasing expertise and continuous improvement of both stentgrafts and their delivery systems, increasing success rates and decreasing complications and reintervention rates are observed (Lovegrove et al., 2008).

After several years of experience in EVAR for unruptured AAAs this technique has gradually extended its indication and is currently used to treat feasible patients with a ruptured AAA (Yusuf et al., 1994). However, the applicability for EVAR depends on several anatomical and logistic conditions. Anatomical suitability for EVAR is assessed on a preoperative CTA scan and evaluated for infrarenal aortic neck length, neck angulation and iliac and femoral access arteries that need to be large enough to accommodate the introducer system (Kapma et al., 2005). Approximately half of the ruptured AAAs is considered anatomically suitable for eEVAR according to the preoperative CTA scan (Hoornweg et al., 2007). However, logistic problems are often reported which frequently led to the exclusion of EVAR-suitable patients for undergoing endovascular repair (Yilmaz et al., 2002; Reichart et al., 2003; Kapma et al., 2005; Franks et al., 2006; Peppelenbosch et al., 2006; Visser et al., 2006; Acosta et al., 2007). Logistic criteria for EVAR in patients with a ruptured AAA are the instant availability of a CT-scanner, the 24/7 availability of an operating room that is adequately equipped to perform endovascular procedures as well as an endovascular trained staff. Financial burden is sometimes the availability of a large variety of 'off-the-shelf' stent-grafts (Mehta et al., 2006).

3. EVAR versus open surgery

In a recent systematic review of 61 controlled and uncontrolled clinical studies of patients with an unruptured AAA, EVAR is described as a feasible and safe technique, showing

decreased mortality and morbidity rates compared to a conventional open procedure (Drury et al., 2005). Considering these benefits, EVAR has been generally accepted as the preferred treatment option.

Since its first description in 1994 by Yusuf et al (Yusuf et al., 1994), over 400 reports of EVAR for patients with a ruptured AAA are available. The minimal invasive approach implies the opportunity to use local anaesthesia, which has been proven to be feasible and effective in EVAR (Henretta et al., 1999; Bettex et al., 2001). As described by Lachat et al in 2002, local anaesthesia is not attended with the acute haemodynamical changes which are normally seen during induction of general anaesthesia (Lachat et al., 2002). However, these benefits did not lead to standard application of local anaesthesia, since 19 comparative observational studies show considerable variation in the percentages of patients undergoing local anaesthesia (0-97%). Furthermore, eEVAR involves no crossclamping and minor surgical exposition compared to open surgery.

The above mentioned advantageous consequences of the minimally invasive endovascular approach of acute AAA might reflect on perioperative mortality. Approximately 26 studies comparing EVAR with conventional open surgery in patients with a ruptured AAA can be identified (Ohki & Veith 2000; van Sambeek et al., 2002; Verhoeven et al., 2002; Yilmaz et al., 2002; Reichart et al., 2003; Resch et al., 2003; Lee et al., 2004; Alsac et al., 2005; Brandt et al., 2005; Kapma et al., 2005; Larzon et al., 2005; Vaddineni et al., 2005; Arya et al., 2006; Coppi et al., 2006; Dalainas et al., 2006; Dillavou et al., 2006; Franks et al., 2006; Greco et al., 2006; Hinchliffe et al., 2006; Peppelenbosch et al., 2006; Visser et al., 2006; Acosta et al., 2007; Anain et al., 2007; Moore et al., 2007; Ockert et al., 2007; Egorova et al., 2008). Twenty-four of these studies compared early mortality of EVAR compared to open surgery (Ohki & Veith 2000; van Sambeek et al., 2002; Verhoeven et al., 2002; Yilmaz et al., 2002; Reichart et al., 2003; Resch et al., 2003; Lee et al., 2004; Alsac et al., 2005; Brandt et al., 2005; Kapma et al., 2005; Larzon et al., 2005; Vaddineni et al., 2005; Arya et al., 2006; Coppi et al., 2006; Dalainas et al., 2006; Franks et al., 2006; Greco et al., 2006; Hinchliffe et al., 2006; Peppelenbosch et al., 2006; Visser et al., 2006; Acosta et al., 2007; Anain et al., 2007; Moore et al., 2007; Ockert et al., 2007). One of these studies is a prospective randomised trial by Hinchliffe et al, which showed identical 30-day mortality rates in both treatment groups (9/17 in the open surgery group versus 8/15 in the EVAR group) (Hinchliffe et al., 2006). However, the study is underpowered and served as a pilot study for future randomised studies. The remaining 23 studies are observational studies of which 4 showed no reduction in early mortality compared to open surgery (Ohki & Veith 2000; van Sambeek et al., 2002; Hinchliffe et al., 2006; Ockert et al., 2007). Using Review Manager 4.2.10, provided by the Nordic Cochrane centre, a forest-plots can be created (Figure 1). The overall effect of EVAR compared to open surgery, taking 1 randomised controlled trial and 23 available observational studies into account, showed a 38% decrease in 30-day or hospital mortality rate (peto-odds ratio 0.62; 95% CI 0.52 to 0.74).

Additionally, the 30-day, or hospital mortality is reported in five recent systematic reviews (Table 1) (Harkin et al., 2007; Visser et al., 2007; Mastracci et al., 2008; Rayt et al., 2008; Sadat et al., 2008). Two reviews only discuss the results of the endovascular procedure (Mastracci et al., 2008; Rayt et al., 2008) and three reviews compared the endovascular with the open procedure (Harkin et al., 2007; Visser et al., 2007; Sadat et al., 2008). The first review showed a pooled mortality rate after EVAR of 24% (95% CI 20-28%) across 31 studies concerning 982 patients (Rayt et al., 2008). In 18 observational studies describing 436 people who underwent EVAR, the second review found a pooled mortality of 21% (95% CI 13-29%) (Mastracci et al., 2008). According to two reviews comparing both treatment groups, pooled mortality is 18%

(Harkin et al., 2007) and 22% (Visser et al., 2007) in the EVAR group compared to 34% (Harkin et al., 2007) and 38% (Visser et al., 2007) in the open surgery group. In the fifth review, Sadat et al showed that EVAR is associated with a significant reduction in mortality with a pooled odds ratio of 0.62 (95% CI 0.52-0.75) (Sadat et al., 2008). Visser et al found similar results with an odds ratio of 0.45 (95% BI 0.28-0.72) (Visser et al., 2007). However, after adjustment for patients' hemodynamic condition, the odds ratio was 0.67 (95% CI 0.31-1.44) and therefore no longer significant.
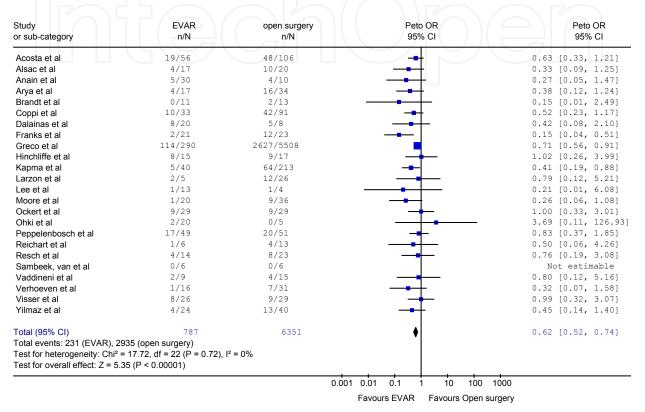
Study or sub-category	EVAR n/N	open surgery n/N	Peto OR 95% CI	Peto OR 95% CI
Acosta et al	19/56	48/106		0.63 [0.33, 1.21]
Alsac et al	4/17	10/20		0.33 [0.09, 1.25]
Anain et al	5/30	4/10		0.27 [0.05, 1.47]
Arya et al	4/17	16/34		0.38 [0.12, 1.24]
Brandt et al	0/11	2/13		0.15 [0.01, 2.49]
Coppi et al	10/33	42/91		0.52 [0.23, 1.17]
Dalainas et al	8/20	5/8		0.42 [0.08, 2.10]
Franks et al	2/21	12/23		0.15 [0.04, 0.51]
Greco et al	114/290	2627/5508		0.71 [0.56, 0.91]
Hinchliffe et al	8/15	9/17		1.02 [0.26, 3.99]
Kapma et al	5/40	64/213		0.41 [0.19, 0.88]
Larzon et al	2/5	12/26		0.79 [0.12, 5.21]
Lee et al	1/13	1/4		0.21 [0.01, 6.08]
Moore et al	1/20	9/36		0.26 [0.06, 1.08]
Ockert et al	9/29	9/29		1.00 [0.33, 3.01]
Ohki et al	2/20	0/5		3.69 [0.11, 126.93]
Peppelenbosch et al	17/49	20/51		0.83 [0.37, 1.85]
Reichart et al	1/6	4/13		0.50 [0.06, 4.26]
Resch et al	4/14	8/23		0.76 [0.19, 3.08]
Sambeek, van et al	0/6	0/6		Not estimable
Vaddineni et al	2/9	4/15		0.80 [0.12, 5.16]
Verhoeven et al	1/16	7/31		0.32 [0.07, 1.58]
Visser et al	8/26	9/29		0.99 [0.32, 3.07]
Yilmaz et al	4/24	13/40		0.45 [0.14, 1.40]
Total (95% CI)	787	6351		0.62 [0.52, 0.74]

Total events: 231 (EVAR), 2935 (open surgery)
Test for heterogeneity: Chi² = 17.72, df = 22 (P = 0.72), I² = 0%
Test for overall effect: Z = 5.35 (P < 0.00001)

0.001 0.01 0.1 1 10 100 1000
Favours EVAR   Favours Open surgery

Fig. 1. Forest plot of 30-day or hospital mortality in 24 studies comparing EVAR and open surgery in patients with a ruptured AAA.

Review	Studies n	Patients n	30-day/in-hospital mortality		
			EVAR % (95% CI)	Open % (95% CI)	Odds ratio EVAR vs Open
Rayt et al., 2008	31	982	24% (20-28)	-	-
Mastracci et al., 2008	18	436	21% (13-29)	-	-
Harkin et al., 2007	34	891	18% (0-53) *	34% (0-70) *	-
Visser et al., 2007	10	478	22% (16-29)	38% (32-45)	0.45 (0.28-0.78)
Sadat et al., 2008	23	7040	-	-	0.62 (0.52-0.75)

CI = confidence interval, vs = versus, * = % (range of included studies)

Table 1. 30-day or in-hospital mortality in patients treated with open or endovascular repair according to five systematic reviews.

In addition, the systematic reviews showed that EVAR is associated with significant reduction in blood loss, reduced procedure time, reduction in systemical complications and

reduced intensive care and hospital stay compared to open surgery (Harkin et al., 2007; Visser et al., 2007; Mastracci et al., 2008; Sadat et al., 2008).

4. Discussion

Theoretically, both the endovascular and the conventional open technique have benefits. During open repair the aorta is clamped short after the initiation of the procedure, ceasing the blood loss. During endovascular repair on the other hand, the ruptured aneurysm remains part of the circulation until the entire endograft is deployed and correctly positioned without major endoleak.

Reported results of reduced early mortality after EVAR for the treatment of a ruptured AAA compared to open surgery seems conclusive (table 1). However, the currently available, mainly observational, studies are small and add considerable heterogeneity and methodological limitations (Yilmaz et al., 2002; Reichart et al., 2003; Resch et al., 2003; Lee et al., 2004; Alsac et al., 2005; Brandt et al., 2005; Castelli et al., 2005; Hechelhammer et al., 2005; Kapma et al., 2005; Larzon et al., 2005; Vaddineni et al., 2005; Arya et al., 2006; Coppi et al., 2006; Franks et al., 2006; Hinchliffe et al., 2006; Peppelenbosch et al., 2006; Visser et al., 2006; Acosta et al., 2007; Ockert et al., 2007). Heterogeneity is signified by the broad range in percentages of patients treated with EVAR (15-50%) and in percentage of haemodynamical unstable patients (33-73% in the eEVAR group). Even the definition of haemodynamical instability varied between the studies from a systolic blood pressure below 50 mmHg to 100 mmHg. Furthermore, the comparative studies reported so far are flawed by methodological inadequacies such as high potential of selection bias and lack of randomisation (Dillon et al., 2007). Selection bias is created by selecting patients for EVAR constituting a lower-risk category, presuming they need to be haemodynamically more stable for preoperative imaging and have a more favourable (EVAR-suitable) anatomic configuration. In a previous report, though not randomized, we eliminated selection bias due to inadequate patient matching by reporting a comparison of EVAR and open surgery in patients who all had the same preoperative imaging protocol, irrespective of haemodynamic condition, and who were all anatomically suitable for EVAR (Ten Bosch et al., 2010). This study showed a significant reduction in 30-day and 6-month mortality of EVAR compared to open ruptured AAA repair. However, a larger conducted prospective randomised trial such as the Amsterdam Acute Aneurysm Trial, which is currently performed in the Netherlands, is needed to identify possible benefits of EVAR over open surgery in patients with a ruptured AAA. The pilot study of Hinchliffe et al showed the possibility to recruit patients with a ruptured AAA to a randomised trial of open surgery and EVAR (Hinchliffe et al., 2006). However, a RCT might give ethical concerns, given the accumulation of superior results with EVAR based on the available observational studies. In addition, a RCT in an acute, severe condition like a ruptured AAA, appears difficult to perform (Hinchliffe et al., 2006). Furthermore, long term effects on outcome still need further investigation.

In case randomised trials demonstrate a clinically relevant reduction in mortality and morbidity for endovascular repair, consequences for care organisation will be major. Treatment of ruptured AAAs has to be performed in hospitals that are able to guarantee permanent availability of endovascular trained staff, implicating regionalisation and centralisation of acute AAA care.

5. Conclusion

The minimally invasive endovascular procedure (EVAR) is theoretically likely to reduce early mortality in patients with a ruptured AAA. The majority of observational studies show a clear trend toward an improved short term effect of EVAR and a significant reduction in early mortality compared to conventional open surgery. Therefore, EVAR has become a generally accepted treatment option for ruptured AAAs. However, studies comparing EVAR with conventional open surgery have to be interpreted with caution due to the likelihood of methodological inadequacies such as selection bias, heterogeneity, and lack of randomisation. Can endovascular repair of the ruptured AAA be considered as the treatment option of first choice? This question has not been answered yet. Further research in terms of randomised controlled trials with adequate follow-up will be required in order to clarify the role of endovascular repair as treatment option for ruptured abdominal aortic aneurysms.

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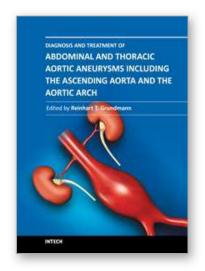
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Diagnosis and Treatment of Abdominal and Thoracic Aortic Aneurysms Including the Ascending Aorta and the Aortic Arch
Edited by Prof. Reinhart Grundmann

This book considers diagnosis and treatment of abdominal and thoracic aortic aneurysms. It addresses vascular and cardiothoracic surgeons and interventional radiologists, but also anyone engaged in vascular medicine. The book focuses amongst other things on operations in the ascending aorta and the aortic arch. Surgical procedures in this area have received increasing attention in the last few years and have been subjected to several modifications. Especially the development of interventional radiological endovascular techniques that reduce the invasive nature of surgery as well as complication rates led to rapid advancements. Thoracoabdominal aortic aneurysm (TAAA) repair still remains a challenging operation since it necessitates extended exposure of the aorta and reimplantation of the vital aortic branches. Among possible postoperative complications, spinal cord injury (SCI) seems one of the most formidable morbidities. Strategies for TAAA repair and the best and most reasonable approach to prevent SCI after TAAA repair are presented.

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