Management of small asymptomatic abdominal aortic aneurysms – a review

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The approach for abdominal aortic aneurysms (AAAs) larger than 55 mm is well defined due to the risk of rupture being higher than 10% per year, and a 30-day perioperative mortality rate between 2.5% and 5%. However, the approach for small asymptomatic AAAs is less well defined.

There are different definitions given to describe a small AAA. The one the authors accepted and applied is "a localized, permanent and irreversible dilation of the aorta of at least 50% in relation to the normal adjacent infrarenal or suprarenal aorta, with a maximum diameter between 30-55 mm".

The investigators of the largest study on small AAAs (United Kingdom Small Aneurysm Trial [UK-SAT]) concluded, in brief, that ultrasound monitoring is the most appropriate solution because the results do not support a policy of surgical restoration for AAAs with a diameter of between 40 mm and 55 mm.

The aim of the present review article is to highlight several challenges that could change the limits or create a more flexible deciding factor in the management of AAAs. There are multiple factors that influence surgical decision-making, and the limit on aneurysm diameter that indicates surgery should depend on the patient's age, life expectancy, general status, associated diseases, diameter in relation to body mass, risk factors, sex, anxiety and compliance during the follow-up period. Monitoring is an acceptable alternative for AAAs between 40 mm and 55 mm, and is probably the best solution for high-risk patients. Surgery is the most reasonable solution for patients who are at moderate risk, have a significant life expectancy, are less than 70 to 75 years of age, and/or have aortic aneurysms larger than 50 mm.

Key Words: *Mortality rate; Operative risk; Rupture risk; Small AAA; Ultrasound*

The natural evolution of an abdominal aortic aneurysm (AAA) leads to growth and rupture. The risk is determined by the maximum aortic diameter. The relationship between the risk of rupture and the diameter describes an exponential curve.

The approach for AAAs larger than 55 mm is well defined. The risk of rupture is higher than 10% for each year the aneurysm exists, while perioperative mortality after 30 days varies between 2.5% and 5% (1). However, the approach for small asymptomatic AAAs is less well defined. If we refer to the conclusions of the authors of the United Kingdom Small Aneurysm Trial (UK-SAT) (2,3):

- AAAs between 30 mm and 40 mm require ultrasound (US) monitoring every 12 months and, in the case of an annual growth rate higher than 10%, every six months;
- AAAs between 40 mm and 55 mm require US monitoring every three to six months, with possible recourse to surgery if certain requirements are met.

The authors of the above study concluded in 1998 (2) and 2002 (3) that the "results do not support a policy of surgical restoration for abdominal aortic aneurysms with a diameter between 40-55 mm". Consequently, because surgery is not beneficial for 'small' AAAs, with a maximum diameter of between 40 mm and 55 mm, monitoring remains the most adequate solution. But are things that simple?

WHAT IS A SMALL AAA?

The normal inter-renal aorta has a mean diameter of 21 mm in men and 18 mm in women. Although there is no unanimity, the definition of a small AAA is "a localized, permanent and irreversible dilation of the aorta of at least 50% in relation to the normal adjacent infrarenal or suprarenal aorta, with a maximum diameter ranging between 30-55 mm" (4).

Other definitions have also been proposed (5):

- A ratio higher than 1.5 between the transverse or anteroposterior diameter of the infrarenal aorta, and the diameter of the suprarenal aorta;
- A maximum transverse or anteroposterior diameter of the infrarenal aorta greater than 30 mm;
- A maximum transverse or anteroposterior diameter of the infrarenal aorta greater than 40 mm;
- The maximum diameter of the infrarenal aorta is at least 5 mm greater than the maximum diameter of the aorta between the origin of the inferior mesenteric artery and the origin of the left renal artery;
- Any aortic dilation located between the diaphragm and the aortic bifurcation;
- Any localized aortic dilation with a diameter at least twice the diameter of the superjacent aorta; and

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TABLE 1
Normal diameter of the abdominal aorta related to segments and sex

	Aortic segment diameter (cm)		
Sex	**Supraceliac**	**Suprarenal**	**Infrarenal**
Men	2.50–2.72	1.98–2.27	1.41–2.39
Women	2.10–2.31	1.86–1.88	1.19–2.16

Values presented as a normal range. Data reproduced from reference 6

TABLE 2
Normal diameter of the abdominal aorta related to age and sex

	Age group normal diameter (mm)				
Sex	**40–45**	**46–50**	**51–55**	**56–60**	**Total**
Men	19.1±2.1	19.9±1.7	20.5±2.2	21.7±3.1	20.2±2.5
Women	17.1±1.4	17.0±1.4	17.1±1.3	17.0±1.6	17.0±1.5

Data presented as mean ± SD. Data reproduced from reference 43

TABLE 3
Correlation and analyses of aortic diameter variance in men

Variable	r^2	P	F ratio	P
Age	0.358	<0.001	15.7	<0.001
Height	0.027	NS	11.0	0.001
BMI	0.142	0.002	6.2	0.013
SP	0.165	<0.001	0.16	NS
DP	0.117	0.009	3.4	NS
Cholesterol	0.083	NS	0.4	NS
HDL	0.001	NS	0.9	NS
Triglycerides	0.080	NS	0.6	NS
Wall calcification	0.427	<0.001	23.7	<0.001

BMI Body mass index; DP Diastolic blood pressure; HDL High-density lipoprotein; NS Not significant; SP Systolic blood pressure. Data reproduced from reference 43

- The maximum anteroposterior diameter of the infrarenal aorta is at least 5 mm greater than the maximum diameter of the suprarenal aorta.

All these definitions highlight the problem of the method used to establish the diameter of the aorta. In the UK-SAT study, the maximum anteroposterior diameter was evaluated using US, with a tolerance of ±2 mm. The following four observations can be made:

1. US is an operator-dependent examination. The difficulty in accurately assessing the dimensions of the aorta, especially in obese patients or in the presence of marked meteorism, should not be minimized. Because of the limitations inherent to this method, the establishment of a strict 55 mm limit for the determination of the surgical indication seems unrealistic.

2. There is no consensus based on anatomical or anatomoclinical grounds for the use of the anteroposterior, transverse or maximum diameter. In the literature, as well as in current practice, the three diameters are used at random.

3. The evaluation of the diameter of the abdominal aorta varies depending on the method used. Since 1991, the standards reported by North American societies have reflected this variability as well as the differences noted depending on the measurement method (6). Sprouse et al (7) demonstrated that the maximum diameter of AAAs measured by computed tomography (CT) is significantly greater than the same diameter measured by US. This study showed that, in 95% of cases, CT measurement gives superior results compared with US measurement – the mean difference is 1 mm. Another study (8) produced similar results, but the difference was even higher, reaching 3.9 mm. This difference can be explained by the nonperpendicular incidence of CT sections on an aorta, presenting a degree of angulation. Elkouri et al (9) reported a feasibility rate of US measurements of 54% for patients with a body mass index higher than 30 kg/m^2, and 81% for patients with a body mass index lower than 30 kg/m^2. Also, a mean difference of 2.9 mm for the anteroposterior diameter and 1.8 mm for the transverse diameter is noted in favour of CT. The *Association Française de Formation Continue en Angiologie* (AFFCA) study (10), performed in 80 patients for the comparison of US, CT and intraoperative data, demonstrated that US underestimated the anteroposterior diameter of the aneurysm by approximately 2.16 mm, the transverse diameter by 4.29 mm, the anteroposterior diameter of the lumen of the circulatory canal by 5.54 mm, and the diameter of the upper neck by 2.74 mm. At the same time, there were no significant differences between CT and intraoperative measurements.

4. An absolute value arbitrarily set at 55 mm does not take into account the increase in diameter in relation to the superjacent aorta. Sonneson et al (11) demonstrated a constant increase in aortic diameter of approximately 24% between 25 and 70 years of age. The same author observed a significant difference in aortic diameter between the two sexes after 25 years of age. The measurements performed for the determination of the diameter of the infrarenal aorta in healthy subjects (12) confirmed the presence of a significant correlation between the aortic diameter and the patient's weight ($r=0.84$, $P<0.001$), height ($r=0.77$, $P<0.001$) and body surface ($r=0.83$, $P<0.001$). Age and body surface area are the factors that influence aortic diameter the most, and the diameter of the suprarenal aorta is strongly influenced by these parameters (13). Another study (14) performed in 906 men between 65 and 74 years of age showed significant correlations between the maximum aortic diameter, and age and height, and insignificant correlations for body weight. Aortic diameter in the female sex is certainly smaller than in the male sex (15). The morphological explanation is evident in the body surface difference. Finally, it seems that the mean diameter of the normal aorta is greater in black subjects (16). The data for normal aortic diameter are synthesized in Tables 1 to 4.

All these data show the relative character of the supposedly 'normal' measurements and the lack of realism in establishing

TABLE 4
Correlation and analyses of aortic diameter variance in women

Variable	r^2	P	F ratio	P
Age	0.002	NS	1.2	NS
Height	0.236	<0.001	37.2	<0.001
BMI	0.172	<0.001	21.0	<0.001
SP	0.060	NS	2.6	NS
DP	0.149	0.001	11.6	0.001
Cholesterol	–0.057	NS	0.000	NS
HDL	–0.025	–0.141	0.2	NS
Triglycerides	–0.014	NS	5.8	0.016
Wall calcification	0.012	NS	0.1	NS

BMI Body mass index; DP Diastolic blood pressure; HDL High-density lipoprotein; NS Not significant; SP Systolic blood pressure. Data reproduced from reference 17

an absolute threshold. The following question can then be posed: are we going to adopt the same approach for a 48 mm AAA in a 75-year-old man (height 185 cm, weight 100 kg) and a 52 mm AAA in a 50-year-old woman (height 155 cm, weight 50 kg)? This scenario is not fiction, and is likely to be found in practice.

IS EVIDENCE EVIDENT?

The authors of the UK-SAT study concluded in 1998 that the "results do not support the orientation toward conventional surgery for AAAs between 4 and 5.5 cm" (2,17). In 2002, after the publication of eight years of follow-up, they confirmed the above conclusion (3). Although it significantly improves knowledge in this field, the study has many weaknesses that make the conclusions excessive (18). The study was designed to be a superiority trial attempting to show a 5% difference with 80% power. The authors concluded that the two groups (surgery and US monitoring) had equal outcomes, which would have normally required an equivalence trial, not a superiority trial. On seeing the results after five years, a question could be asked: are the two groups equivalent or does the trial not have sufficient power to detect a difference? At eight years follow-up, the authors noted that mortality is 7.2% lower in the early surgery group (P=0.003). However, the conclusion is ambiguous: "In patients with a small AAA, we found no differences in mean long-term survival between the early surgery group and the monitoring group, although global mortality at eight years follow-up was significantly lower in the early surgery group" (3). The main explanation is that a low mortality rate following surgery is due to better medical care and monitoring of the postsurgical patients, which is perfectly credible. The presentation of the results leads us to another possible explanation – the trial did not have sufficient power to detect a difference after five years and this difference appeared after eight years in favour of the early surgery group.

Other weaknesses or questionable aspects of the study should be mentioned:

- In the US monitoring group, there were 150 deaths, of which 17 were attributed to rupture. Given the biopsy verification rate of only 29%, we wonder whether the number of deaths by aneurysm rupture was underestimated.

TABLE 5
Decrease in death risk by groups – five-year outcomes

	Surveillance deaths/total (n)	Early surgery deaths/total (n)	Hazard ratio*
Age (years)			
60–66	42/181	36/181	0.76
67–71	60/180	51/183	0.80
Aneurysm diameter (mm)			
49–55	52/145	51/174	0.79

**Within a 95% CI. Data reproduced from reference 2*

- Thirty-eight patients randomly assigned from the US monitoring group underwent surgery without meeting the operative indication criteria, and 43 patients randomly assigned from the surgery group did not undergo surgery. This resulted in a protocol violation rate of 7%, which is enough to change a slightly significant result.
- Although the authors deny there would be interest in an analysis by subgroup, it seems that the benefit for the surgical group is more significant when subjects are younger than 70 years of age and aortic diameter is greater than 50 mm (Table 5).
- The risk of rupture was significantly higher in the female sex, but the conclusions cannot be applied to the subgroup represented by female subjects.
- The examiners responsible for US monitoring were selected according to performance criteria, while the surgical teams were not, which raises questions about their experience and the homogeneity of the results of the surgical group.
- US monitoring was performed every three months for AAAs larger than 50 mm, without the loss of the patients' compliance. This extraordinary compliance is probably the result of enrollment and informed consent, but it does not reflect the current reality of clinical research, where patient compliance has decreased dramatically over time.

The final conclusions that can be drawn from the results of the UK-SAT study are that the benefit of immediate surgery for AAAs between 40 mm and 55 mm is reduced, and this benefit appears in the long term (it is significant at eight years and insignificant at five years) for younger subjects, women, and patients with a maximum aortic diameter greater than 50 mm.

RISK OF RUPTURE

The mortality determined by AAA rupture remains extremely high in spite of the progress achieved over the past 50 years regarding the treatment of arterial pathology. Two sof three patients die before arriving at the hospital. For the others, global mortality exceeds 50%, of which 20% die before surgery. Operative mortality remains discouraging, between 40% to 50%, in spite of all the progress achieved and the tremendous experience acquired. Fifteen thousand people die every year in the United States from ruptured AAAs, which indicates aneurysms as the 13th cause of global mortality and the 10th cause of mortality in men older than 55 years of age.

TABLE 6
FIve-year rupture rate related to abdominal aortic aneurysm (AAA) diameter

Maximum AAA diameter (cm)	Five-year rupture rate (%)
<4.0	2
4.0–4.9	3–12
5.0–5.9	25
6.0–6.9	35
≥7.0	75

Data reproduced from reference 44

TABLE 7
Annual rupture rate (%) in relation to abdominal aortic aneurysm diameter

	Diameter (cm)		
Authors, year (ref)	2.0–3.9	4.0–4.9	5.0–5.9
Nevitt et al, 1989 (45)	0	1	11
Reed et al, 1997 (46)	0	1	11
Limet et al, 1991 (21)	0	5.4	16
Guirguis and Barber, 1991 (47)	0.25	0.5	4.3

Ref Reference

The risk of rupture depends on several variables. The main risk factor is the size of the aneurysm, which was demonstrated for the first time by Szilagyi et al in 1972 (19). The rates of ruptured AAAs depending on diameter are shown in Tables 6 and 7.

For Ouriel et al (20), the most important aspect was the ratio between the maximum transverse diameter of the aneurysm and the transverse diameter of the third lumbar intervertebral disc. In this study, all ruptured aneurysms presented a ratio greater than 1, while no patient with a subunitary ratio had an AAA rupture. Because the direct relationship between rupture and the size of the aneurysm is unanimously recognized, it is important to know the growth rate of small aneurysms. Two growth patterns have been described:

- The triphasic (exponential) pattern is the most frequent. A first rest or quiet phase is noted, which can last for years; it is characterized by extremely slow growth, almost paralleling aging. This is followed by a slow growth phase, which lasts for months or years. This is characteristic of patients with small AAAs between 30 mm and 45 mm under monitoring. This phase is followed by a critical moment, a "turning point" according to Limet et al (21), continuing with a third rapid growth phase, with a considerable increase in the risk of rupture.
- The linear pattern is less frequent, during which the increase in diameter is equal over time, without the identification of a critical moment.

In a study published in 1985, Cronenwett et al (22) stated that the growth rate of small diameter aneurysms (3 cm to 4 cm) is more rapid in the transverse diameter (7.9 mm/year) than in the anteroposterior diameter (1.9 mm/year). The mean annual growth rate for AAAs between 4 cm and 6 cm is estimated to be 11% by Limet et al (21), a value identical to that calculated by Cronenwett et al (23). Larger aneurysms have an increased tendency to expand than small aneurysms. The growth rates reported in the literature are shown in Tables 8 and 9.

TABLE 8
Growth rate for small abdominal aortic aneurysms

Author, year (reference)	Cases (n)	Follow-up (years)	Diameter (cm)	Growth/year (cm)
Bernstein and Chan, 1984 (48)	99	2.4	3.0–5.0	0.40
Cronenwett et al, 1985 (22)	67	3	3.0–4.0	0.79
Sterpetti et al, 1987 (49)	57	2.2	3.5–5.0	0.48
Litooy et al, 1989 (50)	149	10	3.5	0.79
Nevitt et al, 1989 (45)	103	NS	NS	0.21
Cronenwett et al, 1990 (23)	73	3	4.0	3.10
Limet et al, 1991 (21)	114	2.2	<4.0	0.53
			<5.0	0.69
			>5.0	0.75

NS Not significant

TABLE 9
Growth rate for abdominal aortic aneurysms

Initial aortic diameter (cm)	Mean growth rate (cm/year)	95% CI
3.0–3.9	0.39	0.20–0.57
4.0–4.9	0.36	0.21–0.50
5.0–5.9	0.43	0.27–0.60
6.0–6.9	0.64	0.16–1.10

Data reproduced from reference 44

A study published in 2002 (24) on aneurysms between 30 mm and 39 mm showed a mean annual global growth rate of 0.11 cm. No rupture was detected during the study, but the growth rate was significantly different for AAAs between 30 mm and 34 mm, and those between 35 mm and 39 mm.

The simple observation that not all aneurysms rupture when they reach a certain size leads to the idea that there are other variables that influence this risk. Although variables such as the thickness or the resistance of the aortic wall are known to play an important direct role in the risk of rupture, the current technical impossibility of quantifying them prevents their use in current practice.

Arterial hypertension, diastolic hypertension in particular, considerably increases the risk of rupture, as Foster et al (25) demonstrated. In this study, 72% of the patients who died from a ruptured AAA were hypertensive. Szilagyi et al (19) determined that 67% of patients with a ruptured AAA were hypertensive, compared with 23% of patients with an unruptured AAA.

Chronic obstructive pulmonary disease, bronchiectasia and pulmonary emphysema are other independent parameters predictive of the risk of rupture. In this sense, Sterpetti et al (26) presented a study in which the incidence of emphysema was

67% in patients who died from a rupture versus 42% in patients without a rupture. This can be explaned by a proteinase imbalance that concomitantly affects pulmonary and aortic connective tissue.

Smoking is another important risk factor for ruptured AAAs, alone or in association with chronic obstructive pulmonary disease. The risk of rupture is approximately five times higher in smokers than in nonsmokers – 2.4 times higher for cigar smokers, 4.6 times higher for cigarette smokers and 14.6 times higher for hand-rolled cigarette smokers (27).

The presence of a family history of AAA also increases the risk of rupture. Darling et al (28) found an increase in the frequency of ruptured AAAs depending on the number of first-degree relatives with AAAs – 15% for two relatives, 29% for three relatives and 36% for four relatives. In addition, the presence of a family history of AAA determined a 10-year reduction in the mean age at which the AAA rupture occurs.

Aneurysms with an eccentric sacciform development show a higher risk of rupture than fusiform aneurysms. The more dissymmetric the dilation, the higher the aortic wall tension, which is demonstrated by computer simulations. The type of dilation is the second most important risk factor for rupture, after the diameter of the aneurysm (29).

The authors of the UK-SAT study (30) analyzed the risk of rupture in 2257 patients (1090 randomly assigned and 1167 nonrandomly assigned patients), of which 103 had a rupture during follow-up. Of these, 26 patients (25%) died before arriving at the hospital, 53 patients (51%) died before surgery, 13 (13%) died within 30 days postoperatively and 11 (11%) survived. Parameters that significantly correlated with the rupture of the aneurysm included female sex, initial diameter, maximal forced expiratory volume per 1 s and arterial blood pressure.

MONITORING OF SMALL AAAs

The most widely used method for the monitoring of AAAs is mode B US. The weaknesses of this method are its operator-dependent character and the limitations given by the patient's morphotype (obesity, meteorism). CT is more accurate, but also more expensive, and subject to a certain risk caused by repeated radiation, and allergy or renal impairment from the injected iodine contrast product.

Patient compliance plays a particularly important role in monitoring the evolution of small AAAs. Valentine et al (31) performed a study of 110 patients with small AAAs. They showed that only 69% of patients were really compliant over the duration of the study. Of noncompliant patients, 10% presented with a ruptured aortic aneurysm. The incomplete understanding of the situation, correlated with education level and socioeconomic status, represented the main cause of patient noncompliance.

US or CT monitoring examinations are performed at intervals varying from three to 12 months, depending on the study and the initial diameter observed. These time intervals are set relatively arbitrarily. They should not be too short, so as not to reduce patient compliance, or too long, so as not to increase the risk of a fatal accident. Several studies have chosen time intervals varying between three mosnths (3) and 24 months (32), but the optimum interval seems to be six months. The algorithm proposed by Vardulaki et al (32) is the following: aortic diameter of 40 mm or less, monitoring every 24 months; between 41 mm and 45 mm, monitoring every 12 months; between 45 mm and 50 mm, monitoring every six months; and 50 mm or greater, monitoring every three months.

The goal of monitoring programs is not to establish the ideal time interval, but to identify the specific growth pattern for each aneurysm.

Current research focuses on two main directions: the biochemistry of the aortic wall and biomechanical modelling. The starting point was the finding that rupture was possible even in the case of small aneurysms, and that some operated aneurysms would probably not have ruptured in the absence of surgery. The first research direction attempts to identify a biochemical marker of the aneurysm growth rate and to establish correlations between its blood level and the risk of rupture. The reduced inhibitory capacity of proteinases in patients with AAAs is associated with high elastolytic activity, which results in the degradation of the aortic wall matrix and the expansion of the aneurysm. This low inhibitory capacity varies depending on aortic diameter and is restored to normal following surgical treatment (33).

Other studies have shown the role of proteinases in the degradation of the aortic wall. The plasma level of proteinases represents a predictive factor of the growth of the aneurysm (34). AAA expansion is also correlated with the plasma levels of plasmin-antiplasmin complexes; plasmin is a common activator of the proteolytic systems involved in the development of AAAs (35). Their predictive value would be similar to the best predictive serological factor known – serum elastin peptides. The responsible proteolytic systems are serine-dependent proteases (high elastase levels in AAAs), cysteine-dependent proteases (low cysteine C levels in AAAs, not correlated with the growth rate) and metal-dependent proteases (high matrix metalloproteinase 2 and matrix metalloproteinase 9 levels, correlated with the growth rate) (36).

The macrophage migration inhibitory factor (MIF) is an inflammatory cytokine released by macrophages and activated lymphocytes. The predictive potential of the serum levels of this proinflammatory cytokine is suggested by the significant association between serum MIF levels and initial diameter, as well as between serum MIF levels and the aneurysm growth rate (37).

The second research direction focuses on biomechanical modelling. Different studies have tried to establish a correlation between the risk of rupture and a certain type of aneurysmal dilation. Starting from aortic wall tension, several geometric models have been studied depending on the characteristics of the aneurysm. Thus, sacciform dilations have a higher risk of rupture than fusiform or cylindrical dilations, which can be explained by a significantly increased wall tension in the first case. In the future, the systematic monitoring and the rigorous detection of all morphological changes, associated with computer simulations, could substantially improve the 'red area' diagnosis, in which the risk of rupture is significantly increased.

SMALL AAAs AND OPERATIVE RISK

Similar to any surgical intervention, the surgery of small aortic aneurysms is not without risks and complications. These risks are dependent on several factors – age and sex of the patient, and various associated diseases, without overlooking the experience of the surgical team and the hospital size.

Steyerberg et al (38) tried to identify the independent predictive factors that influence operative mortality for the elective

TABLE 10
Independent risk factors for surgical mortality

Risk factor	OR	95% CI
Creatinine >137 µmol/L	3.3	1.5–7.5
Congestive heart failure	2.3	1.1–5.2
Electric ischemia	2.2	1.0–5.1
Altered respiratory function	1.9	1.0–3.8
Age (by decades)	1.5	1.2–1.8
Women	1.5	0.7–3.0

Data reproduced from reference 38

surgery of AAA in general (Table 10). Among these, the most important are renal function (creatinine level above 137 µmol/L), congestive heart failure and rest ischemia. The influence of age is less important than that of the other factors – mortality in the series of octogenarians is comparable with that of younger patients.

The experience of the surgical team and the specific mortality rate of the medical institution are also important prognostic factors. Between 1985 and 1987, mortality for the elective surgery of AAA in New York state, USA (39) was 9% for surgeons who performed a maximum of five operations per year, and 4% for those who performed more than 26 operations per year. Mortality was 12% in hospitals with a maximum of five operations per year, compared with 5% in hospitals with more than 38 operations per year.

There are few studies that investigate operative risk and mortality rate in the surgery of small aortic aneurysms. Results after 30 days vary between 2.7% in the Aneurysm Detection and Management (ADAM) trial (16), 4.6% in the study by Katz and Cronenwett (40), and 5.8% in the UK-SAT study (2).

Globally, it can be considered that operative risk for small aortic aneurysms is not significantly different from the data known for AAAs in general. However, it is known that with the increase in AAA diameter, the length of the proximal neck tends to diminish, which induces a higher degree of difficulty for surgical treatment.

SMALL AAAS AND ENDOVASCULAR TREATMENT

The recent development of endovascular (EV) techniques is the logical consequence of the progress achieved in image digitization, the miniaturization of EV material, and the variety of the instruments conceived and made available to vascular surgeons. The appearance of these procedures may significantly change therapeutic strategies. Although some long-term results are not yet known, it is already known that early mortality from EV treatment is lower than that of conventional surgical treatment. This benefit is certainly more important for patients at high operative risk. The essential problem of EV treatment is related to a still relatively high rate of secondary complications, which justifies rigorous monitoring following the procedure.

The feasibility of EV treatment decreases with the increase in the diameter of the aneurysm in general. This decrease seems to be accompanied by an increase in the rate of complications, especially migration and endoleak. The explanation can be seen in the morphological evolution of the aneurysm neck as the diameter increases. In a study of 206 patients, Arko et al (41) noted the best results of EV treatment occurred in patients with small AAAs (less than 50 mm) than in those with medium (51 mm to 60 mm) or large AAAs (greater than 60 mm). With the increase in the diameter of the aneurysm, a mean 15% increase in angulation and a mean 27% diminution in the length of the upper neck for large AAAs was observed compared with small AAAs. Because the aneurysm increases transversally and longitudinally, the increase in size is accompanied by an angulation of the aneurysm neck, as well as of the iliac arteries, which become tortuous and significantly increase the complexity of EV treatment.

A recently published study (42) showed very good results in favour of EV treatment for small AAAs. In 312 patients, the authors found a 1.9% mortality rate (compared with 5.8% in the UK-SAT study). The rate of fatal ruptures was 0.2/100 patients per year (0.8/100 patients per year in the UK-SAT study) and the global mortality rate was 6.4/100 patients per year (8.3/100 patients per year in the UK-SAT study). The unequivocal conclusion is that EV treatment of AAAs between 40 mm and 55 mm significantly reduces the aneurysm-related risk of rupture and death and increases global survival. The final answer may be given by the publication of the results of the Positive Impact of Endovascular Options for Treating Aneurysms Early (PIVOTAL) study that began in July 2005. This trial is aiming to recruit 1700 patients from more than 50 surgical centres in the United States to compare the results of early elective EV treatment with those of monitoring for AAAs smaller than 50 mm.

CONCLUSION

There are multiple factors that influence surgical decision-making, and the arbitrary limit on aneurysm diameter should be removed. The patient's age, life expectancy, general status, associated diseases, risk factors and sex determine operative risk. The anxiety of the patient and of those who surround him or her, the patient's compliance during the follow-up period and especially the diameter of the aneurysm considered in relation to body mass represent factors that can influence the treatment strategy, and survival.

Monitoring is an acceptable alternative for AAAs between 40 mm and 55 mm, and is probably the best alternative for patients at high risk. Surgery is the most reasonable solution for patients at moderate risk with a significant life expectancy (ie, less than 70 to 75 years of age) and with aortic aneurysms larger than 50 mm.

The progress of EV treatment, which currently offers a benefit in terms of survival for patients at high risk, is expected to further improve results, which will allow optimum treatment for these patients.

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