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POPULATION BASED DATA

Long Term Outcome of Screen Detected Sub-Aneurysmal Aortas in 65 Year Old Men: a Single Scan After Five Years Identifies Those at Risk of Needing AAA Repair

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WHAT THIS PAPER ADDS

This contemporary large population based study with long term follow up of 65 year old men with screen detected sub-aneurysmal aorta (SAA), that is an aortic diameter of 25 – 29 mm, confirms previous findings from older and smaller studies, that a majority of SAAs eventually progress to an AAA, of which a substantial proportion require elective repair for a large AAA within 10 years. Furthermore, the results indicate that the cohort of men with SAAs at risk of later AAA repair can safely be identified by a single scan five years after their initial screening.

Objective: The epidemiology of sub-aneurysmal aortic dilatation (SAA) 25 – 29 mm is not fully understood, and the management of SAA is debated. Lack of evidence is particularly problematic in the screening setting. This study aimed to evaluate the long term outcome of men with screen detected SAAs, focusing on progression to an abdominal aortic aneurysm (AAA), and on the AAAs reaching the threshold diameter for surgical repair.
Methods: Between 2006 and 2015, all 65 year old men with a screen detected SAA in middle Sweden were re-examined with ultrasound after five and 10 years. The primary outcomes were expansion to AAA $\geq$ 30 mm and progression to AAA $\geq$ 55 mm. Secondary outcomes were risk factors for progression, repair rate, and mortality.
Results: A total of 1 020 65 year old men with a SAA were identified, of whom 940 (92.2%; 95% confidence interval 91.0 – 93.8) had follow up. The Kaplan–Meier estimated incidence of AAA $\geq$ 30 mm development after the five year follow up (which was *de facto* carried out after a mean of 4.9 years) was 65.8% (61.6 – 69.4), all $<$ 55 mm. The corresponding KM-estimated incidence after the 10 year follow up (carried out after a mean of 11.9 years) was 95.1% (90.1 – 97.4), and 29.7% (18.0 – 39.7) reached $\geq$ 55 mm. All 41 SAAs eventually expanding to $\geq$ 55 mm were $\geq$ 30 mm at the five year follow up. Of these, 32 had surgical repair with 100% survival, six have scheduled repairs, and three (7.3%) were unfit for repair. The KM estimated all cause mortality rates at five and 10 years were 7.0% and 17.9%, respectively, with no proven AAA related deaths.
Conclusion: A majority of SAAs eventually progress to an AAA, of which 30% are estimated to eventually reach the threshold for repair within 10 years. A follow up policy with an ultrasound examination after five years can safely and effectively identify those SAAs at risk of developing into clinically significant AAAs needing repair and may be considered for anyone with reasonably good life expectancy.

Keywords: Abdominal aortic aneurysm, Ectatic aorta, Prevention and control, Screening, Subaneurysmal aorta, Ultrasound
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INTRODUCTION

Abdominal aortic aneurysm (AAA) is a significant health issue, and ruptured AAA is a common cause of death in Europe and North America. To prevent rupture, early

detection, and preventive surgical repair in selected individuals is recommended.[1] AAA screening programmes have been implemented in several countries. With the expanding number of screening programmes, a substantial number of individuals with borderline infrarenal aortic dilatations of just below 30 mm will be detected, and there are uncertainties over how to manage them.[2,3] Many screening programmes exclude these individuals from further follow up, which is supported by several studies, indicating that aneurysm related mortality in people with an aortic diameter $<$ 30 mm is rare.[4,5] However, emerging evidence suggests that the subgroup of aortas measuring 25 — 29 mm in diameter, termed sub-aneurysmal aortic dilatation (SAA), do not represent a normal aorta and that many of those eventually will develop into an AAA,[6–9] of which some will reach the threshold for surgical repair or rupture.[10–12] There is only a limited body of evidence regarding benefits or harms of including persons with SAA into a surveillance programme.[13] Several aspects of SAA need further research, such as its clinical relevance and the long term effects and cost effectiveness of surveillance.[1]

This population based cohort study aimed to evaluate the long term natural course of men with a screen detected SAA regarding development to an AAA, with a focus on the proportion progressing to the threshold diameter for surgical repair ($\geq$ 55 mm).

METHODS

All men attending a general ultrasound based AAA screening programme between one January 2006 and 31 December 2014, at the age of 65 years (born 1941 to 1950) in the four counties of Uppsala, Dalarna, Sörmland, and Gävleborg were identified. All measurements were performed by registered nurses, specially trained in ultrasonography, or ultrasound technicians. The maximum anteroposterior diameter of the infrarenal aorta was measured according to the leading edge to leading edge (LELE) principle.[14]

All men with a screen detected SAA (aortic diameter 25 — 29 mm) were re-invited for an ultrasound scan every five years, at ages 70 and 75 years. For various reasons (medical and other causes) some of the men were re-examined at shorter intervals. Surveillance intervals for SAA progressing to AAA (aortic diameter $\geq$ 30 mm) were: 30 — 39 mm every second year, 40 — 44 mm annually, 45 — 49 mm every six months, and $>$ 50 mm every three months. Once the aortic diameter reached $\geq$ 55 mm, repair was considered. Men with an aortic diameter $<$ 25 mm at baseline screening were discharged from the screening programme. Information on acute or elective AAA repair details was retrieved from the patient records. The dates and causes of death were retrieved from medical records linked to the National Population Registry. Data on risk factors, family and medical history were obtained from standardised health questionnaires and medical records. A history of AAA in first degree relatives, smoking status (never, former, and current smoker, as well as smoke duration in smoke and package years), coronary heart disease (defined as angina pectoris and/or myocardial infarction), diabetes mellitus (diet or medical treatment), cerebrovascular disease (transient ischaemic attack or stroke), hypertension, hyperlipidaemia, claudication, renal failure, chronic obstructive pulmonary disease (COPD), and ongoing medication with antiplatelet drugs and statins were recorded at the time of baseline screening.

Statistical analyses were performed with IBM SPSS Statistics software version 25.0 (IBM, Armonk, NY, USA) and R, version 4.0.3. (R Foundation for Statistical Computing, Vienna, Austria). Rates of progression from SAA to aortic diameter $\geq$ 30 mm or $\geq$ 55 mm were estimated using the Kaplan—Meier method and 95% CI were based on the log-log transformation of the cumulative incidence. Time was assessed from the date of the initial scan to the first scan that was equivalent to or greater than the respective threshold diameter. Men below the threshold diameter were censored at the last recorded scan.

The cumulative risk of reaching AAA $\geq$ 55 mm was estimated separately for those who had developed an AAA $\geq$ 30 mm after five year follow up and those who had not. Kaplan—Meier (KM) analyses were also applied to estimate mortality rates and the duration from AAA $\geq$ 55 mm to the date of intervention. The log rank test was used to determine statistical differences in survival between the two subgroups, aortic diameter $\geq$ 30 mm and aortic diameter $<$ 30 mm based on the five year re-scan. The chi square test was used to compare categorical variables, and for continuous data, an independent sample *t* test was used. Proportions were presented with 95% confidence intervals (CI). A two tailed *p* value of $<$ .050 was considered to be statistically significant. Annual growth rates were determined by computing the diameter of the last scan minus the diameter of the first scan (mm) divided by the time interval (years).

The study complied with the principles of the Declaration of Helsinki and was accepted by the ethics committee of the Uppsala/Örebro Region (Dnr 2006:112 and Dnr 2018/099). As specified by the ethics committee, informed consent was not required.

RESULTS

Between 1 January 2006 and 31 December 2014, a total of 60 475 men were invited to AAA screening at age 65 years, of whom 52 221 were screened (attendance rate 86.4%; 95% CI 86.1 — 86.7). A total of 1 020 men with SAA (25 — 29 mm) were detected (2.0%; 95% CI 1.8 — 2.2), of whom 940 (92.2%; 95% CI 91.0 — 93.8) had any follow up scan after the baseline screening (Fig. 1). In 65 (6.9%; 95% CI 5.3 — 8.5) of those, a follow up examination was carried out prematurely for various reasons, for example the individual's own wishes because of anxiety or a concomitant iliac aneurysm that needed to be re-examined before the five year interval. However, 50 of the 65 later had their pre-planned five year re-scan as well. Thus, 925 (98.4%; 95% CI 97.6 — 99.2) of the included men contributed with five year re-scan measurement and all with some kind of follow up measurement.

A total of 142 (15.1%; 95% CI 12.8 — 17.4) men had a follow up scan after $\geq$ 10 years of follow up. The mean

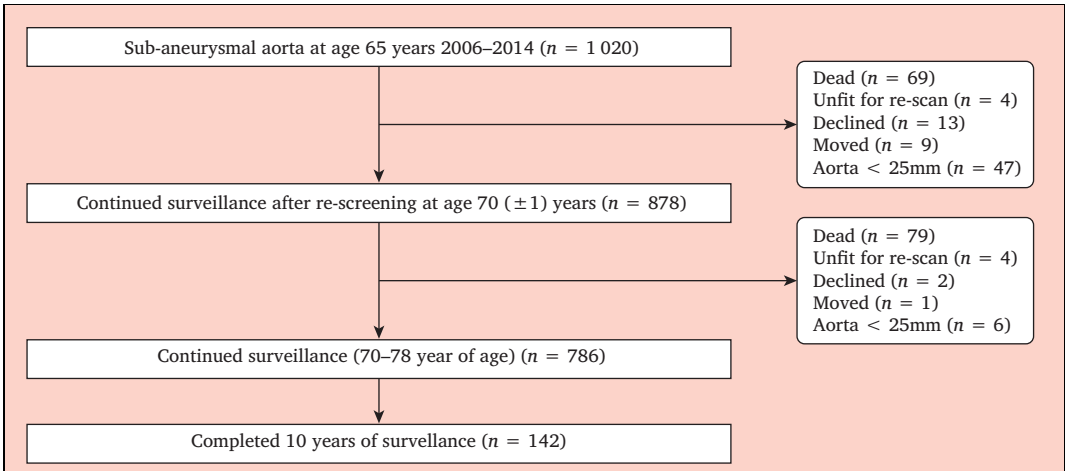

Figure 1. Flow chart of follow up of the sub-aneurysmal aorta (SAA) cohort of men with SAA measuring 25 — 29 mm in ultrasound screening.

anteroposterior diameter at baseline screening was 26.7 mm (standard deviation [SD] ± 1.4 mm). Mean duration of complete follow up was 6.5 years (SD ± 2.2; range 0.5 — 13.1 years) and the mean annual growth rate was 0.73 mm/year (range −3.03 — 6.12).

The KM estimated incidence of AAA ≥ 30 mm development after the five year follow up scan (which was *de facto* carried out after a mean of 4.9 years) was 65.8% (95% CI 61.6 — 69.4), all < 55 mm. Fifty-three of 940 men (5.6%; 95% CI 3.9% — 6.7%) had an aortic diameter < 25 mm at re-examination.

After the 10 year follow up scan (carried out after a mean of 11.9 years), it was estimated that 95.1% (95% CI 90.1 — 97.4) had developed AAAs ≥ 30 mm, and 29.7% (95% CI 18.0 — 39.7) AAAs ≥ 55 mm (Fig. 2). Based on the status at the five year re-scan, 37.2% (95% CI 25.2 — 47.4) of aortas ≥ 30 mm and none in the < 30 mm group reached ≥ 55 mm within the 10 year follow up (Fig. 3). As a sensitivity analysis, those who actually had ≥ 10 years of follow up ultrasound scans were evaluated separately. Of the original 1 020 men with SAA, a total of 142 had ≥ 10 years of follow up (Supplementary Table S1), of whom 38 had progressed to ≥ 55 mm. Thus 26.8% (95% CI 19.5 — 34.0) of the individuals with sufficient follow up had reached the threshold for surgical repair within ≥ 10 years.

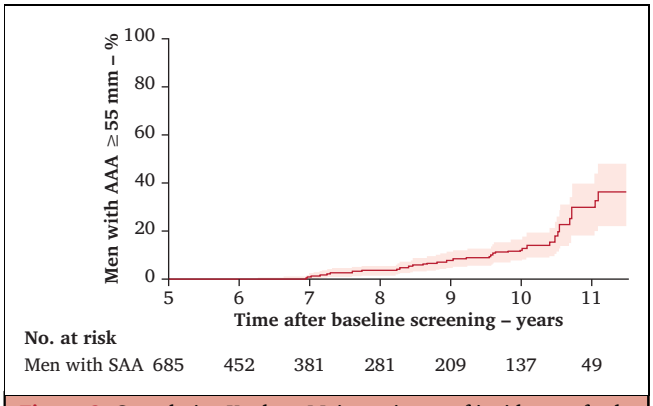

Figure 2. Cumulative Kaplan—Meier estimate of incidence of sub-aneurysmal aortas (SAA) progressing to abdominal aortic aneurysm (AAA) ≥ 55 mm during follow up in men. Shaded area represents 95% confidence interval.

Forty-one men reached the aortic diameter of ≥ 55 mm, of whom 38 (92.7%) were eligible for repair. During the study period, 32 of those (84.2%) had undergone aortic repair (31 received elective surgery, and one man underwent emergency rAAA repair), while six men were waiting for scheduled repair. Three men (7.3%) were considered unfit for intervention.

There were three ruptured abdominal aortic aneurysms (rAAAs), all treated surgically with 100% survival. One ruptured 37 mm mycotic aneurysm occurred 4.5 years after baseline screening. The remaining two rAAAs occurred seven years after baseline screening, one presenting with a 47 mm saccular lesion associated with plaque rupture, and one presenting with a 56 mm rAAA five months after the last scan with measurement; 49 mm.

No known AAA repair related or rAAA related deaths occurred; however, one man died from an undetected ruptured iliac aneurysm 5.5 years after the initial scan. The estimated cumulative all cause mortality rate was 7.0% (95% CI 5.4 — 8.6) at five years and 17.9% (95% CI 15.0 — 20.8) at 10 years. The estimated cumulative mortality was significantly higher among those with an SAA expanding to AAA compared with men having a stable SAA at the five year follow up; ($p <$ .001). During the study period, the overall crude mortality rate was 14.5% (95% CI 12.5 — 16.9), with malignancy (43.9%) and cardiac disease (20.3%) as the most common causes of death. The causes of death are displayed in Table 1.

The main factors associated with SAA expanding to ≥ 30 mm within five years follow up (*vs.* not) were frequency of smoking (current and former) ($p <$ .001) and smoking duration (smoke years and pack years) ($p <$ .001). Ever smoked and current smoking yielded the highest odds ratios (OR 2.6; $p <$ .001) and (OR 2.4; $p <$ .001), respectively. The frequency of coronary artery disease (OR 1.6; $p =$ .002) and cerebrovascular disease (OR 1.7; $p =$ .042) and the

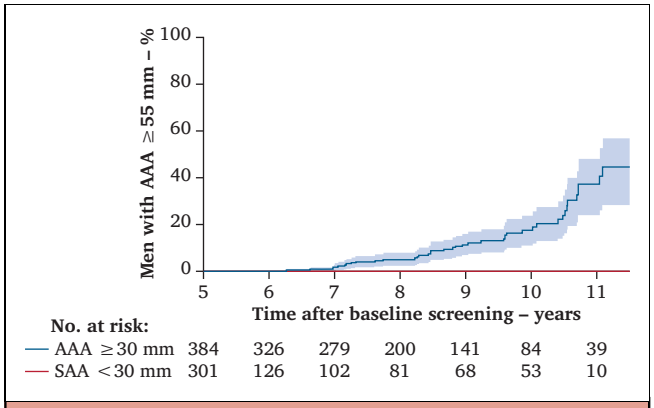

Figure 3. Cumulative Kaplan–Meier estimate of incidence of subaneurysmal aortas (SAA) progressing to abdominal aortic aneurysm (AAA) ≥ 55 mm stratified by aortic diameter of 25 – 29 mm and ≥ 30 mm at five year follow up screening in men. Shaded area represents 95% confidence interval.

infrarenal aortic AP diameter at 65 years (OR 1.8; $p < .001$) were higher among the subgroup of men with SAA expanding to AAA ≥ 30 mm within five years than among those with SAA that did not expand to AAA (Table 2).

DISCUSSION

This population based screening follow up study with high attendance (86.4%) reports the long term fate of a large cohort of men with screen detected SAA. In total, two of three of the individuals with SAA at baseline screening are estimated to eventually progress to an AAA ≥ 30 mm after five years and most (95%) progress to an AAA after 10 years. More importantly, as many as 30% are estimated to eventually progress to a large AAA requiring surgical repair within 10 years. These results confirm the findings from a previous multicentre cohort study by Wild *et al.* and a screening study by Oliver-Williams *et al.*[11,12] Unlike the current study, however, the multicentre study included older cohorts of patients with a broader range of age at baseline measurements and a mixed cohort (women and men) as well as varieties in the measurement methods.[11] In the Gloucester study by Oliver-Williams *et al.*, an aortic inner to inner diameter of 26 – 29 mm was considered a SAA compared with LELE diameter of 25 – 29 mm in the present study. In that study, the proportion of men with a SAA developing large AAAs increased from 10% to 28% between 10 and 15 years follow up.[12]

Table 1. Causes of death among the 148 men diagnosed with a sub-aneurysmal aorta measuring 25 – 29 mm in diameter on ultrasound screening who died during the study period

Cause of death	Patients (n = 148)
Malignancy	65 (43.9)
Cardiac disease	30 (20.3)
Pulmonary disease	13 (8.8)
Stroke	7 (4.7)
Trauma	5 (3.4)
Sepsis	3 (2.0)
Ruptured iliac aneurysm	1 (0.7)
Ruptured aortic aneurysm	0 (0)
Other	12 (8.1)
Unknown	12 (8.1)

Data are presented as n (%).

A key question is the risk of developing a large AAA that is eventually repaired, and the outcome of such repairs. In the present study, 90% of those reaching the diameter threshold for surgical repair were treated or scheduled for surgical treatment, while only 7.5% were considered not eligible for aortic repair because of comorbidity. The turndown rate appeared lower compared with a study by Lim *et al.*, who reported that 17.6% of SAAs and AAAs reaching > 55 mm were deemed not eligible for repair in the Gloucestershire screening programme.[15] The results in the present study come from a more contemporary setting and may be caused by increased use of less invasive endovascular procedures as well as improvements in the management of comorbidities.

Lindholt *et al.*[16] and Svensjö *et al.*[8] reported outcomes after five year follow up when no one had yet reached the surgical threshold and needed surgical repair. This was confirmed in the present study, which supports that a repeat scan within five years is not required to prevent AAA rupture. The value of the five year re-screening is instead to identify individuals at risk of developing clinically relevant AAAs and thus may need continued follow up, and just as importantly those who have a low risk and can avoid continued follow up.

The observed 18% all cause mortality rate after 10 years in the present SAA cohort appears lower than in a previous observational studies.[7,17] In this more contemporary cohort, the falling prevalence of smoking,[18] improvements in the management of cardiovascular disorders,[19] and malignancies as well as generally increased survival may explain the observed better survival.

In the present study, malignancy and cardiac disease were the main causes of death, while no AAA related events were observed, which may suggest that the surveillance strategy of SAA used is safe and reliable. In 12/148 (8.1%) of the deaths, the causes were unknown. It is unlikely, however, that a large number of those suffered AAA related deaths as the majority died early after being diagnosed with a SAA (< 30 mm).

The association between AAA and risk factors such as smoking, cardiovascular disease, hypertension, and hyperlipidaemia is reported in numerous studies.[18,20–23] Smoking is not only found to be positively associated with AAA, but also associated with growth rate and rupture risk.[24] In a recently published observational study, the present authors demonstrated that the risk factor profile of men with SAA was very similar to that of men with AAA, particularly among men with SAA expanding to an AAA within five years.[9] A strong association between smoking and aortic disease severity was reported, and a dose response relationship was suggested. This association was confirmed in the present study by comparing risk factors between men with an aortic diameter ≥ 30 mm or below the threshold diameter 30 mm after the five year re-scan. The most important risk factor was smoking, with 2.5 fold increased

Table 2. Characteristics of 925 men originally detected with a sub-aneurysmal aorta (SAA) measuring 25 − 29 mm in diameter on ultrasound screening, with aortic diameter of < 30 mm or ≥ 30 mm after five year follow up ultrasound scan

Factor	Patients with SAA growth (95% CI)		Odds ratio (95% CI)	*p* value*
	Aorta < 30 mm (*n* = 392)	Aorta ≥ 30 mm (*n* = 533)		
Current smoker	20.3 (16.5 − 24.1)	38.4 (34.1 − 42.7)	2.4 (1.8 − 3.3)	<.001
Ever smoked	70.0 (65.6 − 74.2)	85.6 (82.5 − 88.7)	2.6 (1.9 − 3.5)	<.001
Never smoked	29.8 (25.5 − 34.1)	14.4 (11.3 − 17.5)	0.4 (0.3 − 0.6)	<.001
Mean duration of smoking − y				
Smoke years	21.1 (19.1 − 23.1)	32.0 (30.2 − 33.9)	1.03 (1.03 − 1.04)	<.001
Pack years	16.6 (14.4 − 18.8)	25.6 (23.6 − 27.6)	1.03 (1.02 − 1.04)	<.001
Mean infrarenal aortic AP diameter at 65 y screening − mm	26.1 (26.0 − 26.2)	27.1 (27.0 − 27.2)	1.8 (1.6 − 2.0)	<.001
First degree relative with AAA	12.0 (8.9 − 15.2)	9.6 (6.6 − 12.3)	0.8 (0.5 − 1.2)	.25
Coronary artery disease	22.3 (18.4 − 26.2)	31.3 (27.3 − 35.4)	1.6 (1.2 − 2.1)	.002
Hypertension	53.3 (48.6 − 57.9)	59.3 (55.0 − 63.6)	1.3 (1.0 − 1.7)	.066
Hyperlipidaemia	34.3 (29.8 − 38.8)	38.5 (34.2.-42.8)	1.2 (0.9 − 1.6)	.18
Cerebrovascular disease	5.7 (3.5 − 7.9)	9.3 (6.7 − 11.8)	1.7 (1.0 − 2.8)	.042
Claudication	2.5 (1.0 − 4.0)	4.8 (3.0 − 6.7)	2.0 (1.0 − 4.1)	.063
Chronic obstructive pulmonary disease	5.5 (3.4 − 7.6)	7.0 (4.8 − 9.2)	1.3 (0.8 − 2.2)	.33
Diabetes mellitus	16.7 (13.2 − 20.2)	12.5 (9.6 − 15.4)	0.7 (0.5 − 1.0)	.069
Renal insufficiency	0.9 (0 − 1.8)	1.4 (0.4 − 2.4)	1.6 (0.5 − 5.3)	.48
Antiplatelet use	25.5 (21.4 − 29.6)	35.6 (31.4 − 39.8)	1.6 (1.2 − 2.2)	<.001
Statin use	31.6 (27.2 − 35.9)	39.8 (35.5 − 44.1)	1.4 (1.1 − 1.9)	.010

Data are presented as % (95% CI) unless stated otherwise. AAA = abdominal aortic aneurysm; AP = anteroposterior; CI = confidence interval.
* p value of difference between aortic diameter < 30 mm and aortic diameter ≥ 30 mm; $p < .050$ was considered to be statistically significant.

risk of progressing to an AAA for current smoking men and men who have ever smoked. Also, coronary artery disease and cerebrovascular disease were significantly more frequent in the ≥ 30 mm group compared with < 30 mm group after five years. Because of the clear association between smoking and coronary artery disease, men with SAA could benefit from targeted smoking cessation and secondary cardiovascular prevention programmes.

The 2019 European Society for Vascular Surgery (ESVS) Clinical Practice Guidelines on the Management of Abdominal Aorto-Iliac Artery Aneurysms issued a weak recommendation to re-scan men with SAA after 5 − 10 years.[1] With an observed substantial risk of progressing to large AAA after 10 years and a majority of those being eligible for surgical repair, the present findings support this recommendation. Furthermore, the present data suggest that the need for follow up may be stratified based on the five year re-scan. After 10 years of follow up, none of those with a screen detected SAA still < 30 mm after five years progressed to a clinically relevant AAA.

Diagnosis and surveillance for asymptomatic disease could have negative psychological effects. However, in a recent systematic review, it was concluded that these effects of AAA screening and surveillance of small AAAs showed no significant long term adverse effects on quality of life.[25] Nevertheless, these aspects need to be evaluated also in the setting of SAA, to ensure that detection and monitoring of men with SAA does not do more harm than good.

The present study has several important limitations. The time to event analysis is to some extent misleading as the event is based on the surveillance interval, rather than a continuous event. This is especially true for the transition to an AAA (30 mm), while the time to reach the diameter threshold for repair (55 mm) is more reliable as the follow up frequency increases with diameter. Furthermore, the lack of a control group means that it cannot be determined how many SAAs would have undergone a new examination anyway (incidental detection). However, this is of less importance as the main purpose was to describe the natural course of SAAs regarding the risk of developing a large AAA requiring surgical repair later in life, regardless of how it was detected. This study only reports the actual growth rate, while there are more advanced methods for modelling growth patterns.[12,26] It is also important to clarify that the main results are KM estimates. Although everyone contributed with follow up data for as long as they could in the analysis, only a fraction *de facto* contributed with follow up data ≥ 10 years. However, the KM estimate and the actual proportion reaching ≥ 55 mm within 10 years were relatively consistent (29.7% and 26.8%, respectively), suggesting that the results are robust.

General screening of 65 year old men is highly cost effective according to several studies,[27−30] even with today's low AAA prevalence of about 1.5%.[31] Estimates from the Swedish Screening Program[30] indicated that approximately 40% of all screen detected AAAs require surgical treatment over a lifetime. Consequently, 40% of 1.5% = 0.6% of everyone screened could potentially benefit from the screening and subsequent follow up. In this targeted subgroup of men with SAA, many more than 0.6% of those offered surveillance could benefit from ultrasound surveillance, with figures indicating that > 30% progress to repair within five years after being stratified at the five year follow up. Also, the repair would occur at an age where the men still may benefit from preventive surgery. With increased longevity of the elderly population and the expanded use of endovascular aneurysm repair (EVAR), more patients are being offered surgical repair

and there is lower per-operative mortality, especially in the elderly population.[32,33] A follow up policy and risk factor modification strategies on this fairly small cohort of SAA (< 2% of the entire population) has the potential to improve further the effectiveness of AAA screening programmes. Although the simple estimate above shows that the follow up of SAA is probably cost effective, a formal cost effectiveness analysis and longer follow up surveillance are required to prove its value definitively.

Conclusion

A majority of SAAs eventually progress to an AAA, of which 30% eventually reach the threshold for repair within 10 years. A follow up policy with an ultrasound examination after five years can safely and effectively identify those SAAs at risk of developing into clinically significant AAAs needing repair and may be considered for anyone with reasonably good life expectancy.

CONFLICT OF INTEREST

None.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2021.05.039.

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COUP D'OEIL

Past Poliomyelitis with Limb Atrophy Is Not a Contraindication to EVAR

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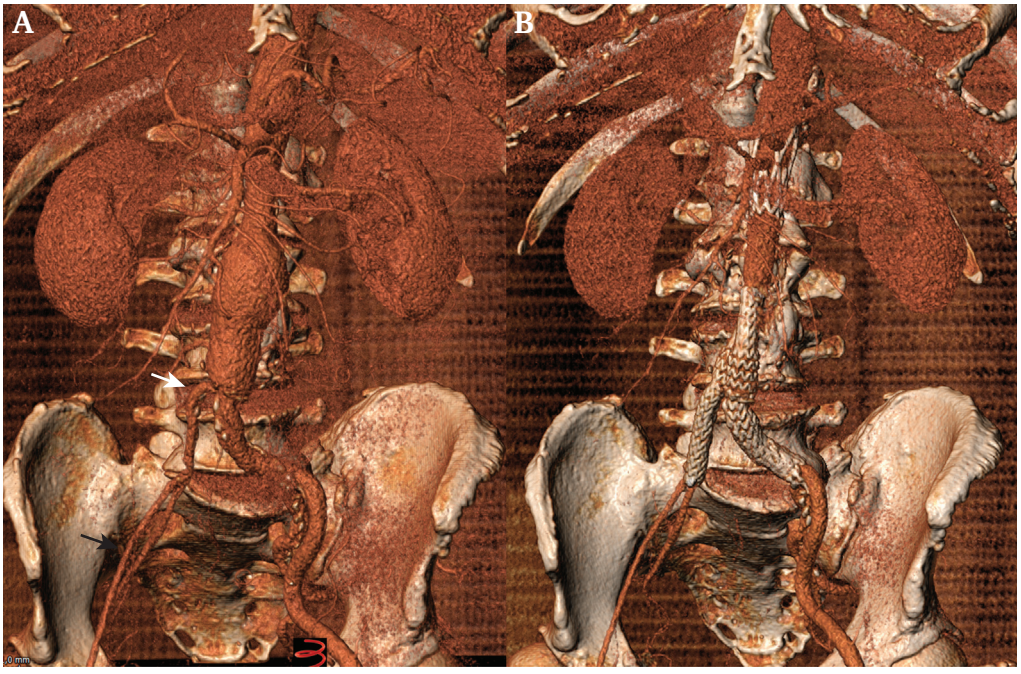

A 63 year old man presented with a 55 mm abdominal aortic aneurysm. His right lower limb was atrophic due to poliomyelitis at the age of two. (A) Computed tomography angiography showed hypoplastic right common (7 mm; white arrow) and external (4 mm; black arrow) iliac and femoral (8 mm) arterial diameters. Open access was chosen bilaterally and pre-dilation of the right external iliac artery was not necessary. The patient was treated successfully with the Ovation stent graft (Endologix, Santa Rosa, CA, USA). (B) At the one month follow up, the right axis remained patent without any dissection, kinking, or stenosis.

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