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What is This?

Surveillance strategies according to the rate of growth of small abdominal aortic aneurysms

Stephen A Badger[1], Claire Jones[1], Jane McClements[1], Louis L Lau[1], Ian S Young[2] and Christopher C Patterson[2]

Abstract

The management of small abdominal aortic aneurysms (AAA) is by ultrasound surveillance. The study aimed to calculate their growth rate, identify risk factors and determine appropriate screening intervals. The local screening programme and hospital records were used to identify patients with a small (< 5.5 cm) AAA. The dates and maximum diameter of serial scans of patients with two or more scans were obtained. Patients were subdivided by 0.5 cm increments above 3.0 cm. The rate of growth was calculated by linear regression for each patient using both the absolute measurements and logarithmically (ln) transformed measurements. The 95th centile of growth rate within each subgroup was used to estimate the minimum time to grow to 5.5 cm. A total of 252 were included. The mean (± SD) AAA size on the initial scan was 3.9 (± 0.7) cm. Statin use and initial size were predictive factors for the growth rate. The median rate of growth increased according to size from 0.075 to 0.432 cm/year for AAA < 3.5 cm and > 5.0 cm, respectively. It also steadily increased for ln measurements from 0.022 (or 2.2%/year) to 0.078 or (7.8%/year). The minimum time (months) to reach 5.5 cm was 61, 17, 11 and 5 for AAA < 3.5 cm, 3.5–3.9 cm, 4.0–4.4 cm and 4.5–4.9 cm, respectively. Based on ln measurements, the times were similar at 60, 17, 10 and 4 months. In conclusion, the rate of growth increased steadily with AAA size. An aneurysm < 3.5 cm does not require a repeat scan for 5 years, while those measuring 3.5–3.9 cm and 4.0–4.4 cm require a repeat scan after 17 and 11 months.

Keywords

abdominal aortic aneurysm; rate of growth

Introduction

The role for abdominal aortic aneurysm (AAA) screening is now irrefutable, adding impetus to national programmes.[1] Although screening will prevent rupture of large aneurysms, a large number of small aneurysms would be diagnosed. Medical optimization and risk factor modification are designed to retard the rate of growth and prevent disease progression. There is an association between initial size and subsequent rate of growth, although the nature of this is debateable.[2–5] Other inconsistent predictors of progression include smoking, hypertension, diabetes mellitus, age, sex and certain classes of drugs.[2–8]

The Aneurysm Detection and Management (ADAM) trial and the UK Small Aneurysm Trial (UK SAT) showed that AAA < 5.5 cm should have ultrasonographic surveillance as follow-up, rather than early surgery.[9,10] While the validity of the results has been questioned, the applicability of the conclusions in the endovascular era have also been challenged.[11–14] However, the frequency of serial measurements is variable between institutions, with no consistent evidence to support guidelines.

It could, therefore, be hypothesized that the rate of growth and associated surveillance intervals could be determined by risk stratification and aneurysm size. The primary aim of the study was to calculate the average rate of growth of small aneurysms. The secondary aims were to identify any risk factors for increased rate of growth and to determine appropriate screening intervals, according to the initial size of the AAA.

Patients and methods

The details of all patients who underwent an ultrasound coded as 'ultrasound of aorta' over a 13-month period from January 2005 to January 2006 were obtained from the

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Belfast City Hospital Department of Radiology. This was supplemented by a list of patients diagnosed with AAA as a result of attendance at the screening programme from August 2004 to August 2006. To ensure complete capture of patients, the small aneurysm database in the unit was searched.

Inclusion criterion was patients with an aneurysm measuring less than 5.5 cm, while those greater than 5.5 cm were excluded. The computerized radiology records were searched for each patient and the results of the follow-up ultrasound and CT scans were obtained. The rate of growth was then calculated for all patients with two or more scans, at least 3 months apart. The overall cohort of patients was divided into subgroups depending on the initial maximal aortic diameter: 3.0–3.4 cm, 3.5–3.9 cm, 4.0–4.4 cm, 4.5–4.9 cm, 5.0–5.4 cm. Exclusion criteria were patients with a normal aorta, miscoded as an aneurysm; those who had a large AAA; missing data or patient who had only a single scan in the radiology records.

Past medical and drug history at the time of initial diagnosis was recorded, where available. The risk factors recorded included smoking (current or former), hypertension requiring treatment, ischaemic heart disease, diabetes mellitus, chronic obstructive pulmonary disease, hyperlipidaemia and a family history of AAA. The drugs included in the study analysis were antiplatelets, statins, warfarin and beta blockers. Although these risk factors and medications may have changed during the follow-up period, the measurements were standardized to the start of the follow-up. An estimated glomerular filtration rate was also obtained from the biochemistry records to quantify any renal impairment; a value of less than 60 ml/min was taken as indicating renal impairment.

Statistical analysis

The data were stored in Microsoft Excel and subsequently analysed by SPSS Version 15 (SPSS Inc., Chicago, IL, USA). Individual rates of growth were calculated by linear regression both on the original scale of size measurement and also on a (natural) logarithmically transformed scale. The former provided an absolute rate of increase (cm/year). The latter provided a proportionate rate of increase (%/year), after anti-logarithmic transformation, which could be expressed as the percentage increase per year, as such a proportionate increase of 1.045 was equivalent to a 4.5% increase per year (%/year). Growth rates were closer to normally distributed when calculated on the logarithmic scale than on the original scale. The influence of each factor on rate of growth, on the logarithmic scale, was assessed initially by independent samples *t*-test and one-way analysis of variance, and confirmed in a two-level random effects model fitted using the xtmixed command in Stata release 11 (StatCorp College Station, TX, USA). In light of some non-normality of growth rates, even when calculated on the logarithmic scale, the 95th centile was used to calculate the appropriate surveillance intervals in each subgroup defined by initial AAA size. Taking 5.5 cm as the target size, the

minimum time taken to reach this size for each subgroup could then be estimated. The 95th, rather than the 50th centile was used, so that even the most rapid aneurysm growth would be captured. Thus, the recommended surveillance intervals should be regarded as a minimum, with each strategy tailored according to the patient's individual growth pattern. Finally, the individual patient growth patterns were graphically examined to validate the calculated surveillance intervals.

Results

After the exclusion criteria were applied, 252 patients formed the study cohort. This comprised of 214 male and 38 female patients. The number of follow-up scans ranged from 2 to 17 (median 5). The mean (SD) size of AAA on the initial scan taken at the time of diagnosis was 3.9 (0.7) cm. The screening programme provided 94 patients, while the remaining 158 were from the Department of Radiology records.

Risk factors

The distribution of risk factors at the time of initial diagnosis is detailed in Table 1. The influence of each risk factor on the subsequent rate of growth is detailed in Table 1. The only significant ($p < 0.05$) predictor of rate of growth was statin therapy: patients receiving statins having a slower growth (geometric mean 1.045 or 4.5% per year) than patients who did not (geometric mean 1.075 or 7.5% per year).

Rate of growth and surveillance intervals

The overall cohort was subcategorized according to initial AAA diameter on diagnosis (Tables 2 and 3). The number of patients in each subgroup decreased with increasing size from 72 in the < 3.5 cm category to 20 in the 5.0+ cm category. The mean rate of growth (cm/year), based on the absolute measurements, increased across the five categories of initial size from 0.09 cm/year to 0.41 cm/year. The corresponding geometric mean proportionate rates of increase from the analysis of logarithmically transformed data ranged from 1.025 (or 2.5%/year) to 1.075 (or 7.5%/year) and differed significantly (one-way analysis of variance, $p = 0.002$). The distribution of rates of growth is illustrated in Figure 1.

The 95th centile of rate of growth was calculated within each subgroup defined by initial size and used to estimate a screening interval as shown in Tables 2 and 3. Based on the absolute measurements, those patients with AAA 3.0–3.4 cm should have a repeat ultrasound scan in 61 months. Those with AAA 3.5–3.9 cm should have their next scan in 17 months, while those in the next subgroup should be scanned after 11 months. The patients with AAA 4.5–4.9 cm need a repeat scan in 5 months. Based upon ln rate of growth, the times were similar at 60, 17, 10 and 4 months, respectively. Examination of the plots for individuals showed that no patient breeched the 5.5 cm threshold within

Table 1. The influence of risk factors on the rate of growth in 252 small AAA patients

Risk factor[a]	Prevalence n/N (%)	Average growth rate (%/year)[b]		p
		Present	Absent	
Female sex	38/252 (15%)	5.0	5.6	0.84
Age > 70 years	148/252 (59%)	5.5	5.5	0.88
Screened-detected AAA	94/252 (37%)	5.3	5.6	0.16
Smoker	44/148 (30%)	6.1	5.3	0.49
Ex-smoker	58/148 (39%)	5.7	5.2	0.60
Hypertension	71/150 (47%)	5.6	5.2	0.67
Ischaemic heart disease	61/150 (41%)	5.8	5.2	0.93
Diabetes mellitus	17/150 (11%)	5.4	5.4	0.64
Chronic obstructive pulmonary disease	13/147 (9%)	2.8	5.8	0.25
Hyperlipidaemia	45/146 (31%)	7.3	4.8	0.14
Chronic renal failure (eGFR < 60 ml/min/1.73 m^2)	3/54 (5%)	5.9	5.5	0.66
Family history of AAA	28/148 (19%)	5.5	5.4	0.86
Antiplatelet therapy	86/143 (60%)	4.9	6.7	0.43
Statin therapy	92/143 (64%)	4.5	7.5	0.005
Warfarin therapy	11/143 (8%)	5.1	5.6	0.97
Beta-blocker therapy	66/143 (46%)	5.6	5.6	0.69

[a]Some factors were recorded only in 150 patients whose clinical details were available.
[b]Derived from the geometric mean of proportionate increases estimated from regression of logarithmically transformed sizes.

Table 2. Rates of growth derived for each patient by linear regression analysis and summarized according to initial AAA size together with an estimate of time to attain a size of 5.5 cm assuming a growth rate at the 95th centile

Initial size (cm)	n	Growth rate (cm/year)			Max initial size (cm)	Growth to attain 5.5 cm (cm)	Time to attain 5.5 cm (months)
		Mean	95% CI	95th centile			
< 3.5	72	0.09	0.05–0.14	0.42	3.4	2.1	61
3.5–3.9	64	0.25	0.17–0.33	1.11	3.9	1.6	17
4.0–4.4	53	0.31	0.22–0.40	1.18	4.4	1.1	11
4.5–4.9	43	0.40	0.24–0.56	1.52	4.9	0.6	5
5.0–5.4	20	0.41	0.19–0.63				

Table 3. Rates of growth derived for each patient by linear regression analysis of logarithmically transformed size and summarized according to initial AAA size together with an estimate of time to attain a size of 5.5 cm assuming a growth rate at the 95th centile

Initial size (cm)	n	Growth rate (%/year)			Max initial size (cm)	Growth to attain 5.5 cm (ln cm)	Time to attain 5.5 cm (months)
		Mean[a]	95% CI	95th centile			
< 3.5	72	2.5	1.2–3.8	10.0	3.4	0.481	60
3.5–3.9	64	5.9	3.9–7.9	25.8	3.9	0.344	17
4.0–4.4	53	6.7	4.9–8.6	28.9	4.4	0.223	10
4.5–4.9	43	7.8	4.7–11.1	33.1	4.9	0.116	4
5.0–5.4	20	7.5	3.3–11.9				

these intervals, thus providing internal validation of the calculations (Figure 2).

Analysis of the correlation structure of the repeated size measurements revealed that correlations between measurements declined with elapsed time. The initial size measurement showed a correlation of 0.79 with measurements taken the following year, but this reduced to 0.45 for measurements taken 10 or more years later. Consequently, the unstructured option was selected for the correlation matrix in the Stata xtmixed command. The multilevel random effects analysis showed that a quadratic term in time was significant when AAA size was analyzed ($p < 0.001$) but was not significant when AAA size was analysed on a logarithmic scale ($p = 0.17$), indicating that growth rates were closer to linear when analyzed on a logarithmic scale. The analysis also confirmed that screen-detected cases
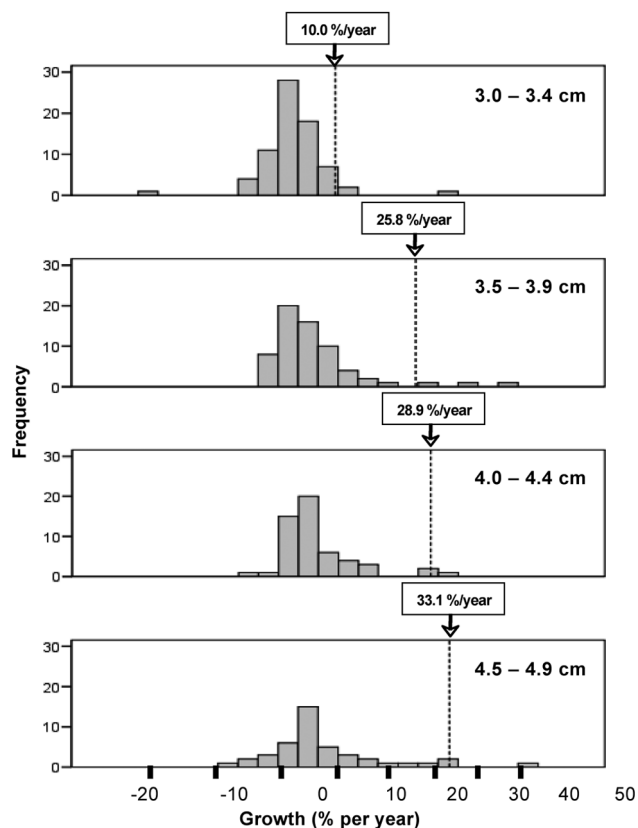
Figure 1. The distribution of rates of growth from linear regression analysis of logarithmically transformed size measurements broken into subgroups of initial size and showing the 95th centile of growth rate in each subgroup.

were not significantly different in their growth rates (*p* = 0.16) but that cases treated with statins had significantly slower growth rates (*p* = 0.005). Finally, the growth rates were found to be faster in the aneurysms of largest initial size (*p* = 0.004). These results therefore confirmed the findings obtained using the simpler statistical analyses of slopes described earlier.

Discussion

The available evidence on the aetiology of AAA shows that it is multifactorial. Most studies have assessed each risk factor with regard to disease presence. Recognized predisposing factors include advanced age, male sex, cigarette smoking, elevated cholesterol, hypertension and other atherosclerotic disease manifestations.[7,15–17] Less evidence is available for the risk factors for the rate of growth of AAA. The results of this study revealed some trends towards independent predictors of the rate of growth that are of clinical importance.

It is interesting to note that smoking did not emerge as a predictor of rate of growth. Over 90% of AAA patients have a history of smoking, with half of them continuing to do so at the time of diagnosis.[17] Several studies, including UK SAT, have demonstrated a correlation thereafter between the expansion and continued smoking.[2,7,18] It has been previously reported in a subgroup of this present

cohort that smoking was important in AAA formation.[19] The lack of continuing association with subsequent growth would suggest that tobacco is perhaps more important as a trigger to formation, rather than persistent promotion of subsequent growth, although only cautious conclusions can be drawn since almost 40% of patients lacked documented smoking history. Although current smoking is associated with an increased growth rate, this does not persist with lifetime measures of exposure, which show no evidence of a dose-dependent relationship.[2] Brady et al. demonstrated a 15–20% increase in growth rate for current smokers, but considered this insufficient to warrant more frequent screening intervals.[2] While the present study provides important new evidence about the role of smoking, it would be rash to conclude that smoking is not involved and that smoking cessation advice is not vital in these patients, particularly when the much larger UK SAT indicated otherwise.

Statin therapy at the time of AAA diagnosis showed a definite trend of slower rate of growth in this study. It is known to retard progression of atherosclerosis, with improved clinical outcomes. This appears to be as a result of a reduction in both atherogenic lipoproteins and other pleiotrophic effects, such as lowering C-reactive protein levels.[20] Interestingly, elevated cholesterol is associated with the presence of AAA, but not the rate of growth.[2,15,16,18,21] Nevertheless, the use of statins do slow the progression of AAA. This is substantiated by the present results, where although there was no relationship with elevated lipids (*p* = 0.14), there was slower growth with the use of statins (*p* = 0.005). This trend may have become stronger if it was possible to compensate for inevitable contamination from commencement of statins during the study period. Similar results were demonstrated by Schlosser et al., with statin-reduced growth retardation, independent of cholesterol levels.[22,23] Therefore, statins appear to stabilize the aortic wall by other means apart from cholesterol reduction.

No other Framingham cardiovascular risk factors in this study, as listed in Table 1, emerged as predictors of growth, as would be expected. Previous studies have shown diabetes and peripheral vascular disease to retard the growth rate by up to 30%, further enforcing the hypothesis that atherosclerosis has only a minor role in AAA disease.[2,24]

The rate of AAA growth in the UK SAT ranged from 0.1 to 0.61 cm/year, which are not dissimilar to the range shown by the present study, as stratified by initial size. The size-related growth rate was shown previously, with 0.21 vs 0.47 cm/year calculated for AAA 3.0–3.9 cm and 4.0–4.9 cm, respectively.[6] This more rapid expansion is associated with reaching 5.0 cm and undergoing surgical repair. Subgroup analysis in this present study showed a gradation of growth rates, with rates similar to those reported by others.[3,6,25] A bimodal distribution of the rate of AAA growth may exist, helping to explain why about 25% of small AAA fail to grow at all.[24] Within this growth pattern, an increased rate was shown to be associated with significant clinical AAA-related events. As a result, the authors recommended
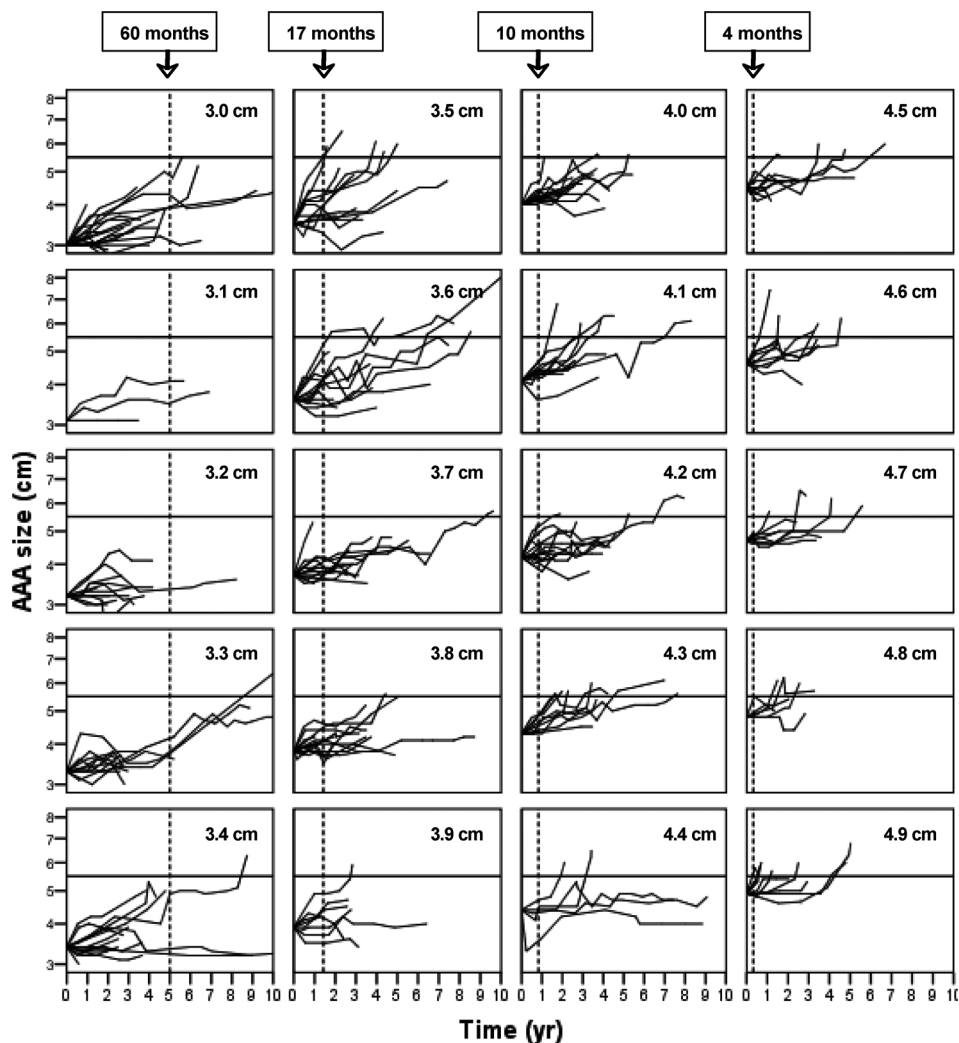
Figure 2. Individual growth patterns for each patient plotted on a logarithmic vertical scale, in 0.1 cm increments of initial size, showing a horizontal 5.5 cm size threshold and the estimated screening intervals derived from the data.

that the growth rate, as well as aortic diameter, should be accounted for when determining the screening interval. Insufficient evidence is available at present to be certain if early growth rate is an important determinant of later growth rate and warrants more research. This clearly has major implications for the service provision and cost-effectiveness of any screening programme, since the intensity of follow-up scans impacts upon workforce planning. This postulated growth pattern is not universally accepted, with staccato growth suggested by some researchers.[2,26]

The present study has a few weaknesses. First, it is well recognized that participants in a screening programme often tend to be more health conscious, with a better risk profile. Since a large proportion of this study cohort was derived from the local screening programme, the biasing effect of these patients may have altered the results of the growth rates. In addition, the lack of significance for most risk factors to influence the growth rate may be due to this subgroup of patients. Second, the retrospective collection of data is likely to have results in an over-representation of slow-growing aneurysms, thus altering the calculations, with the additional problem of missing data.

The optimal interval period between two consecutive scans is not clear.[27] A Danish study into this subject used a diameter of 50 mm as the endpoint for determining screening intervals.[28] The decisive variable was the initial diameter, with the resultant different rate of growth. Their recommendations were that rescanning should be in 4, 2 and 1 year for AAA 3.0–3.4 cm, 3.5–3.9 cm and 4.0–5.0 cm, respectively. These recommendations are in keeping with those proposed by several groups and may help to reduce the psychological distress associated with repeated scanning.[29] Cook and Galland also recommended annual screening for aneurysms < 40 mm, while those greater than this should be scanned every 6 months.[30] Current guidelines for the UK National Screening Programme are for a repeat ultrasound in 1 year if < 4.5 cm and in 3 months if greater than this. A recent study of 1743 patients demonstrated that if intervals of 36, 24, 12 and 3 months were adopted for aneurysms of 35, 40, 45 and 50 mm, respectively, the risk of breeching the 5.5 cm threshold at re-screening was less than 1%.[2] It is, however, important to distinguish between the clinical threshold for surgery and the imaging threshold of 5.5 cm. While the small

aneurysm trials indicate that aneurysms greater than this diameter should be considered for surgery, the growth pattern is gradual and breeching this does not represent a clinically adverse event. The decision to operate should be based upon the individual patient, with co-morbidities taken into consideration as well as size, thus introducing an inevitable flexibility to the threshold for surgery according to the clinical situation. Therefore, any guidelines for screening intervals need to be practical and convenient for both patient and clinician. Thus, the patients whose AAA is 4.5 cm or more could be followed up in 3–6 months, as suggested by the UK National Screening Programme and ADAM study, with the smaller aneurysms requiring less intensive intervals according to the present results.[15]

In conclusion, these results have demonstrated an increasing rate of growth according to initial AAA size, even after logarithmical transformation. Statin use was the only variable, which showed retardation of aneurysm growth. It would therefore be prudent to tailor screening intervals according to the AAA size and previous growth patterns.

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