SYSTEMATIC REVIEW

# Risk of Rupture and All Cause Mortality of Abdominal Aortic Ectasia: A Systematic Review and Meta-Analysis

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**WHAT THIS PAPER ADDS**

This systematic review and meta-analysis comprising 8 369 patients with abdominal aortic ectasias (AAEs; 25 — 29 mm) reveals that AAEs grow slowly, at a rate of 0.83 mm/year, and that the risk of rupture and aneurysm related deaths is 0.1% in the first five years. Notably, 13% of patients with AAEs die, indicating that AAEs carry a similarly high risk of death as abdominal aortic aneurysms; the major causes of death are cancer and cardiovascular diseases. A follow up interval of five years for AAE is reasonable, but additional caution around cancer and cardiovascular diseases is required.

**Objective:** To clarify the natural history of abdominal aortic ectasia (AAE) measuring 25 — 29 mm in maximum diameter, and to determine the optimal follow up based on the growth, risk of rupture, and overall mortality of AAE.

**Data Sources:** MEDLINE, Web of Science Core Collection, and Google Scholar.

**Review Methods:** This was a systematic review and meta-analysis of AAE in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. MEDLINE, Web of Science Core Collection, and Google Scholar were searched, with the help of a health sciences librarian, up to 11 August 2021. Studies with longitudinal outcomes of AAE (prevalence, annual growth rate, aneurysmal enlargement, rupture, aneurysm related death, and all cause mortality) were included. Meta-analyses were conducted with a random effects model

**Results:** Twelve studies describing a total of 8 369 patients were eligible. The prevalence at population based settings was 3.2% (95% confidence interval [CI] 2.4 — 4.0); annual growth rate was 0.82 mm/year (95% CI 0.20 — 1.45). The estimated risks of aortic diameters exceeding 30 mm and 55 mm in five years were 45.0% (95% CI 28.5 — 61.5) and 0.3% (95% CI 0 — 0.6) respectively, while those beyond five years were 70.2% (95% CI 46.9 — 93.6) and 5.2% (95% CI 2.2 — 8.2). The rates of rupture and aneurysm related death were minimal until five years (0.1% and 0.1%, respectively) and beyond (0.4% and 0.2%, respectively). Overall mortality was 7.5% (95% CI 3.9 — 11.0) and 17.3% (95% CI 9.5 — 25.1) up to and beyond five years. Overall mortality from three studies showed no statistical difference between AAE and aneurysms (hazard ratio 0.62, 95% CI 0.32 — 1.21; $p = .16$). Cancer (35.0%) and cardiovascular diseases (31.9%) were major causes of death.

**Conclusion:** AAE carries minimal risk of aneurysm related lethal events during the first five years, but a similar overall mortality risk as abdominal aortic aneurysm. Cancer and cardiovascular diseases are leading causes of death in patients with AAE.

**Keywords:** Abdominal aortic aneurysm, Abdominal aortic ectasia, Meta-analysis, Rupture, Mortality, Follow up
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## INTRODUCTION

Abdominal aortic aneurysm (AAA) is a significant burden, especially among those aged ≥ 65 years in developed countries, and is known to grow in size in its natural

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course.[1] AAA is defined as a locally enlarged abdominal aorta with a maximum diameter of ≥ 30 mm. Abdominal aortas from 25 mm to 29 mm in diameter are termed abdominal aortic ectasia (AAE).[2–5] Both AAA and AAE can be identified incidentally in the setting of population based screening or general practice; the management of AAA is well defined, with optimal follow up intervals, expected growth rate, rupture risk (lethal event), and when to intervene.[2,3] However, evidence for AAE is scattered and strong recommendations are lacking.

AAE is considered to be a precursor lesion of AAA, but not all AAE grow or rupture. Overestimation of rupture risk of AAE can lead to patient anxiety, unnecessary hospital visits, and socioeconomic burden. Lack of knowledge about the fate of AAE can threaten patients' lives. Precise understanding and estimation of natural growth, rupture, and mortality rate of AAE are indispensable for better patient care. In this context, unsolved clinical questions include the optimal follow up strategy and potential prevention of any complications in patients with AAE.[2]

In the Multicentre Aneurysm Screening Study (MASS) randomised trial, the long term outcomes showed that population based screening of men aged 65 — 74 years significantly reduced AAA related mortality by 42%, the incidence of AAA rupture by 43%, and even all cause mortality by 3% over 13 years of follow up.[6] Most of the findings were confirmed in a recent meta-analysis,[7] highlighting the potential significance of AAA related death and its prevention in healthcare management. However, the same study found that late rupture, eight years after the initial screening, occurred among those who had a maximum aortic diameter of 25 — 29 mm and were not recalled for further screening. As fatal events occurring in the population initially diagnosed with AAE can be prevented with further screening, the long term follow up of AAE is warranted.[6,7]

The aims of this study were to clarify the natural course of AAE, and to determine the best course of follow up based on the growth, risk of rupture, and overall AAE mortality rate.

## MATERIALS AND METHODS

This study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[8] As this was a systematic review and meta-analysis using published data, it was exempt from Institutional Review Board approval. The data underlying this article will be shared upon reasonable request to the corresponding author. The study was not registered in PROSPERO.

### *Search strategy*

A comprehensive search of MEDLINE, Web of Science Core Collection, and Google Scholar, in consultation with a health sciences librarian (A.D.), was conducted in August 2021. No publication date or language restrictions were imposed. The full literature search strategy is provided in Supplementary Appendix S1. Studies describing the longitudinal outcomes of AAE with outcomes of interest were included. AAE was defined as local dilatation of the abdominal aorta ranging in maximum diameter from 25 to 29 mm. Substitute search terms such as "subaneurysmal", "pre-aneurysmal", "mild dilatation", or "ectatic" were also used. Two independent authors (T.S. and H.T.) reviewed the search results and selected the studies.

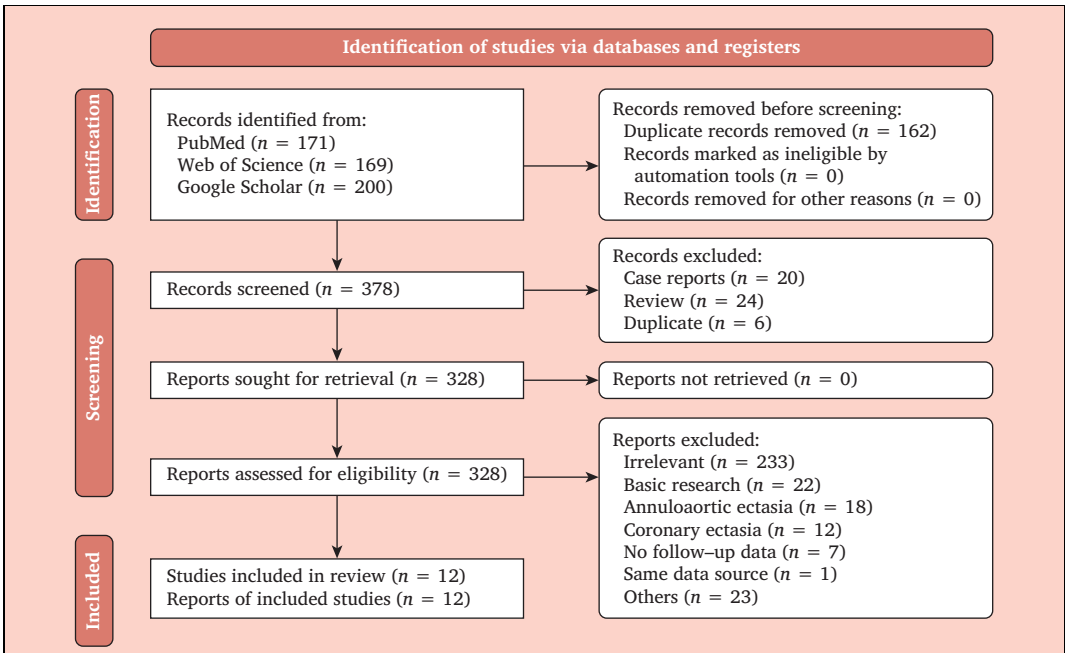


**Figure 1.** Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram for systematic review of studies describing outcomes of patients with abdominal aortic ectasia.

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### Study eligibility and risk of bias assessment

Studies that described follow up data for AAE were included. The definition of AAE used in the current study is maximum infrarenal diameter of 25 − 29 mm; the definition of AAA used was a maximum diameter of ≥ 30 mm. Studies lacking follow up data for AAE were excluded. Case reports, review articles, systematic reviews, letters, and editorials were not eligible for inclusion. Some full text articles were reviewed for eligibility and any discrepancies were discussed among the same authors (T.S and H.T.). The Risk of Bias Assessment Tool for Nonrandomised Studies (RoBANS) was used to assess the risk of bias.[9] Two authors (T.S. and H.T.) worked independently on the risk of bias assessment.

### Data extraction and synthesis

Original data were extracted according to the PRISMA checklist.[8] Two independent authors (T.S. and H.T.) reviewed the data eligible for analyses. The following information was retrieved from the studies: country; study period; setting of the study; the age of targeted patients; sex; follow up period; imaging modality; size criteria; and method of measurement. Comorbidities such as hypertension, diabetes mellitus, dyslipidaemia, coronary artery disease, cerebrovascular disease, chronic obstructive pulmonary disease, and peripheral artery disease, as well as smoking status and family history of aortic aneurysms, were noted. Caliper placement for measurement and training of examiners were also reviewed. The prevalence of AAE (%), annual growth rate (mm/year), the rate to reach 30 − 55 mm (%), rupture rate (%), aneurysm related death (%), and all cause mortality (%) were extracted. The hazard ratio (HR) of all cause mortality of AAE *vs.* AAA was collected from the studies that reported outcomes of both AAE and AAA. Causes of death were also reviewed. To estimate the prevalence of AAE and the rate of each outcome, the number of events and total number of eligible patients were retrieved to represent estimates at the time of the mean follow up period. HRs and 95% confidence intervals (CIs) were extracted when available, preferably for adjusted outcomes. In other cases, HRs were calculated from Kaplan—Meier curves using the HR calculations spreadsheet provided by Tierney *et al.*[10]

### Study outcomes

The prevalence of AAE in the setting of population based screening was estimated to ascertain the magnitude of AAE. Other outcomes were the annual growth rate (mm/year) and the rate to reach 30 mm and 55 mm each: 30 mm was defined as an AAA and 55 mm the threshold for invasive surgical treatment. Risk of rupture and aneurysm related death were estimated. Risk of all cause mortality, relative mortality compared with that of AAA, and cause of death data were also collected. If the data were derived from the same source (e.g., same screening programme), only the outcomes from the study with longer follow up were used.

### Statistical analysis

OpenMetaAnalyst version 12.11.14 was used to estimate one group meta-analysis for quantitative data (continuous and discrete) with the DerSimonian-Laird method as the random effects model. RevMan version 5.4 (Nordic Cochrane Centre, the Cochrane Collaboration, 2012, Copenhagen, Denmark) software was used to incorporate the pooled rates and HRs with 95% CI using the random effects model. The inverse variance weighted average of logarithmic HRs was combined using the DerSimonian—Laird method as the random effects model. Significant heterogeneity between studies was considered to exist if the $I^2$ index was > 50% or the $p$ value for heterogeneity was <.050.[11] Analyses for growth (30 mm and 55 mm in diameter), rupture, aneurysm related death, and overall mortality rate were performed according to two separate time periods: up to five years and beyond five years. Sensitivity analysis was conducted solely using population based screening data to confirm the robustness of the results.[12]

## RESULTS

The search initially identified 540 relevant articles (171 from MEDLINE, 169 from Web of Science, and 200 from Google Scholar; Fig. 1). After the exclusion of duplicates, 378 records underwent title and abstract screening. A further 50 articles were excluded (20 case reports, 24 reviews, and six duplicates). Title and abstract screening excluded 233 articles for irrelevance, 22 for basic research, 18 for annuloaortic ectasia, 12 for coronary ectasia, seven for no follow up data, one from the same data source, and 23 for other reasons. Finally, 12 studies were included for analysis.[13—24] As is shown in Supplementary Fig. S1, the RoBANS assessment demonstrated an unclear or high risk of bias regarding participant selection (25%), confounding variables (0%), measurement of exposure (25%), blinding of outcome assessment (0%), incomplete outcome data (58%), and selective outcome reporting (58%).

As is shown in Table 1, nine studies used population based screening, two used targeted screening at a Veterans Affairs hospital, and one was carried out at a vascular surgery department. Among the population based screening studies, six were from the UK, two were from Sweden, and one was from Denmark; they all targeted only male patients. As two separate studies were from Gloucestershire Aneurysm Screening Programme (GASP), they were not used at the same time, in order to avoid potential overlap (McCarthy *et al.*,[14] up to five years; Oliver-Williams *et al.*,[20] beyond five years). Another study collected aneurysm related death and ruptures in the longer term,[25] which were not described in the study by Oliver-Williams *et al.*[20] Six studies invited the population when they reached the age

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**Table 1. Characteristics of twelve included studies defining of outcomes of abdominal aortic ectasia**

| First author | Country | Period | Setting | Age | Male sex − % | Mean follow up − y | Imaging modality | Size criteria − mm | Measurement | Ectatic aorta − *n* (%) | Screened patients − *n* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Population based screening* | | | | | | | | | | | |
| Lindholt[13] | Denmark | 1994−1998 | PB | 65−73 | 100 | 5 | US | 25−29 | AP and T | 348 (5.5) | 6 339 |
| McCarthy[14] | UK | 1990−2002 | PB | 65 | 100 | 4.5 | US | 26−29 | AP | 625 (2.1) | 29 906 |
| Hafez[15] | UK | 2004−2006 | PB | 65 | 100 | 9.7* | US | 25−29 | AP and T | 119 (2.5) | 4 762 |
| Devaraj[16] | UK | 1992− | PB | 65−75 | 100 | 5.4 | US | 26−29 | AP | 358 (NA) | |
| Gibbs[17] | UK | 1996−2008 | PB + PC | 65 | 100 | 4.3 | US | 25−30 | AP | 413 (2.0) | 20 750 |
| Duncan[18] | UK | 2001−2004 | PB | 65-74 | 100 | 7.4* | US | 25−29 | AP | 669 (8.2) | 8 146 |
| Svensjö[19] | Sweden | 2006−2007 | PB | 65 | 100 | 5 | US | 25−29 | AP | 40 (1.5) | 2 736 |
| Oliver-Williams[20] | UK | 1990−2015 | PB | 65 | 100 | 7.8 | US | 26−29 | AP | 1 233 (1.5) | 81 150 |
| Thorbjørnsen[21] | Sweden | 2006−2014 | PB | 65 | 100 | 6.5 | US | 25−29 | AP | 1 020 (2.0) | 52 221 |
| *Other settings* | | | | | | | | | | | |
| d'Audiffret[22] | USA | 1992−2000 | VA | 50−79 | 97.2† | 5.9 | US | 25−29 | AP and T | 223 (1.8) | 12 500 |
| Basnyat[23] | UK | NA | VS | 48−90 | 77.6 | 2* | US | ≤29 | AP | 116 (NA) | |
| Chun[24] | USA | 2007−2016 | VA | 65−75‡ | 100 | 6.4 | CT, US, MRI | 25−29 | Max. | 3205 (16.3) | 19 649 |

AP = anteroposterior; CT = computed tomography; MRI = magnetic resonance imaging; NA = not available; PB = population based screening; PC = primary care; T = transverse; US = ultrasound; VA = Veterans Affairs medical centre; VS = vascular surgery department

* Expressed as median.

† From the original study.

‡ Whoever smoked.

of 65 years, and three invited those aged 65 − 75 years. Outside population based screening, one study used follow up data from the Aneurysm Detection and Management (ADAM) trial, which looked at 50 − 79 year old veterans (97.2%, male). Another study used retrospective data from Veterans Affairs medical centre hospitals, which screened 65 − 75 year old men who had ever smoked at least 100 cigarettes. The other study was a retrospective cohort study of patients diagnosed with AAE at a vascular surgery department. Seven studies used 25 − 29 mm as the size criteria, two programmes applied 26 − 29 mm, and another study 25 − 30 mm. Baseline characteristics are shown in Supplementary Table S1. Seven population based screening studies did not report comorbid conditions. In five studies, the median proportion of comorbidities was as follows: hypertension 57%; diabetes mellitus 14%; dyslipidaemia 28%; current smoker 23%; ever smoker 81%; coronary artery disease 22%; cerebrovascular disease 8%; chronic obstructive pulmonary disease 15%; peripheral artery disease 10%; and family history 11%. Methods of caliper placement were inner to inner in two studies, leading edge to leading edge in two, outer to outer in one, and not specified in seven. In six studies, description of trained examiners was available.

### *Prevalence of abdominal aortic ectasia and annual growth rate*

The prevalence of AAE was estimated using the seven population based screening studies. Of a total of 176 104 screened (65 − 73 years of age, exclusively male), 3 842 patients were diagnosed with AAE. The prevalence of AAE was estimated to be 3.2% (95% CI 2.4 − 4.0; Fig. 2A). As age can affect the prevalence of AAE, subgroup analyses targeting different ages was performed. In five studies that screened 65 year old men, the estimated prevalence was 1.9% (95% CI 1.6 − 2.2; Supplementary Fig. S2A), while in those studies targeting 65 − 74 year olds, the prevalence was 6.8% (95% CI 4.2 − 9.5; Supplementary Fig. S2B). In a targeted screening programme, the diagnostic rate among 65−75 year old males who had ever smoked at least 100 cigarettes was as high as 16.3% (95% CI 15.8 − 16.8). The natural growth rate of AAE was available in seven studies at a median follow up of 5.9 years. The growth rate was estimated to be 0.82 mm/year (95% CI 0.20 − 1.45; Fig. 2B).

### *Enlargement, rupture, and mortality rates*

Longitudinal follow up data were separately analysed up to and beyond five years (Table 2). The pooled estimate

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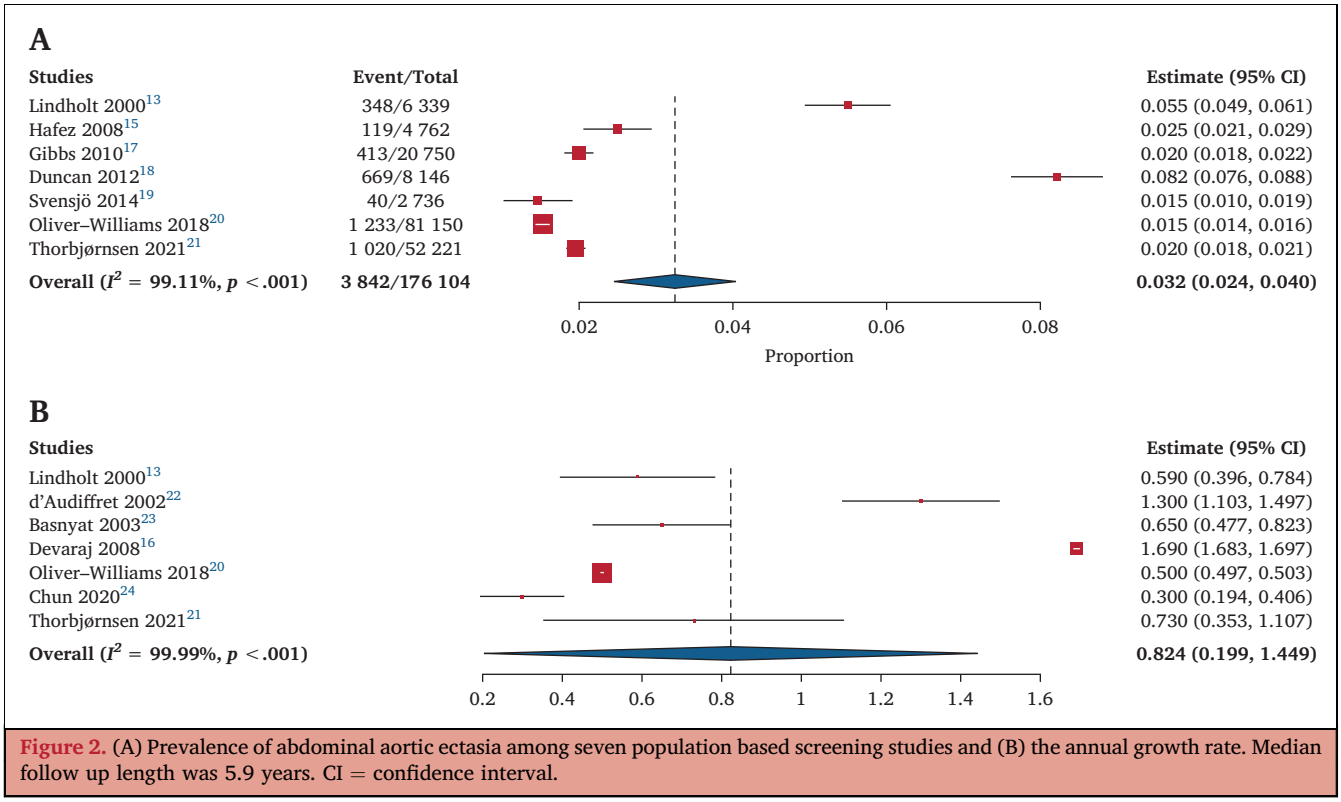


**Figure 2.** (A) Prevalence of abdominal aortic ectasia among seven population based screening studies and (B) the annual growth rate. Median follow up length was 5.9 years. CI = confidence interval.

revealed that 45.0% (95% CI 28.5 — 61.5) and 70.2% (95% CI 46.9 — 93.6) of AAE reached 30 mm within five years and after five years, respectively; 0.3% of AAE reached 55 mm in the first five years (95% CI 0 — 0.6) and 5.2% (95% CI 2.2 — 8.2) within 5 — 10 years. A study by Hafez *et al.*[15] was excluded from the analyses of aneurysm related death as the authors reported a disproportionately high mortality rate after elective surgery in their small cohort (three deaths in 17 patients; 17.6%), which is inappropriate for statistical interpretation of aneurysm related death. Pooled estimates of the rupture rate were 0.1% (95% CI 0 — 0.2) up to five years, and 0.4% (95% CI 0 — 0.7) beyond five years. Four cases of rupture were reported as follows: 47 mm aneurysm by ultrasound 10 weeks before rupture;[17] 37 mm infected aneurysm 4.5 years after initial screening; 47 mm saccular aneurysm at seven years; and 56 mm aneurysm at seven years, which grew rapidly at a rate of 7 mm/five months.[21] Additionally, 13 ruptures without details were reported in GASP.[25] Pooled analysis gave aneurysm related mortality rates of 0.1% (95% CI 0 — 0.2) up to five years and 0.2% (95% CI 0 — 0.5) after five years. Overall mortality data after the detection of AAE was available in 11 studies. During the first five years, the overall mortality rate for AAE was 7.5% (95% CI 3.9 — 11.0). The overall mortality rate over 5 — 10 years was 17.3% (95% CI 9.5 — 25.1). Cause of death was reported in five studies, comprising of a total of 360 fatal events in 2 624 patients (Table 3).[18,19,21,22,24] The leading cause of death was cancer ($n$ = 126; 35.0%), followed by a cardiovascular cause (chronic heart failure, myocardial infarction, and stroke) in 115 cases (31.9%) and chronic obstructive pulmonary disease in 27 cases (7.5%). Of note, ruptured aortic aneurysm and ruptured iliac artery aneurysm accounted for only one patient (0.3%) each. Relative overall mortality in patients with AAE *vs.* AAA was

**Table 2. Growth, rupture, aneurysm related death, and overall mortality rate of abdominal aortic ectasia (AAE) based on 12 studies describing a total of 8 369 patients**

| AAE feature | Follow up until five years | | Follow up from five to ten years | |
|---|---|---|---|---|
| | Estimate — % | 95% CI | Estimate — % | 95% CI |
| Exceeding 30 mm | 45.0 | 28.5–61.5 | 70.2 | 46.9–93.6 |
| Exceeding 55 mm | 0.3 | 0–0.6 | 5.2 | 2.2–8.2 |
| Rupture | 0.1 | 0–0.2 | 0.4* | 0–0.7 |
| Aneurysm related death | 0.1 | 0–0.2 | 0.2* | 0–0.5 |
| Overall mortality | 7.5 | 3.9–11.0 | 17.3 | 9.5–25.1 |

CI = confidence interval

* One study reported rupture and aneurysm related death in patients with at least 10 years of follow up. Otherwise, the data are derived from between five and 10 years of follow up.

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**Table 3. Causes of death of patients with abdominal aortic ectasia reported in the literature**

| Cause of death | Patients ($n$ = 360) |
|---|---|
| Cancer | 126 (35.0) |
| Cardiovascular cause | 115 (31.9) |
| Chronic obstructive pulmonary disease | 27 (7.5) |
| Renal failure | 2 (0.6) |
| Central nervous system | 2 (0.6) |
| Diabetes mellitus | 1 (0.3) |
| Aneurysm | 1 (0.3) |
| Ruptured iliac aneurysm | 1 (0.3) |
| Unknown | 49 (13.6) |
| Other | 36 (10.0) |

Data are presented as $n$ (%).

available in three studies. There was no significant difference in overall survival between patients with AAE and AAA (HR 0.62, 95% CI 0.32 — 1.21; $I^2$ = 77%, $p$ = .16 [Supplementary Fig. S3]).

### *Sensitivity analysis*

Sensitivity analysis only using population based screening studies was conducted. As shown in Supplementary Table S2, the estimated rates of enlargement to ≥ 30 mm and ≥ 55 mm up to five years were 51.7% (95% CI 37.6 —6 5.8) and 0.3% (95% CI 0 — 0.7), respectively. Risks of rupture, aneurysm related death, and overall mortality up to five years were 0.1% (95% CI 0 — 0.2), 0.1% (95% CI 0 — 0.2), and 8.7% (95% CI 5.0 — 12.4), respectively. During the 5 — 10 years after the initial screening, 78.4% (95% CI 61.7 — 95.0) of AAEs were estimated to reach 30 mm in diameter, while 9.3% (95% CI 3.2 — 15.5) of AAEs exceeded 55 mm. Similarly, rates of rupture, aneurysm related death, and overall mortality beyond five years were 0.7% (95% CI 0 — 1.5), 0.7% (95% CI 0 — 1.5), and 19.7% (95% CI 10.1 — 29.3), respectively.

## DISCUSSION

This meta-analysis considered 8 369 patients diagnosed with AAE and found that the prevalence of AAE was 3.2% among the general male population aged 65 — 75 years and grew at a mean of 0.82 mm/year during the first 5.9 year follow up period. Approximately half of AAEs reached 30 mm during the first five years after diagnosis; however, only 0.3% increased beyond 55 mm in maximum diameter. After 5 — 10 years, 5.2% and 0.4% of AAE reached 30 mm and 55 mm, respectively. The risk of rupture and aneurysm related death in the first five years were both estimated to be 0.1% (0.4% and 0.2%, respectively) after five years. However, the overall mortality risk was as high as 7.5% up to five years and 17.3% beyond five years, with cancer and cardiovascular diseases the most frequent causes of death. Overall mortality from AAE was not statistically different from that of AAA. These robust data from a large number of patients can address the clinical questions of the natural course and optimal follow up for AAE.

AAE is not a rare condition in the elderly male population, which can be encountered in various settings from general screening to vascular workups to incidental diagnoses at radiological imaging for different diseases, although the prevalence may vary depending on geographical location and changing epidemiology. It was found that, in general, AAEs do not grow rapidly; interestingly, 30% do not even reach 30 mm at 5 — 10 years. This contrast with the perception that AAE might be an AAA precursor lesion. Moreover, the risk of rupture was minimal. Some actual cases of rupture were either rapidly growing aneurysms, infected aneurysm, or saccular aneurysm, which are all considered to be special conditions with an increased risk of rupture, irrespective of aneurysm size.[2,3] The validity of the five year interval was confirmed, as AAEs almost never exceeded 55 mm or ruptured in the first five years. However, the rates of AAE exceeding 30 mm and 55 mm, as well as ruptures, increased beyond five years, showing the importance of follow up. Taken together, AAE can safely be observed at a five year interval with an almost negligible risk of rupture. This evidence is helpful in addressing the uneasiness of patients diagnosed with AAE, and to suppress recommendations of unreasonable hospital visits. A second screening at five years can help differentiate the risk of AAE growing to over 55 mm thereafter. Thorbjørnsen *et al.* reported that among stable AAEs remaining under 30 mm after five years, none exceeded 55 mm until 11 years.[21] To estimate the probability of reaching 30 mm at the five year follow up, current smoker and larger initial diameters were described as risk factors.[19,21,24] Therefore, physicians can encourage patients with these risk factors not to miss their five year follow up, as well as to quit smoking.[3]

The annual growth rate of small AAA reportedly correlates with the initial size: 30 — 34 mm, 1.81 mm/year; 35 — 39 mm, 2.66 mm/year; 40 — 44 mm, 3.86 mm/year; and 45 — 49 mm, 4.96 mm/year.[26] Interestingly, the current finding of the 0.83 mm/year annual growth rate for 25 — 29 mm AAE is on the linear relationship between the initial size and annual growth rate. As the estimation was that it would take 8.5 years for 30 mm sized AAAs to carry a 1% risk of rupture in men, it can be assumed that 25 — 29 mm AAEs will take even longer.[26] The risk of rupture for AAE, in general, is quite low, presumably until after 8.5 years. From this point of view, it can emphasised that the five year interval is safe regarding the risk of potential rupture.

Although the risks of rupture or aneurysm related lethal events are infrequent, the overall AAE mortality rate is 7.5% up to five years and 17% beyond five years. Notably, the overall mortality of AAE is no different from that of AAA. AAA is known to have high overall mortality risk, with similar causes of death (cancer, cardiovascular disease, and chronic obstructive pulmonary disease).[3,19,27] This is legitimate because AAAs are strongly associated with a history of smoking, and those causes of death are also smoking related diseases.[28] In other words, AAEs may serve as a predictor for overall mortality similar to the role of ankle brachial index.[29] For improved intervention in patients with AAE, risk factor modifications such as smoking cessation are recommended, similar to AAA. In the current systematic review, comparative mortality between population with

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normal aortas and with AAE was indeterminate because data were only available in two studies with limited numbers of patients.[18,19] Future studies are warranted to investigate whether AAEs actually have a worse prognostic impact on overall mortality than age and sex matched controls (those with normal sized aortas), and to clarify if any prevention or intervention such as enhanced screening for cancer or primary prevention with statins could reduce the overall mortality of patients with AAE.[30]

The current findings were gathered from an almost exclusively male cohort; therefore, care must be taken to extrapolate the data when following up women with AAE. Female sex is a well known risk factor for possible rapid growth and a four times higher risk of rupture in small AAAs, although the prevalence of AAA is lower than men.[26] A population based screening study targetting 70 year old women revealed that the prevalence of AAE was as low as 0.6%, with one elective repair and no rupture reported.[31] In fact, none of the programmes recommends general screening in women.[5,32] AAEs in women are likely to be incidentally encountered at imaging studies for other diseases. Again, future studies are required to better determine the fate of AAEs in women.

There are some limitations to this study. Although some studies guaranteed the measurement by trained examiners, heterogeneity in size criteria and non-available information regarding caliper placement and cardiac cycle at measurement could potentially affect the prevalence estimate of AAE. Exact calculation of the annual growth rate was not available in three studies. The reports by Svensjö *et al.*[19] and Thorbjørnsen *et al.*[21] may potentially have some overlap, which was not explained in the original articles. The comorbidities of the screened patients were not fully available although like AAA, some factors such as smoking and diabetes can influence the prevalence, annual growth rate, or risk of rupture.[26] The study periods of the original articles are inevitably old, partly due to the long follow up period. Some studies include screening from the 1990s, when the demographic prevalence of cardiovascular risk factors among the general population may have been different. The regional and sex disproportion, and limited information on race/ethnicity, should be highlighted.

In conclusion, AAE generally grows slowly and only 0.3% reach a maximum diameter of 55 mm in the first five years, and the subsequent risk of rupture and aneurysm related death remain almost zero in the first five years after diagnosis. Therefore, a five year interval is recommended for secondary follow up. However, discontinuation of follow up is not recommended as 5.2% of AAE reach 55 mm and 0.4% rupture after five years. Overall mortality is 7.5% at five years, similar to that of AAA. Education for smoking cessation and early detection of major causes of death such as cancer and cardiovascular diseases are of particular importance for the population diagnosed with AAE.

## CONFLICT OF INTEREST STATEMENT AND FUNDING

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.2022.05.005.

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