

Increasing incidence of ruptured abdominal aortic aneurysm: A population-based study

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Objective: The aim of the present population-based study was to assess the trends of age- and gender-specific incidence of ruptured abdominal aortic aneurysm (rAAA).

Methods: Patients with rAAA from the city of Malmö, Sweden, were studied between 2000 and 2004. An analysis of trends of incidence and mortality of rAAA in Malmö was possible because of a previous population-based study on patients with rAAA between 1971 and 1986 (autopsy rate 85% compared with 25% for the time period 2000 to 2004). The in-hospital registry of Malmö University Hospital and the databases at the Department of Pathology, Malmö, and the Institution of Forensic Medicine, Lund, identified patients with rAAA, and the in-hospital registry identified all elective repairs for AAA.

Results: Compared with the time period 1971 to 1986, the overall incidence of rAAA significantly increased from 5.6 (95% confidence interval [CI], 4.9 to 6.3) to 10.6 (95% CI, 8.9 to 12.4) per 100,000 person-years (standardized mortality ratio, 1.6; 95% CI, 1.0 to 2.1). In men aged 60 to 69 and 70 to 79 years, the incidence increased significantly from 16 (95% CI, 11 to 21) and 56 (95% CI, 43 to 69) to 46 (95% CI, 28 to 63) and 117 (95% CI, 84 to 149) per 100,000 person-years, respectively, whereas no increase in the age-specific incidence in women could be demonstrated. The overall incidence of elective repair of AAA increased significantly from 3.4 (95% CI, 2.8 to 4.0) to 7.0 (95% CI, 5.6 to 8.4) per 100,000 person-years and increased most significantly from 12 (95% CI, 3.4 to 32) to 68 (95% CI, 34 to 102) per 100,000 person-years in men aged 80 to 89 years and from 5.1 (95% CI, 2.4 to 9.3) to 28 (95% CI, 15 to 41) per 100,000 person-years in women aged 70 to 79 years. The elective-acute repair ratio in women increased from 2.4 to 5.6 and decreased in men from 2.1 to 1.0.

Conclusions: Between 1971 to 1986 and 2000 to 2004, the incidence of rAAA increased significantly, despite a 100% increase in elective repairs and notwithstanding a potential for bias towards underestimation due to lower autopsy rates in recent years. The reason behind this increase is unclear, and further studies are needed to identify risk groups for direction of effective prevention and screening. (*J Vasc Surg* 2006;44:237-43.)

Population screening programs for abdominal aortic aneurysm (AAA), such as a single ultrasound scan in men at 65 years old,^{1,2} have increasingly been advocated by vascular surgeons as a way to reduce the total mortality in ruptured AAA (rAAA). This view is supported by evidence from population-based, randomized controlled trials.³⁻⁵ However, the background data on the incidence and mortality of rAAA to motivate such a program is founded on using death-cause registries and administrative databases, which may have limited precision and validity because of low autopsy rates.⁶⁻¹⁰

The high average autopsy rate of 85% in the population of Malmö between 1971 and 1986 enabled a solid estimation of the incidence of rAAA at that time.¹¹ With recent advances in diagnostics and the establishment of less invasive therapy, such as endovascular AAA repair (EVAR) in Malmö,^{12,13} the possibilities to detect and treat AAA in

routine medical care have also improved. More patients can be treated because EVAR can be performed under local anesthesia, and even fragile octogenarians, otherwise unfit for open surgery, may tolerate this treatment.¹⁴ The potential added benefit from a generalized screening program must be viewed in this context.

Furthermore, epidemiologic changes that might result from changes in population exposure to risk factors for atherosclerotic diseases and aneurysm development must be considered.¹⁵ A decision on screening has wide implications and needs to be backed by robust incidence estimates, targeting the intended target population and reflecting contemporary conditions. Hence, we set out to evaluate the current age- and gender-specific incidence of rAAA in the Malmö population and to view these assessments against the previously reported incidence estimates in Malmö. The substantial reduction in autopsy frequency that followed changes in legislation might bias the assessment of contemporary incidence towards underestimation, and we therefore realized, a priori, the limitations of the present study to detect a true reduction in incidence rates between the two time periods.

MATERIALS AND METHODS

Study population and setting. This study was approved by the Research Ethics Committee of the University

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Competition of interest: none.

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0741-5214/\$32.00

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doi:10.1016/j.jvs.2006.04.037

of Lund. Between 2000 and 2004, the population of Malmö increased from 260,000 to 269,000, (Swedish Central Bureau of Statistics). The population in 2002 was used as the reference population. The city has one hospital for somatic diseases, the Malmö University Hospital. During the 5-year study period (January 1, 2000 to December 31, 2004), all surgical procedures and all diagnoses assigned to inpatients upon discharge or death were classified according to the International Classification of Disease, 10th edition (ICD-10) code and collected in a computerized registry.

The Department of Pathology is the sole referral unit for postmortem examinations in the city. During the period, 14,964 deaths occurred among the Malmö population. In all, 2711 clinical autopsies and 1058 forensic autopsies were performed on Malmö patients, resulting in an average 25% autopsy rate.

The evaluation of incidence changes over time was based solely on the Malmö city population and compared with a previously published⁸ study of patients with rAAA from the population of Malmö between 1971 and 1986. The population in 1978 (240,000 inhabitants) was used as reference population in that study (The Malmö Statistical Year book).

Retrieval of rAAA cases. The identification of Malmö patients with the diagnosis of rAAA managed operatively or nonoperatively was based on ICD code I71.3. All medical records were found and analyzed.

Rupture was defined as extravasation of blood or hematoma outside the AAA at autopsy or on computed tomography (CT) examination, or a hematoma outside the AAA at open repair, or both. In the absence of autopsy, operation, or CT, a patient with a known AAA with acute symptoms compatible with rupture was also considered to have a rAAA. Patients with rAAA referred from areas outside of Malmö or who lived outside of the city of Malmö were excluded from the analysis. Patients with inflammatory or symptomatic AAA without rupture were excluded from this study.

Retrieval of clinical and forensic autopsy-verified cases. The clinical autopsies were performed according to a standardized detailed protocol. Findings were coded according to the Systematized Nomenclature of Medicine (SNOMED), as defined by the College of American Pathologists. The topographic code for aorta (t42) and the code of diagnosis for aneurysm (m32400) or dissection (m32470) were used in a broad search to identify all individuals with rAAA. The computerized autopsy registry at Department of Pathology, Malmö University Hospital, was used to identify and analyze the protocols of the patients with ruptured AAA.

The forensic autopsies were performed at the Institution of Forensic Medicine in Lund, Lund University Hospital, on demand from the police authorities. Only deceased persons referred from the police office in Malmö were analyzed in a computerized database.

Policies for elective surgery in Malmö over time. Elective surgery for an AAA from 1971 to 1986 and 2000

to 2004 was indicated, in the absence of significant comorbidity, if the maximal aneurysm diameter measured on ultrasound was ≥ 5.0 cm (≥ 5.5 cm in men or ≥ 5.0 cm in women measured at CT). After 1993, the growth rate of opportunistically detected small sized AAAs in Malmö patients was surveilled with ultrasound scans at regular intervals. The waiting times for open repair within the two time periods was 1 to 2 weeks, whereas EVAR patients in the latter time period had to undergo more morphologic evaluation with CT/aortography and had to wait another 1 to 8 weeks for the ordered stent-grafts to arrive. EVAR was the first choice method in elective repairs between 2000 and 2004. Relatively young (<65 years old) and fit patients with long life expectancy were selected for open repair, even if the aneurysm morphology was suitable for EVAR.

Evaluation of elective vs acute AAA surgery. Patients within the population of Malmö who underwent elective surgery for asymptomatic AAA were identified (ICD code I714 and the respective codes for open repair or EVAR). All medical records were retrieved and evaluated. Mortality data were obtained by record linkage with the Swedish Population Registry. The patients were monitored from the day of surgery until death or January 1, 2006. Median follow-up time was 29.5 months (range, 0 to 72 months).

Statistical methods. Cause-specific mortality ratios were expressed as number of deaths from rAAA per thousand deaths or thousand autopsies and analyzed in relation to age and gender. Age- and gender-specific incidence rates were based on the number of cases with rAAA or elective repairs of AAA, respectively, and were expressed as number of cases per 100,000 person-years. Age- and gender-specific event rates and incidence rates from 1971 to 1986 and 2000 to 2004 were computed by using, respectively, the 1978 and 2002 Malmö population census as reference. Confidence intervals (CIs) were calculated assuming a Poisson distribution of events, using the exact method for $N < 15$ and the normal approximation for larger numbers. Time trends in rAAA risk were investigated by indirect standardization for population changes between 1978 and 2002. Thus, the annual event rates from 2000 to 2004 that would be expected were computed from the previously found age- and gender-specific morbidity rates from 1971 to 1986, with expression of the standardized morbidity ratio (SMR) between observed and expected annual event rates.

Differences in proportions were evaluated using the χ^2 test. Age distributions were expressed in terms of median and range, and the Mann-Whitney U test was used to evaluate differences. Survival analysis comparing operation for rAAA vs elective operation, and adjusting for age, was performed by using Cox's proportional hazards model, with computation of relative risks and 95% CIs.

RESULTS

Retrieval and characteristics of rAAA patients. During the 5-year period, 141 rAAA patients were identified. Median age was 77 years (range, 54 to 96 years), and 116 patients (82 %) were men. Three patients were <60

Table I. Deaths from ruptured abdominal aortic aneurysm in Malmö, 2000 to 2004: cause-specific mortality ratios in relation to age and gender

Age group	Deaths 2000-2004			Deaths from RAAA			Cause-specific mortality (95 % CI)		
							/1000 deaths		
	M	F	M+F	M	F	M+F	M	F	M+F
0-54	640	344	984	1	0	1	1.6 (0.0-8.7)	0.0 (0.0-10.7)	1.0 (0.0-5.7)
55-64	645	436	1081	3	2	5	4.7 (1.0-13.6)	4.6 (0.6-16.6)	4.6 (1.5-10.8)
65-74	1261	936	2197	22	6	28	17.4 (10.2-24.7)	6.4 (2.4-14.0)	12.7 (8.0-17.5)
75-84	2345	2335	4680	43	9	52	18.3 (12.9-23.8)	3.9 (1.8-7.3)	11.1 (8.1-14.1)
85-94	1641	3476	5117	11	6	17	6.7 (3.3-12.0)	1.7 (0.6-3.8)	3.3 (1.7-4.9)
95+	158	728	886	1	0	1	6.3 (0.2-35.3)	0.0 (0.0-5.1)	1.1 (0.0-6.3)
Total	6700	8264	14964	81	23	104	12.1 (9.5-14.7)	2.8 (1.6-3.9)	7.0 (5.6-8.3)

years old and seven were >90 years old. The diagnosis was established during surgery in 67 patients (48%). Another 51 patients (36%) were under hospital care but not subject to surgery. The diagnosis was established at clinical autopsy in 30 patients, after acute CT scan in eight, and on clinical grounds (previous known AAA together with acute symptoms) in 13. An additional 23 patients (16%) who had died outside the hospital were identified. Nine had been referred for autopsy from the police authorities to the Institution of Forensic Medicine and 14 from general practitioners to the Department of Pathology. Thus, of all 141 rAAA patients, the basis for diagnosis was surgery in 67 (48%), autopsy in 53 (38%), and noninvasive assessment in 21 (15%).

Management of rAAA patients. Of the 118 rAAA patients under hospital care, 83 (70%) were initially managed by surgeons or vascular surgeons. Among 35 in-hospital patients, 24 (20%) were initially managed by internists, 11 (9%) by physicians from other specialties, and only eight (23%) were operated on. Thus, only 59% (83/141) of all detected rAAA cases were managed by surgeons. Of the 141 rAAA patients, 74 (52%) were not subject to surgery. Death in-hospital, without surgery, occurred in 51 (43%) of 118 cases, and 15 (29%) of these 51 patients were assessed in time but were not considered for surgery: The documented factors that contributed to the nonoperative management of these 15 patients were high age in 13, cancer in 3, suprarenal AAA in 2, chronic obstructive pulmonary disease in 1, and patient refusal of surgery in 1. Median age was 75 years (range, 60 to 88 years) in operated on patients vs 77 years (range, 54 to 96 years) in nonoperated patients ($P = .036$).

Epidemiology of rAAA. The early mortality in rAAA, defined as death outside the hospital or during the hospitalization, was 92% (23/25) in women and 70% (81/116) in men, for a total of 74% (104/141). Of patients < 75 years of age, 61% died (34/56) compared with 82% (70/85) in older patients ($P = .006$).

The overall cause-specific mortality rate from rAAA was 7.0 (95% CI 5.6 to 8.3) per 1000 deaths (Table I), and increased to 18 per 1000 deaths in men aged 65 to 74 and 75 to 84 years. The youngest patient with a fatal rAAA was 54 years old. The overall incidence was 11 per 100,000 person-years, increasing to 154 per 100,000 person-years in men aged 80 to 89 years (Table I).

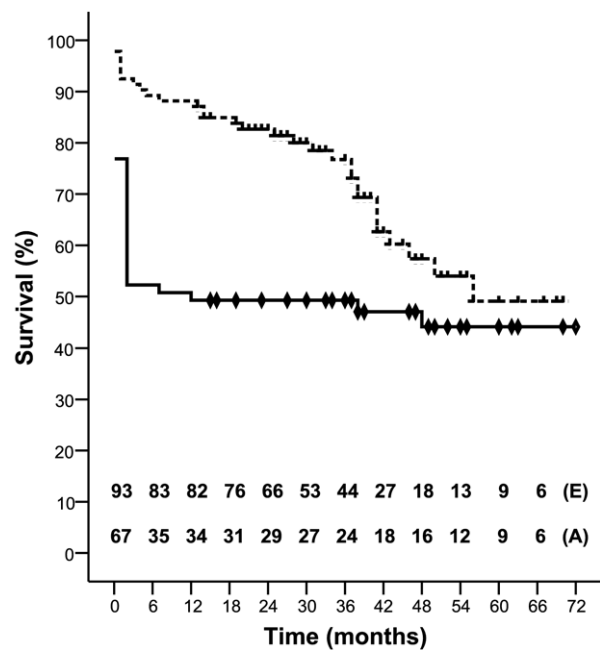


Fig 1. Long term survival after elective surgery for abdominal aortic aneurysm (AAA) (E; dashed line) vs acute surgery for ruptured AAA (A; solid line) in Malmö 2000 to 2004. Censored patients are marked with ticks. Numbers above time axis denote numbers at risk in each group at respective time point.

Patients undergoing elective repair of AAA.

Elective repair of AAA was performed in 93 patients during the study period, 66 (71%) of whom were treated with EVAR. The median age was 73 years (range, 52 to 82 years) among the 28 women (30%) compared with 73 years (range, 56 to 86 years) ($P = .34$) among the 65 men (70%). The median age of patients who had elective operations was 73 years (range, 52 to 86 years) and was higher for patients undergoing EVAR (median, 75 years) compared with those undergoing open repair (median, 72 years) ($P = .014$). Open repair was performed significantly more often in women (57%, 16/28) compared with men (17%, 11/65) ($P < .001$). In-hospital mortality was 7.7% (5/65) for

Table II. Epidemiologic and clinical characteristics in 1971 to 1986 (Bengtsson et al) and 2000 to 2004 (Acosta et al) studies of ruptured abdominal aortic aneurysm

<i>Study</i>	<i>Bengtsson et al.</i>	<i>Acosta et al.</i>
Demographic data		
Study period	1971-1986	2000-2004
Malmö population	240 000	265 000
Percentage >75 years	6%	10%
Ratio F:M in persons >75 years	2:1	1:9:1
Autopsy frequency	85%	25%
Incidence of rAAA*		
Men	8.4 (7.1-9.7)	18.1 (14.8-21.4)
60-69 years	16.0 (10.7-21.3)	45.6 (27.7-63.4)
70-79 years	55.8 (42.7-68.9)	117 (84.3-149)
80+ years	113 (75.9-150)	154 (102-206)
Women	3.0 (2.2-3.8)	3.6 (2.2-5.1)
60-69 years	0.8 (0.1-3.6)	6.5 (1.8-16.6)
70-79 years	8.6 (4.5-12.7)	11.5 (4.6-23.7)
80+ years	43.0 (28.3-57.7)	25.1 (12.5-44.9)
All	5.6 (4.9-6.3)	10.6 (8.9-12.4)
Surgically treated /all rAAA (%) cases		
Men	52/155 (34)	62/116 (53)
Women	9/60 (15)	5/25 (20)
All	61/215 (28)	67/141 (48)
Incidence of elective AAA surgery*		
Men	5.9 (4.7-7.0)	10.1 (7.7-12.6)
60-69 years	22.9 (16.5-29.2)	34.6 (19.1-50.2)
70-79 years	33.5 (23.4-43.6)	67.7 (43.1-92.3)
80+ years	12.5 (3.4-32.1)	68.1 (33.6-102)
Women	1.1 (0.6-1.6)	4.1 (2.6-5.6)
60-69 years	2.8 (1.1-5.8)	12.9 (5.6-25.5)
70-79 years	5.1 (2.4-9.3)	27.9 (14.6-41.1)
80+ years	1.3 (0.0-7.3)	4.6 (0.6-16.5)
All	3.4 (2.8-4.0)	7.0 (5.6-8.4)
Ratio (N/N) elective/acute surgery		
Men	2.1 (108 / 52)	1.0 (65 / 62)
Women	2.4 (22 / 9)	5.6 (28 / 5)
All	2.1 (130 / 61)	1.4 (93 / 67)

*Per 100,00 person years (95% CI).

men and 7.1% (2/28) for women ($P = .93$). The in-hospital mortality was 11.1% (3/27) for the open repair and 6.1% (4/66) for the EVAR groups ($P = .40$).

Long-term survival after surgery for rAAA vs AAA.

The mortality rate after surgery for rAAA was 28.0 deaths per 100 person-years compared with 12.1 per 100 person-years after elective surgery ($P < .001$), with a relative risk of 1.9 (95% CI, 1.1 to 3.2) by age-adjusted Cox regression model. The survival curves converged during the latter part of the follow-up period (Fig 1).

Time trends in rAAA management and risk.

Between 1971 to 1986 and 2000 to 2004, the autopsy rate declined from 85% to 25% (Table II). The proportion of rAAA cases at autopsy only was 70% (151/215) and 38% (53/141), respectively, and the proportions for men and women in the first time period were 66% (103/155) and 80% (48/60), and 33% (38/116) and 60% (15/25), respectively, in the second time period. The proportion of deaths outside hospital was 42% and 17%, respectively. The proportion of deaths in-hospital without surgery was 51% and 43%, respectively. The surgical activity of all rAAA was 34% and 48%, respectively. Documentation of ruptured

suprarenal aortic aneurysms was found in 10 (5%) of 215 patients, of which four underwent attempted repair, between 1971 to 1986, compared with five (4%) of 141 patients, with three attempted repairs (two EVAR and one open repair), in the latter time period. Only one patient in the latter epoch had a CT-verified contained rupture. The surgical mortality decreased from 57% to 46%. The total mortality was 88% and 74%, respectively.

The overall incidence of elective repair of AAA increased significantly from 3.4 (95% CI 2.8 to 4.0) to 7.0 (95% CI 5.6 to 8.4) per 100,000 person years and increased most significantly from 12 (95% CI 3.4 to 32) to 68 (95% CI 34 to 102) per 100,000 person years in men aged 80 to 89 years and from 5.1 (95% CI 2.4 to 9.3) to 28 (95% CI 15 to 41) per 100,000 person-years in women 70 to 79 years old. The total elective-acute repair ratio decreased from 2.1 to 1.4: The ratio decreased in men from 2.1 to 1.0, and the ratio increased from 2.4 to 5.6 in women (Table II).

The crude event rate between 1971 and 1986 of 13.4 events per year corresponds to an expected event rate of 18.0 events per year when standardizing to the 2002 population (Table III). The higher observed event rate of 28.2

Table III. Time trends in ruptured abdominal aortic aneurysm risk: Observed incidence and annual event rates 2000 to 2004 vs expected, based on standardization for population changes over time

<i>rAAA 2000-2004</i>																			
<i>Age groups</i>	<i>Population 2002</i>			<i>N</i>			<i>N/year</i>			<i>Incidence[#]</i>									
	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>							
0-59	104313	103902	208215	7	3	10	1.4	0.6	2.0				1.3	0.6	1.0				
60-69	10974	12364	23338	25	4	29	5.0	0.8	5.8				45.6	6.5	24.9				
70-79	8570	12190	20760	50	7	57	10.0	1.4	11.4				117	11.5	54.9				
80-89	4407	8761	13168	34	11	45	6.8	2.2	9.0				154	25.1	68.3				
All	128264	137217	265481	116	25	141	23.2	5.0	28.2				18.1	3.6	10.6				

<i>rAAA 1971-1986</i>																			
<i>Age groups</i>	<i>Population 1978</i>			<i>N</i>			<i>Crude /year</i>			<i>Exp*/year</i>			<i>Crude estimates</i>			<i>Standardized*</i>			
	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	<i>M</i>	<i>F</i>	<i>M+F</i>	
0-59	91819	92223	184042	14	8	22	0.9	0.5	1.4	1.0	0.6	1.6	1.0	0.5	0.7	1.1	0.6	0.8	
60-69	13672	15625	29297	35	2	37	2.2	0.1	2.3	1.8	0.1	1.9	16.0	0.8	7.9	12.8	0.6	6.3	
70-79	7840	12355	20195	70	17	87	4.4	1.1	5.4	4.8	1.0	5.8	55.8	8.6	26.9	61.0	8.5	28.9	
80-89	1996	4797	6793	36	33	69	2.3	2.1	4.3	5.0	3.8	8.7	113	43.0	63.5	249	78.5	129	
All	115327	125000	240327	155	60	215	9.7	3.8	13.4	12.5	5.5	18.0	8.4	3.0	5.6	10.8	4.4	7.5	

Observed (O) 28.2 events/year.
Expected (E) 18.0 events/year.
*Standardized to the 2002 population.
#per 100,000 person years.

per year during 2000 to 2004 corresponds to a SMR of 1.6 (95% CI, 1.0 to 2.1). Crude and standardized incidence rates in 1971 to 1986 were 5.6 and 7.5 per 100,000 person-years compared with the observed 10.6 events per 100,000 person years. The incidence of rAAA significantly increased between the two time periods 1971 to 1986 and 2000 to 2004 among men overall, men aged 60 to 69, and 70 to 79 years, whereas for women, irrespective of age category, no corresponding increase in incidence could be demonstrated (Table II, Fig 2).

Diagnosed AAA before rupture. Denial for elective repair of an AAA before rupture was documented in 19% (40/215) between 1971 and 1986, and three patients with known small size AAAs (<5.0 cm) were operated on due to rupture.

In the latter time period, 13 (9%) of 141 patients with rAAA were denied elective repair for an AAA. Three patients with known large AAAs (55, 68, and 77 mm of maximal aneurysm diameter) and severe comorbidities were evaluated for AAA repair but ruptured before decision was made. The AAAs in two of these three patients were not repaired, and all died.

DISCUSSION

Despite the imputed effect on detection rate after the dramatic decrease in autopsy frequency between the two study periods, especially among the elderly,¹⁶ and a twofold increase in elective repairs of AAA, the present study was able to demonstrate, also after standardization for changes in the population, a significant increase in incidence of

rAAA in men aged 60 to 69 and 70 to 79 years old, but no such changes over time could be demonstrated in women. Given the likely underestimation of incidence in age groups with notably low autopsy rates, such as the oldest, with a higher proportion of women, other factors that might contribute to this discrepancy should be considered. One explanation might be that the average life span in the male Malmö population has increased.¹⁷ With declining incidence and mortality rates of myocardial infarction in Malmö,¹⁸⁻¹⁹ the long-term survival¹⁷ has also increased in men with cardiovascular disease, who then might become

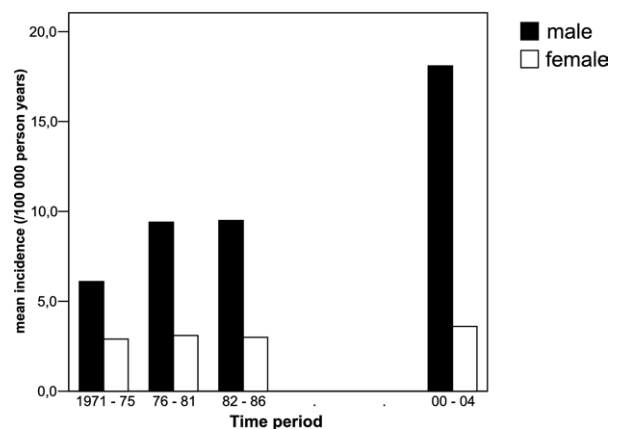


Fig 2. Total population incidence of ruptured abdominal aortic aneurysm in Malmö: evolution between 1971 and 2004.

exposed to mortality from a more gradually developing AAA disease.

One should also consider the possibility that our findings may reflect a true increase in the incidence of AAA disease over time. A previous study based on nearly 46,000 autopsies between 1958 and 1986 in Malmö showed an age-standardized increase in prevalence of AAA among men and women,²⁰ and it is not unlikely that this trend of increase has continued since 1986. The continuous decline in the number of male smokers in Malmö¹⁵ seems to have prevented ischemic heart disease-related events in a relative short time period, whereas a decrease in the prevalence of AAA first might be expected some decades later.

The absence of an apparent increase in the incidence of rAAA in women should also be viewed in light of the corresponding increase over time of elective repairs. The high 5.6 elective-acute repair ratio of AAA in women may be explained by a higher therapeutic activity in women owing to the awareness of that female sex is associated with an increased risk of rupture.²¹ However, the significantly higher mortality in women with rAAA seems to be related to the observation that only one in five of rAAA in women were actually operated on. Although this lower surgical activity to some extent might reflect a higher comorbidity in women,²² one must consider the possibility of a gender difference in disease awareness, with a perception of male preponderance.

Apart from differences in epidemiology, the present study indicated that the aortic aneurysm morphology in women, compared with men, were quite more complex to handle. The significantly higher percentage of elective open repair in women could reflect a higher percentage of anatomic restrictions for EVAR in women because of very short proximal aneurysm necks and, to some extent, narrow iliac arteries.¹² This finding suggests that aneurysm repair may be more difficult in women, irrespective of operative method, and may be partly responsible for the lower survival rate seen in women after surgery for rupture.²²

Male sex and old age are strong and independent risk factors for AAA²³ and thereby define a risk group that might benefit from screening. The risks and benefits of screening programs are notably difficult to assess. Our data suggest that 77 of all 104 AAA deaths happened in men > 65 years, which may serve as reference data in screening debates. Furthermore, almost all of the patients with rAAA between 2000 and 2004 were not known to have an AAA before rupture, despite an increased use of radiologic imaging between the two time periods, which may motivate action towards screening. In the present population, the 5-year survival rate after elective repairs of opportunistically detected AAA was not superior compared with those undergoing surgery for rAAA, which may be attributed to a high prevalence of coronary artery disease among patients with AAA.²⁴ It is therefore suggested that a single ultrasound scan of the abdominal aorta should be included as part in a wider preventive health care program.

One limitation of the study was the contemporary low autopsy frequency of 25%. The autopsy rates have declined

steadily¹⁶ since 1992 because of major changes in the Swedish autopsy law. The responsibility for deceased individuals outside of the hospital shifted from the police authorities to the general health care system, which in practice, meant that a forensic autopsy no longer was mandatory among these individuals. Second, and of vital importance, the relatives' opinion towards postmortem examination have been respected, which have had a great impact on the much decreased autopsy rate. Nevertheless, autopsy rate data have to be taken into account when an epidemiologic analysis is performed on rAAA.

CONCLUSION

Between 1971 to 1986 and 2000 to 2004, the incidence of rAAA increased significantly, despite a 100% increase in elective repairs and notwithstanding a potential for bias due to lower autopsy rates in recent years towards underestimation. The reason behind this increase is unclear, and further studies are needed to identify risk groups for direction of effective prevention and screening.

AUTHOR CONTRIBUTIONS

Conception and design: SA, MÖ, HB, DB, BL, ZZ

Analysis and interpretation: SA, MÖ, HB, DB, BL, ZZ

Data collection: SA, HB, ZZ

Writing the article: SA, MÖ, DB, BL, Critical revision of the article: SA, MÖ, HB, DB, BL, ZZ

Final approval of the article: SA, MÖ, HB, DB, BL, ZZ

Statistical analysis: SA, MÖ

Obtained funding: SA

Overall responsibility: SA

REFERENCES

1. Wanhainen A, Lundkvist J, Bergqvist D, Björck M. Cost-effectiveness of different screening strategies for abdominal aortic aneurysm. *J Vasc Surg* 2005;41:741-51.
2. Henriksson M, Lundgren F. Decision-analytical model with lifetime estimation of costs and health outcomes for one-time screening for abdominal aortic aneurysm in 65-year old men. *Br J Surg* 2005;92:976-83.
3. Ashton HA, Buxton MJ, Day NE, Kim LG, Marteau TM, Scott RA, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. *Lancet* 2002;360:1531-9.
4. Norman PE, Jamrozik K, Lawrence-Brown MM, Le MT, Spencer CA, Tuohy RJ, et al. Population based randomised controlled trial on impact of screening on mortality from abdominal aortic aneurysm. *BMJ* 2004;329:1259.
5. Lindholt JS, Juul S, Fasting H, Henneberg EW. Screening for abdominal aortic aneurysm: single centre randomised controlled trial. *BMJ* 2005;330:750.
6. Best V, Price J, Fowkes F. Persistent increase in the incidence of abdominal aortic aneurysm in Scotland, 1981-2000. *Br J Surg* 2003;90:1510-5.
7. Filipovic M, Goldacre M, Roberts S, Yeates D, Duncan M, Cook-Mozaffari P. Trends in mortality and hospital admission rates for abdominal aortic aneurysm in England and Wales, 1979-1999. *Br J Surg* 2005;92:968-75.
8. Gillum R. Epidemiology of aortic aneurysm in the United States. *J Clin Epidemiol* 1995;48:1289-98.
9. Reitsma J, Pleumeekers H, Hoes A, Kleijnen J, de Groot R, Jacobs M, et al. Increasing incidence of aneurysms of the abdominal aorta in The Netherlands. *Eur J Vasc Endovasc Surg* 1996;12:446-51.

10. Drott C, Arfvidsson B, Örténwall P, Lundholm K. Utbredd begrepps-förvirring och felkodningar vid aortarufturer. Förvånande låg precision i dödsorsaksregistret. *Läkartidningen* 1991;88:2137-9.
11. Bengtsson H, Bergqvist D. Ruptured abdominal aortic aneurysm-a population-based study. *J Vasc Surg* 1993;18:74-80.
12. Dias N, Ivancev K, Malina M, Resch T, Lindblad B, Sonesson B. Does the wide application of endovascular AAA repair affect the results of open surgery? *Eur J Vasc Endovasc Surg* 2003;26:188-94.
13. Resch T, Malina M, Lindblad B, Dias N, Sonesson B, Ivancev K. Endovascular repair of ruptured abdominal aortic aneurysms: Logistics and short-term results. *J Endovasc Ther* 2003;10:440-6.
14. EVAR Trial participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): randomised controlled trial. *Lancet* 2005;365:2187-92.
15. Public Health Report 1996. Malmö City Council. Malmö City Council and Department of Community Medicine, Malmö University Hospital, 1996.
16. Lindström P, Janzon L, Sternby NH. Declining autopsy rate in Sweden: a study of causes and consequences in Malmö, Sweden. *J Intern Med* 1997;2:157-65.
17. Malmström M, Sundqvist J, Bajekal M, Johansson SE. Ten-year trends in all-cause mortality and coronary heart disease mortality in socio-economically diverse neighbourhoods. *Public Health* 1999;113:279-84.
18. Engström G, Göransson M, Hansen O, Hedblad B, Tyden P, Tödt T, et al. Trends in long-term survival after myocardial infarction: less favourable patterns for patients from deprived areas. *J Intern med* 2000;248:425-34.
19. Tyden P, Hansen O, Janzon L. Intra-urban variations in incidence and mortality in myocardial infarction. A study from the myocardial infarction register in the city of Malmö, Sweden. *Eur Heart J* 1998;19:1795-801.
20. Bengtsson H, Bergqvist D, Sternby N H. Increasing prevalence of abdominal aortic aneurysms. A necropsy study. *Eur J Surg* 1992;158:19-23.
21. Brown LC, Powell JT. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial Participants. *Ann Surg* 1999;230:289-96.
22. Norman P, Semmens J, Lawrence-Brown M, Holman C. Long term relative survival after surgery for abdominal aortic aneurysm in Western Australia: population-based study. *BMJ* 1998;317:852-6.
23. Lederle F, Johnson G, Wilson S, Chute E, Hye R, Makaroun M, and the Aneurysm Detection and Management Veterans Affairs Cooperative Study Investigators. The aneurysm detection and management study screening program. Validation cohort and final results. *Arch Intern Med* 2000;160:1425-30.
24. Bergqvist D, Bengtsson H, Sternby NH. Associated atherosclerotic manifestations. In Greenhalgh RM, Mannick JA, Powell JT, editors. *The cause and management of aneurysms*. London: WB Saunders Company; 1990.

Submitted Feb 21, 2006; accepted Apr 16, 2006.