The Rise and Fall of Abdominal Aortic Aneurysm

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In the current issue of Circulation, Svensjö et al1 report on an abdominal aortic aneurysm (AAA) screening program for 65-year-old men in central Sweden. Using the standard screening test (ultrasound) and AAA definition (an aortic diameter of 3.0 cm or larger), they found a prevalence of AAA of 1.7%, which they describe as “the lowest reported in a predominantly white population to this date,” and postulate that the explanation “may be an overall decrease of the disease in the population.” Two other recent studies have observed markedly decreased mortality from AAA in men in the past 2 decades in Western countries, with smaller decreases in women. Sandiford et al2 reported that AAA mortality in men dropped by more than half from 1991 to 2007 in New Zealand, and by a third from 1996 to 2007 in England and Wales; and Norman et al3 found a 38% decline in AAA mortality in men from 1999 to 2006 in Australia, adding that “this suggests a true fall in incidence of AAAs.” These 3 studies may come as a surprise to those who remember a time not so long ago when AAA was a disease on the rise. The first alerts back then came from autopsy studies that charted an increasing occurrence of arteriosclerotic (many would now question this etiology) aortic aneurysms beginning in the late 1940s, just as syphilitic aneurysms went into decline.4 These observations were confirmed in a population-based study from Rochester, Minnesota, that found a marked increase in the incidence of AAA between 1951 and 1980 that seemed to reflect more than just improved case ascertainment.5 This increase was in contrast to the pattern seen for atherosclerotic occlusive vascular disease, which declined after 1960. Lilienfeld et al6 calculated age-adjusted rates of aortic aneurysm and AAA mortality from the US Vital Statistics to demonstrate a severalfold rise from 1951 to 1968 before leveling off at a high plateau. Numerous ultrasound screening studies subsequently documented a remarkably high prevalence of AAA in older men, >5% in some populations.7 These findings prompted randomized trials of ultrasound screening that demonstrated a large reduction in AAA-related mortality in older men. Guidelines and community screening programs followed, and AAA went from being a somewhat unusual and exotic disorder to one of the common conditions that primary care physicians address every day.

So now, with national screening programs gearing up around the world, we learn from these new studies that the target condition may be in steep decline. One might well ask what happened, but the answer, at least in large part, seems clear. The authors of all 3 studies cite reductions in tobacco use in their respective countries as the most likely explanation.1–3 Norman et al point to a drop in the adult prevalence of smoking in Australia from 35% in 1980 to 23% in 2001. Svensjö et al note that the proportion of 65-year-old Swedish men who were daily smokers fell from 32% in 1980 to 11% in 2007.

The strong association between smoking and aortic aneurysm was described by Hammond and Horn in 1958.8 A systematic review that included this and other large studies relating smoking behavior to subsequent mortality showed that the relationship between smoking and aortic aneurysm was several times stronger than the relationship between smoking and occlusive vascular disease.9 A large AAA screening study of US veterans found that smoking accounted for >70% of all AAAs in that population of mostly older men,10 a finding that Svensjö et al1 have confirmed in their similar population. Collin suggested in 1988 that the rise in AAA deaths in the preceding 35 years in England and Wales was “a cohort effect associated with the pattern of tobacco addiction in the 20th century,” particularly affecting those who came of age “between 1916 and 1948 when cigarette smoking was common and fashionable.”11 Svensjö et al go on to speculate that “a further decline in [AAA] prevalence could be expected over time, as the rate of daily smoking among men continues to drop in Sweden.”

Are similar trends evident in the United States? The Figure shows US annual adult per capita cigarette consumption12,13 and age-adjusted AAA mortality per 100 000 white men14 by calendar year. Included are abdominal, thoracoabdominal, and unspecified aortic aneurysm, both ruptured and unruptured (largely elective repair deaths). Besides providing a compelling illustration of the connection between smoking and disease, the Figure lends support to the findings from the other Western countries regarding the declining impact of AAA. The decline may have seemed less dramatic to practicing physicians than it appears in the age-adjusted, standardized Figure because of the concurrent increases in the US population and in the proportion of the population that is older. However, even the raw numbers of AAA-related deaths in all Americans dropped by 30% from their peak of 11 485 in 1991 to fewer than 8000 in 2007.14 This is still a lot of deaths, and our efforts to control the disease should be no more relaxed than were efforts to control coronary artery disease following its decline, but it is prudent to consider the possible effects of this trend.

One obvious expectation is that the number of AAA repair operations would also decline. Yet Norman et al3 have found

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only a small decrease in hospital discharges for nonruptured AAA (again the elective repairs) since 1999 in Australia, and Svensjö et al have found “no signs of a decrease in AAA repair workload in Sweden.” In the United States, the per capita incidence of elective AAA repair remained essentially constant from 1979 to 2002. Possible explanations for the discrepancy between disease and treatment include the following: (1) there may be a long delay before the decline extends to AAAs large enough to require repair, as suggested by Svensjö et al; (2) the reduction in mortality (though not prevalence at screening) could at least in part be the result of successful therapy with elective AAA repair; (3) the workload may be maintained by increased repair of more prevalent smaller AAAs, for which there is some evidence; and (4) the lower perioperative mortality of endovascular repair may have decreased the number of deaths attributed to AAA (although randomized trial data suggest that this would not reflect true long-term gains, and again would not affect screening prevalence). Future studies may determine the proportional contribution of these and other factors.

Another expected effect of a decline in AAA is on screening programs, and the authors of all 3 studies considered their findings to have important implications for AAA screening. Screening programs that fail to target high-risk populations are likely to see very low detection rates that could call the value of the program into question. One large program whose participants were mostly women and mostly younger than 65 years reported a detection rate of 0.8%, less than half the rate seen in the Swedish program. If reduced smoking is causing a reduction in AAA prevalence, as appears to be the case, then the US Preventive Services Task Force recommendation to limit AAA screening to older men who have smoked looks prescient. Programs that screen only men who have smoked may grow smaller but are more likely to remain effective.

Disclosures

None.

References


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