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Ultrasonographic Screening for Abdominal Aortic Aneurysm in Iranian Candidates of Coronary Artery Bypass Graft

Background/Objective: To evaluate the prevalence of abdominal aortic aneurysm (AAA) in Iranian candidates of coronary artery bypass graft and its associated factors.

Patients and Methods: 1,647 consecutive candidates of coronary artery bypass graft underwent abdominal aorta sonography. The relation of AAA with gender, age, smoking, dyslipidemia, hypertension, diabetes mellitus, carotid and coronary stenosis was evaluated.

Results: The prevalence of AAA was 3.7% in men and 1% in women. The prevalence was 5.2% in men older than 65 years. The largest diameter f AAA was 51 mm. Gender, age, smoking and carotid stenosis were associated factors of AAA.

Conclusion: The prevalence of AAA is lower in Iran comparing with western studies (p<0.0001). The mean diameter of aneurysm was lesser in our study too (p<0.0001). The associated factors of AAA in our study were similar with previous reports. Screening of AAA is not recommended in Iranian candidates of coronary artery bypass graft.

Keywords: aortic aneurysm, abdominal, coronary artery disease, mass screening, ultrasonography

Introduction

Increased life expectancy over the last 30 years made abdominal aortic aneurysms (AAA) more prevalent.[1,2] Unfortunately, AAA presents none or few symptoms until rupture. After rupture of AAA the mortality is 60%–80%. With elective surgery, the mortality, however will be 3%–7%. Rupture of AAA accounts for around 1% of the deaths in the western world.[3]

Considering these facts, many researchers studied AAA over nearly the past 40 years.[4] Patients with coronary artery disease (CAD) have a high prevalence of AAA and vice versa.[5] Therefore, candidates of coronary artery bypass graft (CABG) surgery have been a target group for screening AAA in many western studies.

With regard to the fact that the prevalence of AAA differs in western and eastern communities,[4,6-9] we designed this study to evaluate the prevalence of AAA in Iranian candidates of CABG.

Patients and Methods

In a cross-sectional study ran between July 2005 and August 2006, gray scale abdominal aortic sonography was performed on all candidates of CABG (n=1,647) who referred to Tehran Heart Center (a university referral center). Sonography was done by an expert radiologist who had been practicing abdominal sonography on a daily basis for more than seven years. The subjects were examined in supine position. We did not use any bowel preparations. No instruction on food or fluid intake was given prior to the examination. The examination was carried out with Logiq 5 expert (the horizon release) GE system with linear 10 MHz and curvilinear 3.5 MHz transducers.

The abdominal aorta was first visualized in the longitudinal plane and was examined from diaphragm to the bifurcation. The aorta was then examined in axial plane with scans perpendicular to the longitudinal plane. The maximum external aortic diameter was measured with electronic calipers for each patient. A transverse diameter >25 mm was considered abnormal according to Emerton and Calderwood.[5,10] This study was approved by the ethics committee of the hospital.

Color Doppler sonography of the carotid arteries was performed for all patients at the same time. The Nicolaides criteria were used for definition of grade of carotid stenosis as we did in our previous study.[11]

All patients had a lipid profile (triglyceride, cholesterol, LDL, HDL) preoperatively. Cholesterol and triglyceride levels were measured by enzymatic methods, HDL by direct method and LDL by Friedewald's method. All parameters mentioned were measured using Pars Azmon reagents licensed by German Diagnostics. Diabetes mellitus was defined as blood sugar >120 mg/dL on two separate examinations or a history of use of antihyperglycemic drug. All the laboratory measurements were done within 24 hours of abdominal sonography.

The smoking status of patients was determined too. The patients were classified as active smoker; non-smoker defined as those who never smoked or who had history of smoking which has been quitted more than five years before; and ex-smoker defined as those who were previous smokers but have quitted smoking within last five years. Packs-years were calculated as the number of cigarettes smoked per day multiplied by the duration of smoking (years) divided by 20. The results of coronary angiography including the extent of left main coronary artery stenosis and number of diseased coronary arteries were extracted from hospital records.

Results

From 1,647 consecutive candidates of CABG, 27 (1.6%) were excluded because of gassy bowel which was an obstacle for evaluation of the aorta. There was no other exclusion criteria.

The remaining 1,620 patients included 1,130 men (69.7%) and 490 women (30.3%).

The mean±SD age of patients was 59.9±9.2 (range: 31–84) years for men and 61.9±8.1 (range: 33–80) years for women. AAA was found in 42 (3.7%; CI_{95}: 2.6%–4.8%) men and 5 (1%; CI_{95}:0.1%–1.9%) women ($p<0.003$).

Due to the low prevalence of AAA in women, like many other studies, they were excluded from further analysis.[12]

From 1,130 men, 384 (34%) were older than 65 years; 764 (66%) were 65 or less. Twenty (5.2%) AAA were detected in the older group while the prevalence of aneurysm was 2.9% in younger group ($p=0.057$). The mean±SD diameter of aneurysm was 29±5 with a maximum value of 51 mm.

Twenty (3.2%) of 616 non-smoker candidates for CABG had AAA, while 19 (6.1%) of 313 smokers suffered AAA ($p=0.043$). The duration as well as packs-years of smoking had some effects on the prevalence of AAA; 8.8% of patients with history of smoking for ≥40 years had AAA, while the prevalence of AAA was 3% in smokers <40 years and 2.9% in non-smokers ($p=0.002$).

Between 393 smoker and ex-smoker candidates of CABG, the mean±SD packs-years was 33.2±19.1 in patients with aneurysm while it was 25.7±19.2 in patients without aneurysm ($p=0.035$).

The prevalence of AAA was 4.4% in non-diabetics (38 out of 864 patient), while it was 1.5% in diabetic (4 out of 266) patients ($p=0.029$).

There was no significant difference in prevalence of AAA between hypertensive and normotensive patients in this study.

With regard to the serum triglyceride levels, 3.8% of the patients with AAA had desirable triglyceride level while 1.6% had high triglyceride (≥200 mg/dL) ($p=0.7$).

Accrding to their serum HDL level, patients were categorized to three groups with HDL<1.20, 1.20–1.39, and ≥1.40 mmol/L. The prevalence of AAA was 4%, 3.5%, and 1.9% in the above-mentioned groups, respectively ($p=0.56$).

With regard to carotid Doppler study, we categorized patients to three groups: those with carotid stenosis <50% in each side; those with 50%–69% stenosis in at least one side; and those with ≥70% stenosis in at least one side. The prevalence of AAA was 3.1%, 11.8%, and 21.4% in the above-mentioned groups, respectively (p<0.0001). The prevalence of AAA was 14.6% in patients older than 65 years with carotid stenosis >50%.

Our hospital surgery data bank contains details of cardiac catheterization as number of diseased vessel (one, two or three vessels) and left main coronary artery stenosis of >50%. Six and four-tenth percent of patients with single vessel disease, 3% of those with two-vessel disease and 3.8% of patients with three-vessel disease suffered concurrent AAA (p=0.54). Furthermore, 3.8% of patients with left main coronary artery stenosis of >50% had AAA while it was 3.1% in patients without left main stenosis of >50%.

In a multivariate logistic regression analysis with AAA as dependent variable and age, absence of diabetes mellitus and carotid stenosis (according to the three above-mentioned groups), the likelihood ratio of the model was 23.01 (p<0.001). Both diabetes mellitus (p=0.033) and carotid stenosis (p<0.0001) could enter the model; the odds ratio for lack of diabetes (in comparison to presence of diabetes) were 3.14; the odds ratio for carotid stenosis was 3.05. The odds ratio of age was 1.03 (p=0.069). (Table 1)

Discussion

AAA is the 15th leading cause of death in the United States. It remains asymptomatic for many years and ruptures ultimately in one-third of cases. The mortality is up to 80% after the rupture. Repair of AAA before its rupture harbors a very low operative mortality rate, around 5%.[4]

All the previously-mentioned facts and the fact that the incidence of AAA is increasing have intrigued physicians to screen for AAA. Schilling did the first screening of AAA in 1964 by lateral abdominal radiographs.[13] Nowadays, abdominal sonography is the best method for screening of AAA.[4]

The prevalence of AAA reported differently in numerous studies. The first reason for this variance is perhaps the fact that different definitions of aneurysm were used by different authors. Some authors have respected aorta “aneurismal” when its diameter reaches 30 mm; some studies have considered diameter >25 mm as aneurysm; and Morris has defined aorta “aneurysm” when the diameter of the infrarenal aorta exceeds 30 mm in patients aged 65 years or older, and >25 mm in patients younger than 65.[4-6,10,14,15]

The studied population is another factor influencing the prevalence of AAA. Some studies only included subjects with cardiovascular risk factors while other studies evaluated elderly men.[5,8,9,14,16] Singh studied 6,386 inhabitants of the Tromso Norway aged 25–84 years without considering risk factors.[3] At least one study proved that ethnicity affects AAA (northern European vs Mediterranean).[1]

Clear gender difference exists in the prevalence of AAA in literature.[3,9,12,17] The rate in men is 4–6 times higher than in women. In our study, the ratio was 3.7—very similar to other studies. The gender difference is related to the inhibited production of matrix metalloproteinase 9 (MMP-9) by macrophages mediated by circulating estrogen in women.[17]

Table 1. Logistic Regression Model Parameters for AAA

Variable	Odds Ratio	95% CI of Odds Ratio	Model* Coefficient(β)	95% CI of Model Coefficient(β)	P-Value
Age	1.03	0.99–1.07	0.03	-0.001–0.69	0.069
Carotid Stenosis	3.05	1.69–5.50	1.11	0.52–1.70	<0.0001
Absence of Diabetes	3.14	1.09–9.01	1.14	0.08–2.19	0.033

CI: Confidence Interval
* The model constant was -7.59 (95% CI= -10.00 to -5.17, p<0.0001)

Table 2. Prevalence of AAA in men aged >50 years

Study	Current smoker (n)	% of AAA in smokers	Never smoked (n)	% of AAA in never smoked
ADAM	22639	6.3	30033	1.6
THC	313	6.1	616	3.2

ADAM: Aneurysm Detection and Management study screening program[4]
THC: Tehran Heart Center

The age of 65 years was proposed suitable for screening of aneurysm since future death from aneurismal rupture is rare after a negative screening at this age.[4] In our study, the prevalence of AAA is 1.8 times more in patients older than 65 years. AAA occurs in 1 of 20 older men in European and North American countries. In Valdes study the prevalence of aneurysm was 7.6% in elder men with cardiovascular risk factors like smoking, hypertension, and occlusive arterial disease in Chile. The mean diameter of aneurysm was 4.1 cm in that study.[9] This rate was 15.3% in patients in the waiting list for CABG in the UK.[5] The prevalence of 5.2% in our study is lower than that in cardiac patients of the European studies ($p<0.0001$).[5] The mean diameter of aneurysm was lower in our study than that of Valdez.[9]

A population-based study in eastern Australia confirmed that the risk of AAA is higher in men with northern European origin than in those with Mediterranean origin.[1] The lower rate of AAA in our study is compatible with the lower risk in Mediterranean region.

Not only the prevalence of AAA is lower in our study but also its mean diameter is lower than similar studies ($p<0.0001$).[9] There is no indication for AAA surgery or interventional repair until its diameter reaches 55 mm. The diameter of the largest aneurysm in our study was 51 mm which did not need any interventions.

Smoking increases the risk of AAA by three to five times in western population. In our study, smoking was a risk factor for aneurysm but with less influence (Table 2). Duration of smoking (as years) and pack per year are two measurable parameters in evaluation of smoking habit. In our study, both the duration and packs-years of smoking had effects on the prevalence of AAA. In Singh study, smoking had strong association with the risk of developing aneurysm, but the duration of smoking, not the number of cigarettes smoked per year, had association with the risk of AAA. In our study, comparing the non-smokers with smokers smoking >40 years, the multivariate-adjusted odds ratio for AAA increased from 1.4 to 8.0 ($CI_{95\%}$: 5.0–12.6).[3]

Absence of diabetes mellitus was a risk factor for AAA in our study as in other studies.[4]

In our study there was no correlation between the serum triglyceride and HDL levels with the prevalence of AAA. In Singh study, there was a strong correlation between serum HDL level and the prevalence of AAA. However, there was no correlation with triglyceride level.[3] The patients suffering dyslipidemia in our study have been taking medication for it, because they were referred from cardiology clinic, hence, the results of serum lipids were not reflecting the real status of patient lipid status.

Logistic regression analysis indicated that absence of diabetes and higher percentages of carotid stenosis were independently associated with increase risk of development of AAA. The age was borderlinely omitted from the model ($p=0.069$). The narrow confidence interval of the age in the model with such a p-value attributed to the great sample size studied.

There was strong correlation between occurrence of AAA and atherosclerosis of carotid arteries, while there was no correlation between AAA and coronary atherosclerosis—either number of diseased vessel or degree of stenosis of the left main coronary artery. Both abdominal aorta and carotid arteries are categorized as large vessels, while coronary arteries are medium-sized vessels. So it is not surprising if the aneurysm of aorta resembles atherosclerosis of carotid arteries. To the best of our knowledge, there is no large study correlating the prevalence of AAA with the extent of carotid stenosis.

To the best of our knowledge, there is only one article evaluating the prevalence of AAA in Iranian population.[18] In that study, Motevalli found two AAA in 241 men candidates of coronary angiography. There is a main reason for the lower prevalence of AAA in Motevalli's study comparing to ours; in that study, the studied population were candidates of coronary angiography, 39 of whom had normal coronary angiography at all. All the remained 202 patients were not candidates for CABG for sure.[18]

Our study had some limitations. We excluded 27 patients from the study, because of gassy bowel. Although it is a trivial (1.6%) number of patients, it could be reduced by re-evaluating them after bowel preparation. Another limitation in our study was the fact that we did not evaluate some other known risk factors such as peripheral vascular disease or family history of AAA. Doppler sonography of iliac and femoral arteries was used for evaluation of peripheral arterial disease in patients with AAA in other studies. We ignored it due to extra-cost of lower limb Doppler study. Another limitation in our study was sampling of the patients. Our hospital is a referral center, thus, the result may not be representative of all Iranian population. Another study on AAA is undergoing by Tehran University of Medical Science, Iran University of Medical Science and Beheshti University of Medical Science which will solve this problem.

As conclusion, the prevalence of AAA is lower in Iranian candidates of CABG. Only the subgroup of older men aged >65 years with at least one carotid stenosis of >50% harbor high prevalence of AAA of 14.6%. Age, gender, smoking, significant carotid stenosis and absence of diabetes are associated risk factors of AAA.

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