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Original Article

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Screening for Prevalence of Abdominal Aortic Aneurysm During Transthoracic Echocardiography in Patient With Significant Coronary Artery Disease

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Abstract

Background: Prevalence of abdominal aortic aneurysm (AAA) has ethnic differences, and coronary artery disease (CAD) shares several risk factors with AAA. Sparse Indian data are available on this. We evaluated the prevalence of AAA during transthoracic echocardiography (TTE) and risk factors of AAA in patients with CAD.

Methods: This was a prospective observational study carried out in the cardiology department at a tertiary care center from January 1, 2017 to November 30, 2017. All patients with CAD/acute coronary syndrome (ACS) were included in the study, and patients with AAA due to other etiology were excluded. Screening for an AAA was performed directly using an echocardiographic 3.5-MHz cardiac probe.

Results: A total of 526 patients were screened; and AAA was present in 25 (4.8%) of CAD patients. Smoking, hypertension and hyperlipidemia were predominant risk factors for AAA in our study, but were not statistically significant because same risk factors were also prevalent in the comparison group. Diabetes, peripheral vascular disease and family history were statistically significant risk factors for AAA in our study. The mean size of AAA was 34 mm.

Conclusions: Presence of AAA is significantly higher among CAD patients. CAD shares several risk factors with AAA. Therefore, opportunistic examination of the abdominal aorta during routine TTE could be an effective way of screening. Diabetes mellitus, peripheral artery disease and family history were the significant associated risk factors of AAA in CAD patients.

Keywords: Coronary artery disease; Abdominal aortic aneurysm; Echocardiography; Screening

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Introduction

Abdominal aortic aneurysm (AAA) remains an important public health issue being the 14th leading cause of death. The overall mortality of ruptured AAA is 80-90% compared with a 30-day postoperative mortality of 3% after elective surgery. Considering the huge impact of timely diagnosis and management, health organizations recommend at least one-time screening for AAA by ultrasonography for individuals aged between 65 and 75 years with history of smoking, thereby reducing AAA-related mortality rates by 50% [1-5].

The relatively low prevalence of AAA in the general population (5.5% of men over 65 years old) implies target-specific high-risk population to have greater cost-effective benefit ratio.

AAA and coronary heart disease share common risks factors, thus patients with coronary artery disease (CAD) represent a high-risk population in which screening for another vascular bed for atherosclerosis is recommended but is often neglected.

The high prevalence of CAD among patients with AAA is well known fact, with great impact on short-term survival after AAA repair [6]. Indeed, coronary angiography is often required prior to aortic surgery, leading to a concomitant CAD prevalence of 31-90% [7-9]. In contrast, the converse relationship, which is the prevalence of AAA among patients with CAD, has been explored only in few recent cohorts or in subgroups of patients. The possibility that AAA might be more prevalent in CAD patients, as compared with the general population, has been suggested by these studies. However, data are limited, incomplete and often with conflicting results.

Ultrasound scanning using a 3.5-MHz probe is a valid first-line method of screening. It is fast and safe with a sensitivity and specificity close to 100% with a low intra- and inter-observer variability. Previous studies have suggested that the 2.5-MHz probe used during routine transthoracic echocardiography (TTE) may be adequate for the detection of AAA with a sensitivity ranging from 91% to 94% using quick-screen programs and the subcostal view [10, 11]. It requires an additional time of only 4 ± 1 min in addition to the standard TTE duration [12]. As most patients with significant CAD undergo a TTE when hospitalized in the cardiology department, we aimed to evaluate the feasibility of AAA screening by the cardiologist at bedside

during TTE via echocardiography machine and to evaluate the prevalence of AAA in this population.

Materials and Methods

This was a cross-sectional observational study, conducted in the Cardiology Department of a tertiary care teaching institute where patients were enrolled prospectively over a period of 1 year. All patients with significant CAD/myocardial infarction (MI) were included in study, while patients with AAA due to other etiologies (inherited, connective tissue disorders) were excluded from the study. Informed written consent was obtained from all participants. The study protocol was approved by the Institutional Ethics Committee (ECM II-Thesis/P22); and the study was conducted in compliance with the ethical standards of the responsible institution on human subjects as well as with the Helsinki Declaration. Patients with CAD were divided into two groups: group A consisted of patients without AAA and group B comprised of those with AAA. Baseline characteristics were collected including age, gender, body mass index, history of atherothrombotic risk factors, e.g., smoking, diabetes mellitus (DM), hypertension, dyslipidemia and history of chronic kidney disease. Details of presentation of CAD (stable angina, unstable angina, non-ST elevation myocardial infarction (NSTEMI), or ST elevation myocardial infarction (STEMI)) were also noted. Patients underwent detailed cardiovascular examination, documentation of peripheral pulses, recording of blood pressure in all four limbs, measurement of ankle brachial index (ABI) and abdominal examination.

CAD was defined as presence of ≥ 50% diameter stenosis in at least one of major epicardial arteries in invasive or computed tomography (CT) coronary angiography. Peripheral arterial disease (PAD) was defined as presence of symptoms of intermittent claudication, reduced ABI or significant peripheral arterial stenosis documented by ultrasound, CT or invasive angiogram. All patients underwent echocardiography, using GE Vivid 7 Pro System. The abdominal aorta was viewed by same echo machine using 3S transducer probe (1.5 - 3.6 MHz) in longitudinal view for any segmental dilatation. The diameter of aorta at suprarenal and infrarenal segments was taken and largest diameter in any segment was noted. AAA was defined as abdominal aorta diameter greater than 30 mm.

Statistical tools

Categorical variables were presented in number and percentage (%), and continuous variables were presented as mean ± standard deviation (SD). Quantitative variables were compared using unpaired *t*-test between two groups and analysis of variance (ANOVA) between three groups. Qualitative variables were compared using Chi-square test/Fisher's exact test as appropriate. A P value of < 0.05 was taken to be statistically significant. The data analysis was done using Statistical Package for Social Sciences (SPSS) version 21.0.

Results

A total of 526 patients with CAD were included in our study. Out of these, 25 patients were found to have significant AAA (> 3 cm). The study population was divided into two groups as follows, group A without (AAA) and group B with AAA. The AAA prevalence in our study was 4.8% (Table 1). Mean abdominal aortic diameter in aneurysm group was 34.4 ± 2.4 mm. Mean age of study population was 63 years. Study population was distributed predominantly between 61 and 75 years of age (45.1%), elderly defined as age (> 75 years) being 15%. The distribution of AAA among age group was as follows: 30 - 45 years: 2 (8%), 45 - 60 years: 5 (20%), 61 - 75 years: 13 (52%) and > 75 years: 5 (20%). Maximum prevalence of AAA was noted in those above 60 years of age (72%). There was no statistically significant association of different age groups with aortic root and abdominal aortic size. As far as gender difference is concerned in our study, a total of 348 (66.2%) male patients and 178 (33%) female patients were studied. The prevalence of AAA was found to be numerically higher in the male patients (n = 15, 60%) when compared to the female patients (n=10, 40%); but there was no statistically significant difference between the two groups (P value = 0.505). Also, there was no statistically significant association of gender with AAA size. Regarding analysis of associated factors with AAA in our study, we found that in the patients with AAA, 22 (88%) were hypertensive, though there was no statistically significant association of hypertension with AAA (P value = 0.505). There was no statistically significant association of hypertension with aortic root and abdominal aortic size (Table 2). Out of the 25 patients with AAA, 20 (80%) of them were smokers and only five (20%) were non-smokers; but there was no statistically significant association of smoking with AAA (P value = 0.216) in our study. We also found that there was no statistically significant association of smoking with aortic root and abdominal aortic size. Among the patients with AAA, five patients (20%) had a positive family history of AAA whereas 20 of them (80%) did not have any such history. There was a statistically significant association of family history with AAA (P value = 0.001). Hyperlipidemia was found in 10 patients (40%) with AAA when compared to 15 patients (60%) who did not have hyperlipidemia. There was no statistically significant association of hyperlipidemia with AAA (P value = 0.865). Out of the 25 patients with AAA, PAD was noted in 14 patients (56%), while it was absent in 11 patients (44%). There was a statistically significant association of PAD with AAA (P value = 0.001). DM was noted in 21 patients (84%) with AAA when compared to four patients (16%) who did not have diabetes. There was a statistically significant association of diabetes with AAA (P value = 0.019), but there was no statistically significant association of diabetes with aortic root and abdominal aortic size. The significant association of AAA with family history, PAD and diabetes was also evident on doing logistic regression analysis (Table 2). We also studied the prevalence of AAA in different presentations of CAD. We found stable angina in one patient (4%, P value = 0.865); unstable angina in six patients (24%, P value = 0.697); NSTEMI in 11 patients (44%, P value = 0.552); and STEMI in 12 patients (48%, P

Table 1. Clinical Characteristics and Abdominal Aorta (AA) Size of the Study Population

	Total, n	Without AAA, n (%)	With AAA, n (%)	P value
Patients	526	501 (95.2)	25 (4.8)	
Male	348 (66.2)	333 (66.5)	15 (60)	0.505
Female	178 (33.8)	168 (33.5)	10 (40)	
Age	62.83 ± 12.41 (31 - 92)	62.69 ± 12.4 (31 - 92)	65.72 ± 11.10 (43 - 90)	0.232
Hypertension	453 (86.1)	431 (86)	22 (88.0)	0.781
Hyperlipidemia	219 (41.6)	209 (41.7)	10 (40.0)	0.865
Diabetes mellitus	325 (61.8)	304 (60.7)	21 (84.0)	0.019
Chronic kidney disease	139 (26.4)	133 (26.5)	6 (24.0)	0.778
Tobacco chewer	384 (73.0)	365 (72.9)	19 (76.0)	0.730
Smoker	362 (68.8)	342 (68.3)	20 (80.0)	0.216
Peripheral arterial disease	146 (27.8)	132 (26.3)	14 (56.0)	0.001
Cerebrovascular accident	62 (11.8)	59 (11.8)	3 (12.0)	0.973
Family history	29 (5.5)	24 (4.8)	5 (20)	0.001
Stable angina	197 (37.5)	196 (39.1)	1 (4.0)	0.865
Unstable angina	110 (20.9)	104 (20.8)	6 (24.0)	0.697
NSTEMI	262 (49.8)	251 (50.1)	11 (44.0)	0.552
STEMI	230 (43.7)	218 (43.5)	12 (48.0)	0.659
Echo aortic root	28.20 ± 1.78	28.28 ± 1.69	26.64 ± 2.51	< 0.001
AA size (suprarenal)	26.36 ± 2.34	26.05 ± 1.83	32.96 ± 1.65	< 0.001
AA size (infrarenal)	26.52 ± 2.66	26.13 ± 1.98	34.40 ± 2.38	< 0.001
LVID	46.13 ± 17.08	46.16 ± 17.50	45.44 ± 2.00	0.837
LVEF	52.34 ± 8.31	52.44 ± 8.37	50.24 ± 7.01	0.197

AAA: abdominal aortic aneurysm; NSTEMI: non-ST elevation myocardial infarction; STEMI: ST elevation myocardial infarction; LVID: left ventricular internal diameter; LVEF: left ventricular ejection fraction.

value = 0.659). A statistical significant association between type of presentation of CAD and AAA was not evident.

Discussion

Traditionally AAA and CAD have been considered as same disease involving two different vascular beds with atherosclerosis being a common etiopathogenetic factor [13]. However, not all patients with CAD have aortic disease and AAA may have etiologies other than atherosclerosis. This study is a novel attempt to analyze risk factors profile of AAA as compared to that in CAD patients from India.

Among several studies published in the western population, the incidence/prevalence of AAA among CAD patients ranged between 0.48% and 18.2%, and eight studies reported

Table 2. Univariate and Multivariate Analysis of Risk Factors of AAA Using Logistic Regression Analysis

Parameters	Univariate (unadjusted OR)			Multivariate (adjusted OR)		
	OR	95% CI	P value	OR	95% CI	P value
Hypertension	0.840	0.245 - 2.879	0.781	-	-	-
Smokers	0.538	0.198 - 1.459	0.223	-	-	-
Tobacco	0.848	0.331 - 2.167	0.730	-	-	-
Family history	0.201	0.070 - 0.582	0.003*	0.160	0.051 - 0.501	0.002*
Hyperlipidemia	1.074	0.473 - 2.437	0.865	-	-	-
Peripheral artery disease	0.281	0.124 - 0.635	0.002*	0.264	0.113 - 0.617	0.002*
Diabetics	0.294	0.099 - 0.864	0.027*	0.326	0.109 - 0.976	0.045*

*P < 0.05. OR: odds ratio; CI: confidence interval.

prevalence above 9% [14-25]. On the contrary, the prevalence in general population was 3.8% (1990s), 3.3% (2000s) and 4.2% (2010), respectively over three decades [26]. In the Italian (RoCAV) study, the prevalence in general population was 1.9% while in targeted high-risk groups it increased to 4% [27]. Thus it is evident that AAA prevalence is usually higher in CAD and other high-risk patients. Our study supports these finding with the prevalence of AAA at 4.8% despite a small sample size.

The mean age in our study was 63 years. Maximum prevalence of AAA was noted in the 61 - 75 years age group (52%), and AAA involved 72% of patients above 60 years of age. Other studies have also revealed increased prevalence (almost twice) of AAA in individuals > 65 years as compared to general population [18]. There was no statistically significant association of different age groups with aortic root and abdominal aortic size. The overall study population was predominantly male (66.2%). The prevalence of AAA was found to be higher in the males like other studies but the gender difference was not found to be significant. Most studies including both male and female patients showed a trend towards a higher male prevalence among AAA cases or detected AAA in male patients exclusively, in accordance with results of population-based studies. Only one study by Shirani et al showed a statistically significant association between male sex and AAA prevalence [22]. Analysis of associated risk factors revealed that most of the patients with AAA (88%) were hypertensive, though there was no statistically significant association of hypertension with AAA. Also aortic root and abdominal aortic size did not show any significant association with hypertension. Although hypertension has been considered a potential risk factor for AAA, findings from prospective cohort studies are not entirely consistent [28]. Several other studies in CAD patients also failed to show any significant association of hypertension with AAA [15, 16, 18, 23-25]. However, a meta-analysis of 21cohort studies showed a 66% increased risk of developing AAA in hypertensive patients [28]. Thus hypertension, being a common contributor to CAD as well as AAA, failed as significant risk factor for AAA in select population of CAD patients.

Cigarette smoking has been strongly linked with AAA and the association has been shown to be stronger than CAD in some studies [28]. Not only smoking is linked to the occurrence of AAA but also it correlates with progression and rupture [29]. Though, out of the 25 patients with AAA in present study 20 were smokers, there was no statistically significant association of smoking with AAA in our study. This could be explained by the fact that population selected in our study without AAA also had high prevalence (68.3%) of smokers.

Family history was positive in 20% of patients with AAA and this association was statistically significant. This was higher than previous studies like the Leige AAA family study, where it was seen in 10% [30, 31]. But additional ultrasonographic screening of relatives led to a hitherto diagnosed AAA in 13% cases and the prevalence was more in brothers (25%). Similarly, in the DANISH VIVA study, positive family history was seen in 7% of cases with AAA. Racial and methodological differences could have accounted for these variegated results. The importance of family history demonstrated in the Leige study, where the likelihood of AAA rupture was higher in familial cases *vis-a-vis* sporadic cases (three-fold higher). However, unlike the Leige study, we did not have follow-up data.

Dyslipidemia is a known risk factor for CAD, however its association with AAA was not significantly associated (P = 0.865). Most CAD patients are on lipid modifying therapy which includes statins, fibrates and cholestyramine. In such populations, some studies have evaluated the association between serum lipoproteins levels or history of dyslipidemia with prevalence of AAA [16, 22, 23, 32]. None were able to find a statistically significant association. Moreover, in view of the current guidelines on atherosclerosis treatment, a study on association between basal lipid profile and AAA prevalence in CAD patients not taking lipid modifying therapy is not possible. Therefore, we cannot draw any conclusions in such a selective population.

Out of the 25 patients with AAA, peripheral vascular disease was noted in 14 patients (56%) and there was a statistically significant association of peripheral vascular disease with AAA (P value = 0.001). Atherosclerosis is a systemic inflammatory vascular disorder which involves multiple arterial beds, so evaluation of AAA prevalence among patients with concomitant PAD and CAD is of great value. Patients with PAD have been found to be at high risk for AAA development with an overall prevalence more than 10%, which is twice that of the general population [33, 34]. A positive correlation of PAD with AAA (odds ratio (OR): 2.5) has been shown in a systematic review of population-based screening studies [34].

DM was noted in 21 patients (84%) with AAA. There was a statistically significant association of diabetes with AAA (P value = 0.019), but there was no statistically significant association of diabetes with aortic root and abdominal aortic size. Only a handfull of studies till date have focused on the association between DM and AAA prevalence. No association was noted in five studies, while in one study a significant association was seen by univariate analysis which was no longer significant by multivariate analysis [14, 15, 20-24]. Shirani et al [22] also reported that frequency of AAA is significantly higher in diabetic patients compared with non-diabetics (3.2% versus 1.4%, P = 0.033).

We also studied AAA in different presentations of CAD (viz. stable angina, unstable angina, STEMI and NSTEMI). There was no significant association among various presentations of CAD and AAA. While a study of Long et al [24] found that AAA was more prevalent in patients with history of acute coronary events and obstructive CAD. Severity of CAD has been differently categorized in the studies mentioned above. In a study done by Durieux et al, there was a strong association between severity of CAD and prevalence of AAA. Here the association between AAA and triple vessel disease was independent of other traditional risk factors including age [35].

The role of other novel markers like serum uric acid and lipoprotein (a) (Lp (a)) has been evaluated by other researchers. Uric acid is a marker of oxidative stress, and a study done by Ali et al [36] has shown a direct relationship between uric acid levels and aortic diameter. Lp (a) is also currently considered a circulating marker of AAA. A meta-analysis done by Kotani et al [37] confirmed that circulating levels of Lp (a) were higher in patients with AAA compared to controls.

We also found that TTE is as good as abdominal ultrasound for screening of AAA. The use of TTE for patients with CAD to assess the abdominal aorta in the subcostal view was convenient and easy to perform.

Study limitations

Our study has few limitations, one being a single-center study with small sample size. This might have limited the power of the study to analyze seven different variables. Also, we did not study the correlation of prevalence of AAA with severity of CAD on angiography. We did not evaluate correlation of other emerging markers like serum uric acid and Lp (a) with AAA.

Conclusions

Presence of AAA is significantly higher among CAD patients. Diabetes, PAD and family history were statistically significant risk factors for AAA in our study. Smoking and hypertension though prevalent in AAA patients were not statistically significant predictors. The sizes of AAA were small (mean 34 mm), which could not meet the criteria for referral to surgery. Since our study was conducted in a select population of CAD patients, results cannot be extrapolated to general population.

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Financial Disclosure

None to declare.

Conflict of Interest

The abstract of the study was presented at the 70th Annual Conference of Cardiological Society of India.

Informed Consent

Informed consents were obtained.

Author Contributions

Dr PV and Dr PP conceptualized the research project. Dr AP and Dr MB did the literature review. Dr PP collected the data while Dr RS, Dr SC and Dr GC performed the statistical analysis. Dr PV, Dr PP and Dr MB prepared the first draft. Dr SKD, Dr AS and Dr VSN critically reviewed the manuscript. Dr MB and Dr AP made the manuscript submission and revision.

Data Availability

The data supporting the findings of this study are available from the first author and corresponding author upon reasonable request.

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