ORIGINAL ARTICLE

Prevalence of Peripheral Arterial Disease and Abdominal Aortic Aneurysm among Patients with Acute Coronary Syndrome

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SUMMARY

Introduction: Peripheral arterial disease(PAD) and abdominal aortic aneurysm(AAA) are two important under-diagnosed vascular pathologies. As they share common risk factors with coronary arterial disease, we conducted a study to look at their prevalence among patients with acute coronary syndrome(ACS).

Materials and Methods: Patients with ACS admitted to Queen Elizabeth Hospital, Malaysia, from February 2009 till August 2009 were screened prospectively for PVD and AAA. Patients' data and clinical findings were gathered and analyzed. Measurements of ankle brachial index (ABI) and abdominal aortic diameter were performed by a single assessor. PAD was defined as ABI ≤ 0.9 or diabetic patients with signs and symptoms of PAD and absence of pedal pulses. AAA was defined as abdominal aortic diameter ≥ 3cm.

Results: 102 patients were recruited with mean age of 59.5 years old. Male: female ratio was 6:1. 40.2% of patients had NSTEMI; 45.1%, STEMI and 14.7%, unstable angina. Risk factors profile is as follows: hypertension- 68.6%, smoking- 56.9%, hypercholesterolemia- 52.9%, diabetes mellitus- 35.3% and history of stroke- 5.9%. Median ABI was 1.1 with lowest reading of 0.4. Mean abdominal aortic size was 2.0cm with largest diameter of 3.3cm. PAD was present in 24.5% of patients and AAA in 2.0%. 68.0% of patients with PAD were asymptomatic. Smoking and age more than 60 years were independent predictors for PAD among ACS patients.

Conclusions: PAD is strongly correlated with CAD with old age and smoker as independent predictors. However, association between AAA and ACS could not be established.

INTRODUCTION

Peripheral arterial disease (PAD) and abdominal aortic aneurysm (AAA) are two major under-diagnosed vascular pathologies. Undiagnosed patients with such pathologies may develop fatal and debilitating consequences. Up to 12.6% of patients with intermittent claudication needed amputation in 10 years and ruptured AAAs carry mortality rate of up to 80% [1,2]. The prevalence of PAD and AAA are around 3% to 10% and 4% to 5% respectively in epidemiological studies [3,4]. PAD, AAA and coronary artery disease(CAD) share similar risk factors profile and were believed to also share a common pathogenesis. PAD, including AAA, were classified as CAD risk equivalent [5].

We conducted a prospective observational study at Queen Elizabeth Hospital, Malaysia, with primary objective to determine the prevalence of PAD and AAA among patients with acute coronary syndrome(ACS) and secondary objectives to indentify risk factors of PAD and AAA among patients with ACS and to determine patients' awareness of PAD and AAA.

MATERIALS AND METHODS

All patients with ACS admitted to Queen Elizabeth Hospital from February 2009 till August 2009 were prospectively screened by a single assessor for PAD and AAA with a standard clinical assessment. Relevant data were collected according to a standard questionnaire. Patients who succumbed to the disease before assessment were excluded from the study. Patients who were hypotensive from cardiac dysfunction and warranted inotropic support were assessed when their clinical status had improved with inotropic support tapered off.

ACS was defined as a spectrum of coronary arterial disease, which include unstable angina, non-ST elevation myocardial infarction and ST elevation myocardial infarction. Diagnosis of ACS was based on clinical presentation, presence of electrocardiographic changes and elevation of chemical markers. Patients' age, signs, symptoms and awareness of PAD and AAA, cardiovascular risk factors, lower limb pulses, ankle-brachial index (ABI) and abdominal aortic size were gathered and analyzed. Measurement of ABI was done by using hand-held doppler and sphygmomanometer. Systolic pressure of the ankle (dorsalis pedis and posterior tibial) and brachial artery at both sides were measured. The higher pressure of the ankle of the respective limb was normalized to the higher brachial pressure to form the ABI. Measurement of abdominal aortic diameter was done by using either General Electric Vingmed Ultrasound Vivid i n or Aloka SSD-500 portable ultrasound machines. The largest antero-posterior diameter of the abdominal aorta was measured. PAD was defined as ABI ≤ 0.9 or diabetic patients with signs and

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Table I: Correlation Between Risk Factors and PVD among Patients with ACS

Risk Factors		PVD	P Value
Hypertension	Yes	25.7%	0.67
	No	21.9%	
Smoking	Yes	32.8%	0.03
	No	13.3%	
Hypercholesterolemia	Yes	20.3%	0.30
	No	21.2%	
Diabetes Mellitus	Yes	33.3%	0.13
	No	19.7%	
Age > 60	Yes	37.9%	0.03
	No	16.4%	
CVA	Yes	33.3%	0.61
	No	24.0%	

Table II: Logistic Regression Analysis Predicting PVD among Patients with ACS

Risk Factors	P Value	Odd Ratio (95% Confidence Interval)
Age > 60	0.023	3.09(1.16-8.19)
Smoking	0.023	3.41(1.21-9.8)

symptoms of PAD and absence of pedal pulses. AAA was defined as abdominal aortic diameter ≥ 30 mm. Patients diagnosed with either PAD or AAA were investigated and managed accordingly.

Statistical analysis was performed with Statistical Package for the Social Sciences (SPSS) version15.0. The chi square test was used for associations between categorical variables and logistic regression analysis was used in multivariate analysis. The level of statistical significance was set at $P < 0.05$.

RESULTS

In total, 102 patients were recruited in the study. 85.3% of patients were male and 14.7%, female. The commonest age group was 50-59 years old with mean age of 59.5. Kadazan-Dusun was the commonest race affected at 24% and followed by Bajau (23%) and Chinese (22.6%). The remaining patients were made up of other races and indigenous groups in Sabah. 45.1% of patients had ST elevation myocardial infarction, 40.2% had non-ST elevation myocardial infarction and 14.7% had unstable angina. Risk factors profile is as follows-hypertension (68.6%), smoking (56.9%), hypercholesterolemia (52.9%), diabetes mellitus (35.3%) and cerebral vascular event (5.9%). 21.6% of patients had ABI ≤ 0.9 and 3.9% of patients were diabetics with ABI more than 9 but had absent pedal pulses and symptoms of PAD. Hence, a total of 24.5% of ACS patients had PAD. 25.3% of male and 20.0% of female in the study had PAD. 68.0% of patients with PAD were asymptomatic. Only 12.0% of patients with PAD had intermittent claudication and 16.0% of them had previous history of amputation. Mean abdominal aortic diameter was 19.9mm, with maximum diameter of 33.0cm and minimum diameter of 10.0mm. Mean abdominal aortic size for male was 20.2mm and female, 18.2mm. Only 2% of patients have abdominal aortic diameter ≥ 30mm in our study. Both patients were in the 70-79 age group. Of note, 85.3% of all patients screened had never heard of PAD and AAA.

Risk factors correlation is summarized in Table I. Smoking and age more than 60 years old were found to be significant factors related to PAD in patients with ACS($P = 0.03$). When analyzed in multivariate analysis, both smoking and age more than 60 years old were also found to be independent predictors for PAD in patients with ACS($P= 0.023$, odd ratio 3.09 and 3.41 respectively) (Table II).

DISCUSSION

CAD and PAD share the common pathogenesis of atherosclerosis and therefore, they share similar risk factors profile and these two conditions commonly occur together. Risk factors for PAD are similar to CAD, which include diabetes, smoking, hyperlipidemia, hypertension and the metabolic syndrome [5]. In our study, 24.5% of patients with ACS has PAD. Prevalence of PAD in patients with CAD from different studies are around 10% to 30% [3]. Old age of more 60 years old and smoking were found to be independent predictors for PAD among patients with ACS in our study. This finding correlates with that by the POSCH Group [6]. Higher prevalence of PAD among men was also observed in other studies [3,7]. In our study, 25.3% of male patients has PAD, as opposed to 20.0% in female patients. The Trans-Atlantic Inter-Society Consensus Document On Management of Peripheral Arterial Disease II (TASC II) has concluded that the ratio of symptomatic to asymptomatic patients with PAD was 1:3 to 1:4 [3]. Our study revealed similar ratio of 1:3. Given the high prevalence of PAD among CAD patients with majority of them asymptomatic, PAD should be actively sorted for in patients with CAD by health care provider. Treatment for PAD include controlling modifiable risk factors for PAD, such as, smoking cessation, controlling hypertension, blood sugar, hyperlipidemia and body weight [5]. Statins and cilostazol have been shown to increase walking distance by 42% and 54% respectively [8,9]. Antiplatelet agents, such as aspirin, ticlopidine and clopidrogrel, do not increase claudication distance but their usage decease risk of major vascular events [5]. Invasive interventions, both endovascular or bypasses, are reserved for

patients with life style-limiting claudication or critical limb ischemia.

AAA was thought to be a result of advanced atherosclerosis but more recent evidence suggested that AAA is an inflammatory and degenerative process with the presence of over-expression of Interleukin 6 and 8, infiltration of neutrophils, macrophages and T-helper cells with ample expression of metalloproteinases (MMP) and cysteine proteinases [10]. Johnsen *et al* recently concluded, from a sample size of 6446 patients, that there is no consistent dose-response relationship between atherosclerosis and abdominal aortic diameter [11]. Risk factors of AAA from population-based studies include male gender, age, smoking and a family history of AAA [12]. Diabetes mellitus, interestingly, was found to be protective of AAA. In a recent review by Shantikumar et al on the association between DM and AAA, the prevalence of DM found among patients with AAA ranged from 6% to 14% and prevalence of DM among patients without AAA was between 17% to 36%. It was, therefore, concluded that studies so far suggested a protective role of DM for the development of AAA [13]. Gyclation is associated with both collagen crosslinking which stiffens aortic wall and attenuated activity of MMPs, mural neovascularization, macrophage infiltration, medial elastolysis, which are important components of the pathogenesis of AAA [14,15]. Reported prevalence of AAA in patients with CAD is, somewhat, variable. Data appears to show a significant geographical variation. In studies involving Caucasians, the prevalence of AAA in patients with CAD was quoted to be 3.3-18.2% [16,17]. Shirani *et al* and Poon et al, from an Asian perspective, reported a prevalence of 2.09% and 1.8% respectively [18,19]. Our prevalence of 2.0% correlates with figures from Asian counties. This may reflect the generally lower prevalence of AAA in Asia and also consolidates the finding that AAA shares a different pathogenesis from atherosclerosis. The Multicentre Aneurysm Screening Study (MASS) reported a 50% reduction in rupture risk and aneurysm-related death with ultrasound screening for men between 65 to 74 years of age and hence advocates the establishment of screening programmes [20]. However, due to lack of data on the prevalence of AAA in Asia, the role of AAA screening in Asia is uncertain [19].

85.3% of patients in our study had not heard off both PAD and AAA. This figure is disappointing and reflects poor patient's awareness of cardiovascular diseases. Lack of awareness will likely lead on to delayed presentation of vascular diseases and, hence, the outcome of treatment. Great effort is warranted to educate patients with CAD concerning cardiovascular diseases of non-cardiac vascular beds.

In conclusion, PAD is strongly correlated with CAD with old age and smoker as independent predictors. An association between CAD and AAA could not be established in our study. Health care provider should routinely examine patients with CAD for PAD. Role of screening for AAA in Asian population is unclear. Awareness of both PAD and AAA among patients with CAD are low.

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