INTRODUCTION
Ischemic heart disease (IHD) is the major cause of morbidity and mortality all over the world. It is usually attributable to atherosclerotic obstruction of coronary vessels and clinically presents as a spectrum of signs and symptoms ranging from angina pectoris to acute myocardial infarction (AMI), more aptly termed as acute coronary syndrome. The age adjusted incidence of AMI, the most life threatening form of IHD, is 192/1000 in the male population and 19/1000 in the female population of Pakistan. A number of risk factors are known to predispose patients to IHD. Some of these cannot be modified, for example age, gender and family history. Modifiable risk factors include dyslipidemia, hypertension, smoking, diabetes mellitus, obesity, physical inactivity, alcohol consumption and psychological factors. These conventional risk factors do not account for all cases of atherosclerotic coronary artery disease (CAD) and myocardial infarction (MI) still occurs in individuals having no obvious traditional risk factor. These observations underscore the need to identify additional risk factors for coronary atherosclerosis. The presence of peripheral arterial disease measured non-invasively by ankle brachial index (ABI), a ratio of ankle systolic blood pressure to brachial systolic pressure, is used in clinical practice to assess the patency of the lower extremity arterial system and to screen for the presence of occlusive peripheral arterial disease. Epidemiological and clinical studies have found that low ABI levels are associated with cardiovascular risk factors, coronary and carotid artery disease and predict cardiovascular and overall mortality. The conventional cut off point for an abnormal ABI i.e. 0.9, was developed from studies of patients referred for angiography of lower extremities. The objective of this study was to determine the association between an abnormal ankle brachial index (ABI) and the presence of significant coronary artery disease (CAD) on coronary angiography.
METHODOLOGY

Patients referred for diagnostic cardiac catheterization were enrolled from July to October 2007 at Sheikh Zayed Hospital, Lahore, after informed consent. All patients had ankle and arm blood pressures recorded using a blood pressure cuff and a hand-held-Doppler. The ankle brachial index (ratio of the ankle/highest brachial systolic pressure) was calculated before cardiac catheterization. After resting for 5 minutes in a supine position, brachial artery systolic and diastolic blood pressure was recorded in both arms using a mercury sphygmomanometer. Appropriate sized blood pressure cuffs were applied over each brachial artery. The cuff was rapidly inflated to 20 mmHg above the audible systolic pressure in each arm and then deflated at a rate of 2 mm per heart beat to the lowest even reading. Highest systolic reading was measured in both arms as the pressure at which the first sustained sound was audible. Diastolic pressure was recorded at the disappearance [phase five] of Korotkoff sounds. The higher of the two arms’ pressure was taken as index arm. Two more readings were taken on the same arm and the average was taken as the index systolic blood pressure in the arm.

In all cases, ankle pressure in both ankles was measured by Doppler with 8 MHz probe which is the Gold standard. The cuff was positioned on the ankle proximal to the malleoli. The pulse was located with a Doppler probe and the cuff inflated until the pulse was obliterated; the cuff was deflated and the pressure was recorded at the point when the pulse reappeared. The leg with lower systolic pressures was taken as index leg. Within the index leg, dorsalis pedis artery pressure was taken as index ankle pressure if it was higher than the posterior tibial and vice versa. Two more readings were taken on the same artery and the average was recorded.

ABI (ankle brachial index) was calculated by dividing the average systolic blood pressure of the index ankle artery by the average systolic blood pressure of the index arm. Patients underwent diagnostic coronary angiography. Single vessel CAD was defined as > 50% stenosis by qualitative coronary analysis in a major coronary artery i.e. left anterior descending artery (LAD), left circumflex (LCx) or right coronary artery (RCA). CAD in the diagonal or marginal vessels was also classified as single vessel CAD. Double vessel CAD was defined as > 50% stenosis in two major coronary arteries. Severe triple vessel CAD was defined as the presence of CAD in the left main stem or > 3 vessel CAD. Diabetics were defined as patients having either previously diagnosed diabetes mellitus or having abnormal fasting blood glucose levels (> 110 mg/dl) on three consecutive occasions. Hypertensives were defined as patients having blood pressure > 140/90 mm Hg (JNC VII) and hypercholesterolemic patients were defined as patients having total cholesterol > 200 mg/dl. The results were obtained using SPSS version 12, p-value was calculated by chi-square test and p < .05 was considered as significant.

RESULTS

The baseline risk factors for coronary artery disease were diabetes in 20 (49%), hypertension in 22 (54%), smoking in 12 (29%), hypercholesterolemia in 6 (15%) and positive family history of IHD in 22 (54%).

Out of 41 patients, there were 31 male (76%) and 10 female (24%). Three patients (7.31%) had ABI < 0.9 and all 3 had triple vessel disease. Ten patients (24%) had ABI of 0.91-0.99, 2 (20%) of them had single vessel disease, 2 (20%) had double vessel disease and 6 (60%) had triple vessel disease. Twenty-four patients (58%) had ABI 1.00-1.28, 8 (33%) of them had single vessel disease, 3 (12%) had double vessel disease and 13 (55%) had triple vessel disease, while 4 patients had normal coronaries.

In the studied population, a total of 22 patients were found to have triple vessel disease and only 3 (13.6%) of those 22 patients had ABI < 0.9 which was statistically not significant (p=0.07). Direct inverse relationship was not established between significant CAD and ABI as only 3 patients out of 22 patients with triple vessel disease had ABI < 0.9. However, an approximately log linear relationship was noted between CAD risk and ABI which means that not only the average CAD risk increased exponentially at values < 1.0 but also that the CAD risk continued to decline as ABI values increased above 1.0 (Figure 1).

DISCUSSION

Ankle brachial index (ABI) is a good predictor of peripheral artery disease, stroke and cardiovascular events in the middle aged and older population. ABI was used in these patients to assess its relationship with
the presence of significant coronary artery disease as an abdominal AB was hypothesized to be associated with significant coronary artery disease. In the studied population, a direct inverse relationship was not established between ABI and coronary artery disease. The observed difference may be due to the fact that very strong exclusion criteria were used and no patient in the studied population had documented peripheral artery disease. Western studies have shown that the African American populations have a substantially higher prevalence of peripheral artery disease (PAD) and borderline ABI than non African American populations. Other possible reasons could be a smaller sample size and short study duration. Moreover, there were marked proportions who lacked important risk factors such as smoking, hypertension and diabetes.

However, an approximately log linear association was noted between ABI and CAD risk. This means that not only the average CAD risk increased exponentially at values < 1.0 but that CAD risk continued to decline as ABI values increased above 1.0. In this study, all 3 patients who had ABI < 0.9 had triple vessel disease. Out of the 10 patients with ABI, 0.91-0.99 6 (60%) had triple vessel disease. Out of the 24 patients with ABI 1.00-1.29, 13 (55%) had triple vessel disease. This again confirms the log-linear association between an abdominal ABI and CAD risk.

Data from ARIC (Atherosclerosis Risk In Communities) and other studies suggest that the average risk of future coronary heart disease (CHD) events increases with decreasing ABI as a continuous but not linear function. Similar results have been reported for exertional leg pain, for carotid intima media thickness and coronary artery calcium. The choice of relevant ABI cut off at which risk factor modification therapy should be instituted to reduce further CAD risk should be based on absolute rather than relative risk of future CAD events. This requires further long-scale studies with better controlled risk factors and a larger sample size to attain adequate study power.

This study established a log-linear relationship between ABI and CAD risk as it showed that not only the average CAD risk increased exponentially at values < 1.0 but that the CAD risk continued to decline as ABI values increased above 1.0. It is not clear, however, how much additional information ABI can provide as a non-invasive screening tool above and beyond that provided by traditional risk factors at cut-off values that can guide clinical practice.

CONCLUSION

This study was not able to establish a direct association between ABI and significant CAD as only 3 patients out of 22 with triple vessel disease had an ABI < 0.9. However, an approximately log-linear relationship was noted between ABI and CAD risk which means that not only the average CAD risk increased exponentially at values < 1.0 but also that the CAD risk continued to decline as ABI values increased above 1.0.

REFERENCES


