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Original Research Article

Relation between central aortic pulse pressure and coronary artery disease: a coronary angiographic study in a tertiary care center

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ABSTRACT

Background: Aortic pulse pressure is a significant marker of cardiovascular morbidity independently of mean blood pressure and pulse pressure of 60 mm Hg should be considered as the threshold at risk both in normotensives and hypertensives. Coronary perfusion is dependent on diastolic blood pressure and patients with CAD may be susceptible to the adverse effects of low diastolic blood pressure. This study conducted to examine the relation between central aortic pulse pressure and the prevalence and extent of CAD.
Methods: A cross sectional, hospital-based study conducted in Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bengaluru, among patients undergoing diagnostic coronary angiography.
Results: Risk factors like Diabetes, Hypertension, Dyslipidemia and smoking rates were significantly higher in patients with PP of >60 mmHg ($p<0.01$). In the first group, the ratio of having normal coronaries is higher 61.9% vs 38% and diseased coronaries was lower when compared to the other group 38% vs 98%. In patients with aortic pulse pressure >60 mmHg, 4 patients had left main coronary artery (LMCA) disease, 20 patients had single vessel disease, 11 patients had two vessel disease and 20 patients had triple vessel disease.
Conclusions: In this study it was demonstrated aortic pulse pressure of more than 60 mm Hg is associated with significant CAD.

Keywords: Aortic pulse pressure, Coronary artery disease, Coronary perfusion, Hypertension

INTRODUCTION

Aortic pulse pressure is a significant marker of cardiovascular morbidity independently of mean blood pressure and pulse pressure of 60 mm Hg should be considered as the threshold at risk both in normotensives and hypertensives.[1-3]

Although elevated systolic blood pressure, rather than diastolic blood pressure has been co-related more strongly with an increased risk of cardiovascular events, patients with Coronary Artery Disease (CAD) tend to have high systolic blood pressure and low diastolic blood pressure secondary to increased arterial stiffness.[4]

Coronary perfusion is dependent on diastolic blood pressure and patients with CAD may be susceptible to the adverse effects of low diastolic blood pressure. There is increasing evidence of a link between stiffness of the conduit vessels and cardiovascular morbidity. Measures of aortic stiffness have been demonstrated to be associated with left ventricular hypertrophy myocardial infarction and stroke in normotensive and hypertensive populations.[5-7]

Finally, increased levels of pulse pressure have been implicated in the development and progression of large vessel atherosclerosis and small vessel disease.[8-10]

The aim of this study was to examine the relation between central aortic pulse pressure and the prevalence and extent of CAD.

Objectives of this study is to find out the relation between central aortic pulse pressure and the prevalence and extent of coronary artery disease in pts undergoing diagnostic coronary angiography and the risk factors associated with increased aortic pulse pressure and extent of CAD.

METHODS

A Cross sectional, hospital-based study conducted in Sri Jayadeva Institute of Cardiovascular Sciences and Research, Bengaluru. Study Population selected for the study were Patients undergoing diagnostic coronary angiography. 100 consecutively admitted patients for coronary angiography were included in this study.

Patients with h/o Effort angina, Unstable angina, TMT Positive and Post MI patients were included into study. Patients with significant aortic or mitral valvular heart disease and Acute MI patients were excluded from the study.

Central aortic blood pressure was recorded using a low - compliance fluid filled catheter positioned in the ascending aorta. Conventional coronary angiography was performed in all the patients using the standard protocol. Brachial blood pressure values were determined in the recumbent position the morning before cardiac catheterization. Descriptive statistics and unpaired t test was used to compare the means, p value <0.05 was considered statistically significant.

Criteria applied for selection of study subjects:

- The patients were evaluated in 2 different groups: aortic pulse pressure of <60 mmHg and >60 mmHg.
- They were classified into 3 groups according to the severity of CAD:
  a) Normal Coronaries
  b) Minimal CAD (<50-70% stenosis)
  c) Significant CAD (>50-70% stenosis).

RESULTS

Among the 100 patients studied the mean age of the patients with <60 mmHg pulse pressure was 52.15 years and in patients with >60 mm Hg pulse pressure was 55.17 years. There were 34 male and 8 female patients with <60 mmHg pulse pressure. There were 49 male and 9 female patients with >60 mm Hg pulse pressure (Figure 1).

Risk factors like Diabetes, Hypertension, Dyslipidemia and smoking rates were significantly higher in patients with pulse pressure of >60 mmHg and p<0.01 (Figure 2).

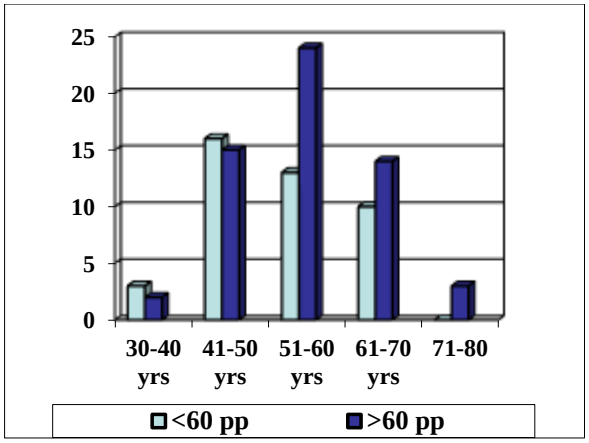

Figure 1: Age wise distribution.

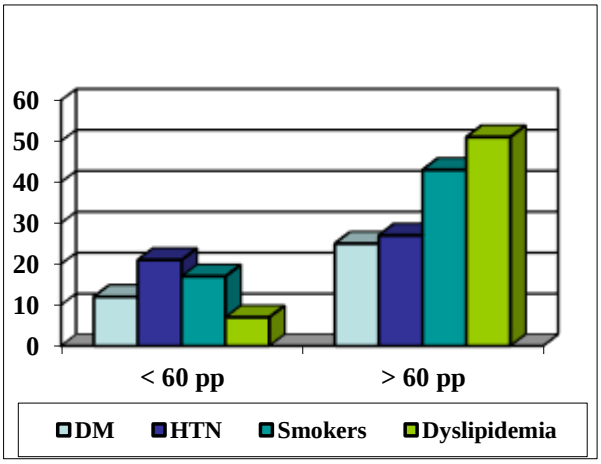

Figure 2: Risk factors among participants.

Based on the clinical presentation there were 14 patients with effort angina, 13 with TMT positive, 4 with unstable angina, 6 with AWMI and 5 with IWMI in first group. There were 10 patients with effort angina, 14 with TMT positive, 8 with unstable angina, 17 with AWMI and 9 with IWMI in the second group (Table 1).

Table 1: Clinical findings among participants.

Clinical findings	<60 PP (42 pts)	>60 PP (58 pts)
Effort Angina	14	10
TMT positive	13	14
Unstable angina	1	6
AWMI	6	17
IWMI	5	9
Post PTCA (TMT + ve)	3	2

In patients with central aortic pressure <60 mmHg, 26 (62%) patients had normal coronaries, 16 (38%) patients had insignificant CAD and none of them had significant CAD. In patients with central aortic pressure >60 mmHg, 1 patient had normal coronaries, 7 patients had insignificant CAD and 50 patients had significant CAD.

In the first group comprising of 42 patients, 21 were hypertensive and 21 were normotensive. In the second group comprising of 58 patients, 27 were hypertensive and 31 were normotensive (Table 2).

Table 2: CAG among participants.

Coronaries	<60 pp	>60 pp	p value
Normal	26	1	<0.001***
In significant CAD	16	7	
Significant CAD	0	50	
Total	42	58	

In patients with aortic pulse pressure <60 mmHg, 10 patients had single vessel disease and 6 patients had two vessel disease. In patients with aortic pulse pressure >60 mmHg, 4 patients had LMCA disease, 20 patients had single vessel disease, 11 patients had two vessel disease and 20 patients had triple vessel disease (Figure 3).

In first group, the ratio of having normal coronaries is higher 61.9% vs 38% and diseased coronaries was lower when compared to the other group 38 % vs 98%.

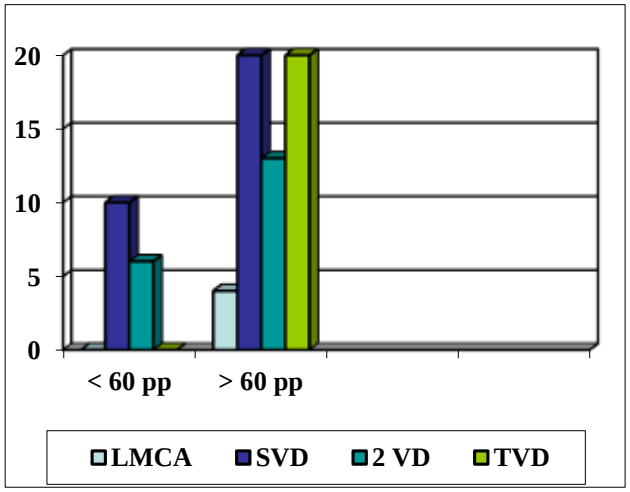

Figure 2: Number of vessels involved among participants.

DISCUSSION

Measurements of central aortic pressure are more closely associated with vascular disease, than are standard brachial pressure readings and are also more predictive of outcomes, suggests an analysis from the Strong Heart Study. Central pulse pressure emerged as stronger than brachial pulse pressure as an independent predictor of CV events.[11]

In the present study, patients with higher central aortic pulse pressure had significant CAD when compared with those patients with lower central aortic pulse pressure. Among the forty-two patients with pulse pressure of <60 mmHg, 26 patients (62%) had normal coronaries and patients with aortic pulse pressure of >60 mmHg: 1 patient had normal coronaries and 50 patients (86%) had significant CAD.

Clinical correlates of increased pulse pressure - Well known correlates of conduit vessel stiffness, including age and history of diabetes or hypertension were found to be related to pulse pressure in the present analysis.[12]

The correlation between pulse pressure and the extent of CAD is consistent with the findings of previous studies.[12,13] A study done by Morihiro Ozaki et al, showed aortic pulse pressure was significantly higher in patients with coronary artery stenosis (p=0.0050).[14] Pulse pressure is also influenced by wave reflections which are also affected by arterial stiffness.[15,16]

In the present study patients with higher central aortic pulse pressure had significant CAD when compared with those patients with lower central aortic pulse pressure. This is in comparison with study done by Morihiro Ozaki et al, where they described that aortic pulse pressure is significantly and independently correlated with angiographically determined coronary artery stenosis.[14]

There are several potential explanations for the association between a higher pulse pressure and cardiac events in patients with coronary artery disease. It may be presumed that the increased pulse pressure was a result of increased conduit vessel stiffness. Conduit vessel stiffening increases the amplitude of the pressure pulse produced by a given flow wave, resulting in higher systolic and lower diastolic pressures for any given mean pressure. Pulse wave velocity is increased in stiffened conduit vessels, and this shortens the time required for the forward pressure wave to travel down the aorta and peripheral conduits to the various reflecting sites and back to the heart. The resulting movement of the reflected wave from diastole into systole has no effect on mean arterial pressure. However, the premature reflected wave further increases the systolic pressure time integral and decreases the diastolic pressure time integral, thereby increasing systolic load while decreasing coronary perfusion pressure. The diastolic perfusion pressure gradient is further compromised if ventricular filling pressures are elevated.[17]

In addition to increasing left ventricular load and diminishing coronary perfusion pressure, conduit vessel stiffness correlates with the presence and severity of atherosclerosis. Because atherosclerosis modifies the physical properties of the conduit vessel wall, increased pulse pressure may simply serve as a marker for advanced or rapidly advancing atherosclerotic disease.[18]

Alternatively, stiffening of the conduit vessels may play a primary role in the development and progression of atherosclerosis. Increased vascular stiffness, independent of the presence of clinically apparent atherosclerosis, is associated with several established risk factors for coronary artery disease, including diabetes, hypertension,

age and a family history of myocardial infarction.[19-21] Arterial stiffening could represent a component of the association between these risk factors and development of atherosclerosis.

Stiffening of the peripheral conduits reduces the transit time of the reflected wave, resulting in progressive overlap between forward and reflected waves in the proximal aorta. This produces a disproportionately large increase in pulse pressure and pulsatile strains in the proximal aorta and thus in the coronary and carotid arteries and may thereby favour development of atherosclerosis in these vascular beds.[22]

In patients with pulse pressure of >60 mmHg there was increased incidence of risk factors like smoking, diabetes, hypertension and dyslipidaemia which is consistent with study done Gokhan Alia et al.[23] A study done by Julio et al showed patients with significant lower diastolic blood pressure of <60 mmHg is associated with increased incidence of CAD.[24]

In patients with pulse pressure of <60 mmHg the ratio of having normal coronaries is higher 61.9% vs 38% ($p<0.001$)and diseased coronaries was lower when compared to the other group 38% vs 98% ($p<0.001$) These results are comparable to a study done by Gokhan et al, who showed in patients with pulse pressure of < 60 mmHg the ratio of having normal CAG was significantly higher (56.4% vs 6.2 $p <0.001$) and also the critical CAD rate was lower than the other group (35.3% vs 84.8% $p<0.001$).[23]

Limitation of this study was to the The dependence of pulse pressure on stroke volume and peak aortic blood flow, both of which may be decreased after extensive infarction, could potentially obscure a relationship between conduit vessel stiffening and mortality after infarction.

Despite this, we found a relationship between pulse pressure and the extent of CAD. This is comparable with a study done by Gary F Mitchell et al where they used Pulse pressure in three different groups of 8-36 mmHg,37-46 mmHg and 47-110 mmHg and showed reduction in event rate in patients with a pulse pressure substantially below the median value of 40 mm Hg.

The dependence of pulse pressure on hemodynamic factors (stroke volume, peak aortic blood flow) other than aortic and peripheral conduit vessel stiffness per se makes this an imperfect indicator of conduit vessel function. Conversely, several important parameters are integrated into this single, easily obtainable measurement. Future studies will need to assess more direct measures of conduit vessel stiffness, such as pulse wave velocity, proximal aortic compliance, characteristic impedance, and a waveform morphology, to determine to what extent increased pulse pressure is a measure of conduit vessel stiffness. The transfer function (alteration in waveform morphology) between central aorta and radial artery has been shown to be remarkably consistent across a wide range of ages. As a result, calibrated non-invasive recordings of radial arterial pressure waveforms using arterial tonometry and a generalized transfer function may allow for accurate determination of central aortic pressure amplitude and morphology and their change under therapy.

CONCLUSION

In the present study it was demonstrated that aortic pulse pressure of more than 60 mm Hg is associated with significant CAD.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethical Board of SJICS

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