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The relation of admission pulse pressure with mortality and severity of coronary artery disease in patients with first STEMI

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A B S T R A C T

Arterial pulse pressure (PP) is a main prognostic factor in many cardiovascular diseases. Data regarding its value in ST segment elevation myocardial infarction is scarce and conflicting, so the aim of this study was to investigate the relation of admission PP with hospital outcome and pattern of coronary artery involvement. Between March 2014 and April 2015, all 103 consecutive patients with first STEMI admitted within 12 hours after the onset of the symptoms were enrolled in this retrospective single center study. Coronary angiography was performed. Data with respect to hospital complications and mortality were collected and analyzed according to admission pulse pressure. P value less than 0.05 was considered statistically significant. Mean PP was 49.84±14.34 mmHg and no significant difference was seen between men and women. Mean PP was lower in patients who died versus survived (39.13±13.01 mmHg vs. 50.75±4.015 mmHg, P=0.027). In patients with three vessel coronary artery disease, mean PP was higher, but the difference was not statistically significant. PP was significantly lower in the patients with Inferior myocardial Infarction than in anterior myocardial Infarction (39±7.41 mmHg vs. 49.11±13.65 mmHg, P=0.03). With respect to hospital complications, apart from cardiogenic shock, which was significantly higher in the low PP group (PP≤ 30) than in the high PP group (PP>60), there was no significant difference between groups in other complications. This study showed a negative relation of admission PP with mortality and crdiogenic shock in patients with first STEMI. The relation of admission PP with multi vessel coronary involvement was not statistically significant.

Introduction

Arterial pulse pressure (PP) is the difference between systolic and diastolic blood pressure. PP reflects pulsatile components of blood pressure and is related to stiffness of aorta and large arteries. PP is a predictor of cardiovascular mortality in general population (1-2) and also, a main prognostic factor in a spectrum of cardiovascular disease (3-4). Both high and low PP have been associated with worse outcome in

many cardiovascular diseases (5-6). Data regarding its implication on ST segment elevation myocardial infarction (STEMI) is scarce and conflicting, so the aim of this study was to investigate the relation of admission PP and hospital outcome in patients presenting with first STEMI and its relation with pattern of coronary artery involvement.

Materials and Methods

Between March 2014 and April 2015, all consecutive patients presenting with first STEMI admitted within 12 hours after the onset of symptoms were enrolled in this retrospective single center study. STEMI was defined according to third universal definition of myocardial infarction (7). Patients with history of previous surgical or percutaneous revascularization, severe valvualr disease, idiopathic cardiomyopathy, and previous myocardial infarction were excluded. We also excluded patients in whom coronary angiography wasn't performed during index hospitalization. Admission blood pressure measurements were recorded by emergency department physicians using a mercury sphygmomanometer at supine position. Brachial PP was defined as difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP). Mean arterial pressure (MAP) was calculated as 2/3 DBP + 1/3 SBP.

Reperfusion therapy by thrombolysis or primary percutaneous coronary intervention (PPCI) was done according to physician decision in emergency room. Patients were categorized to five groups according to admission pulse pressure: PP≤ 30 mmHg, PP= 31-40, PP=41-50, PP=51-60, PP>60 mmHg. Selective coronary angiography was performed by femoral approach according to standard protocol. Three major coronary arteries (the left anterior descending artery, circumflex artery and right coronary artery) were evaluated for extent of coronary atherosclerosis. Coronary artery disease was defined as >50% stenosis in at least one major coronary arteries. Study protocol was approved by local ethical committee and all patients gave written informed consent.

Statistical analysis

Data were expressed as proportions, medians or mean ± standard deviations (SDs). ANOVA test was used to compare continuous variables and x^2 test was used to compare categorical variables. Linear regression analysis with Pearson's coefficient was used to assess the strength of association between variables. P< 0.05 was considered statistically significant. SPSS version 16 was used for analyses.

Results and Discussion

Between March 2014 and April 2015, a total of 170 patients were eligible. We excluded 67 patients who met exclusion criteria and 103 patients enrolled in final analysis. Seventy nine patients (76.7%) were male and 24 (23.3%) were female. The Mean PP was slightly higher in men than women (50.15 ± 14.52 versus 48.83 ± 13.99, P value=0.98) but the difference was not significant. The mean age of patients was 61.18 ± 14.23 years. Baseline clinical characteristics of patients according to different PP subgroups are shown in table 1. There were no significant difference in mean age, prevalence of cardiovascular risk factors, and admission heart rate between PP subgroups. But greater percentage of subjects with Killip Class II-IV were seen in low PP group (P value<0.001). Eight patients died during index hospitalization. The mean PP in patients who died was lower than patients who survived (39.13 ± 13.01 vs

50.75 ± 4.15 mmHg, P value= 0.027). On the other hand, in-hospital mortality rate was higher in the low PP (PP≤ 30 mmHg, mortality=18.2%) subgroup than the high PP group (PP>60 mmHg, mortality=0.0%), although this difference didn't reach statistical significance (P<0.095). The mean SBP and DBP in the dead patients was lower than alive patients (102.62 ± 31.51 vs 129.44 ± 23.90, P=0.004 for SBP and 63.50 ± 20.61 vs 78.63 ± 14, P=0.007 for DBP respectively). Prior medication use including beta blockers, calcium blockers, angiotensin converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) and other drugs was not different between PP subgroups. The mean left ventricular ejection fraction (LVEF) level in patients was 41.57 ± 8.21%.

Left ventricular systolic performance and mean cardiac troponin were not different between PP subgroups although there was a trend toward higher cardiac biomarkers rising in high PP group. We divided patient according to location of myocardial infarction to three groups: Anterior, inferior and others, then we compared PP in these groups. Mean PP was significantly lower in patients with inferior myocardial infarction than in anterior myocardial infarction (39±7.41 mmHg vs 49.11±13.65 mmHg, P=0.03).

With respect to reperfusion therapy, 44 patients (38 male and 6 female) received thrombolysis. Thirty three patients underwent primary percutaneous intervention and in twenty six patients (25.2%) no reperfusion therapy was performed. Main in-hospital complications were as follows:

One patient had post myocardial infarction angina. Cardiogenic shock developed in 7 patients and pulmonary edema in 4 patients. Moreover, primary-VF/VT was also seen in 6 patients whereas minor hemorrhage was seen in 2 patients. (Table 2). Apart from cardiogenic shock, which was significantly higher in the low PP group than others, there was no significant difference between groups in other complications.

The angiographic findings in patients were as follows: 39 patients had one-vessel coronary artery disease, 25 had two-vessel coronary artery disease and 19 had three-vessel coronary artery disease. One had significant Left main lesion. Nineteen patients had normal or non-obstructive coronary angiogram. Mean PP in patients with three-vessel disease was higher than one-vessel disease group but this difference wasn't significant (52.05 ± 19.78 vs. 48.54 ± 13.12 respectively, P value=0.5). No significant linear relationship was seen between PP and the age (P=0.697), LVEF (=0.209), and maximum troponin level (p=0.825).

The findings of present study revealed that the mean PP in patients with first STEMI who died during hospitalization was significantly lower than patients who survived. Moreover, Patients in low PP group (PP≤30 mmHg) had worse hemodynamic status than other PP subgroups and among in-hospital complications, cardiogenic shock was more common in this group.

PP is a marker of large arterial rigidity and reflects pulsatile component of blood pressure. (8) Increased PP has been associated with incident cardiovascular events including myocardial infarction, heart failure and stroke. (9, 10) But impact of PP on short and long-term outcome of acute cardiac events has been a matter of debate.

Increased as well as decreased PP has been associated with worse clinical outcomes in

acute coronary syndromes. Petrie in 2012 reported that in patients with a recent myocardial infarction and reduced LVEF, low PP was associated with mortality in high Killip class (11). In our study, patients with low PP had worse hemodynamic status and higher Killip class (P<0.001) and had higher in-hospital mortality too, although the difference in mortality didn't reach statistical significance (P<0.095), most probably because of small sample size.

Table.1 Baseline clinical characteristic according to pulse pressure at admission

Pulse pressure group Number (%)	≤30 mmHg N=11 (10.7)	31-40 mmHg N=23 (22.3)	41-50 mmHg N=31 (30.1)	51-60 mmHg N=22(21.3)	> 60 mmHg N=16 (15.5)	P
Mean Age- yr	67.36±14.57	59.22±14.53	58.77± 15.41	62.95±11.67	62.00±14.50	0.441
Male Sex- n (%)	8 (72.7)	17 (73.9)	27 (87.1)	14 (63.6)	13 (81.3)	0.958
Mean SBP(mmHg)	92.91±21.58	114.00±11.19	123.90±14.70	137.91±16.80	162.44±20.77	<0.001
Mean DBP(mmHg)	65.18±19.10	74.83±10.78	75.74± 13.94	81.36±15.76	88.00±14.18	0.001
Mean MAP(mmHg)	78.36±20.86	86.35± 11.39	92.48± 13.80	98.56±15.95	111.25±17.40	<0.001
Mean Heart Rate(bpm)	86.45±31.57	82.43± 16.87	78.35± 20.20	84.68±28.64	77.06±20.83	0.714
HTN (%)	5 (45.5)	7 (30.4)	11 (35.5)	9 (40.9)	10 (62.5)	0.167
DM (%)	2 (18.2)	5 (21.7)	4 (12.9)	4 (18.2)	2 (12.5)	0.584
HLP (%)	1 (9.1)	5 (21.7)	3 (9.7)	8 (36.4)	2 (12.5)	0.452
Smoking (%)	3 (27.3)	9 (39.1)	16 (51.6)	9 (40.9)	5 (31.3)	0.961
Killip class I	7(63.6%)	21(91.3%)	30(96.8%)	19(83.3%)	16(100%)	<0.001
Peak troponin	8.38±6.70	196.05±133.05	207.45±193.55	13.41±13.40	14.55±17.27	0.365
Peak CK-MB	151.00±120. 78	14.23±2.86	14.87±1.63	301.29±236.72	304.07±269.75	0.145
Mean LVEF	40.91 ± 7.35	39.74 ± 9.20	42.32 ± 7.76	40.75 ± 9.50	44.29 ± 6.46	0.570
Mean LVEDD (mm)	46.91 ± 2.63	43.21±7.50	47.19 ± 6.47	41.10 ± 14.30	42.71 ± 5.69	0.130

SBP: Systolic Blood Pressure DBP: Diastolic Blood Pressure MAP: Mean Arterial Pressure

Table.2 In hospital complications of patients with STEMI based on Pulse pressure groups

	Pulse Pressure Group					Total	P
	≤ 30 N=11	31-40 N=23	41-50 N=31	51-60 N=22	>60 N=16		
Post MI Angina	0(0.0%)	0(0.0%)	0(0.0%)	1 (4.5%)	0 (0.0%)	1	0.397
Cardiogenic shock	3(27.3%)	2 (8.7%)	0(0.0%)	2 (9.1%)	0(0.0%)	7	0.048
Pulmonary Edema	0 (0.0%)	0 (0.0%)	0 (0.0%)	4 (18.2%)	0(0.0%)	4	0.086
Mortality	2 (18.2%)	3(13.0%)	1(3.2%)	2 (9.1%)	0(0.0%)	8	0.095
Primary VF	0(0.0%)	3(13.0%)	1 (3.2%)	1 (4.5%)	1 (6.3%)	6	0.791
Ischemic Stroke	0(0.0%)	0(0.0%)	0 (0.0%)	1 (4.5%)	0(0.0%)	1	0.397
Bleeding	0(0.0%)	1 (4.3%)	1 (3.2%)	0 (0.0%)	0(0.0%)	2	0.446

In a study by Avazini et al. in 2006, more than 11000 patients with MI were studied and it was concluded that PP>60 mmHg is associated with increased mortality (5). However, in our study other than increased myocardial necrosis markers, no considerable relation was found with increased mortality. This could be due to lower number of patients and lack of long-term follow up in our study. Unlike Avazini, another study in 2011 by El-Menyar in Kuwait, Qatar, and Arab Emirates showed that PP≤30 mmHg was associated with increased mortality in ACS patients (12). Our results are similar to this study, but location of STEMI wasn't defined in El-Menyar's study. Our study may be the first in literature that showed PP is significantly lower in Inferior MI compared to other locations of MI. Precise mechanism is uncertain but may be related to lower SBP usually seen in these patients due to right ventricular involvement. Regarding coronary artery disease, results of present study showed a trend toward more sever CAD in patients with high PP. Some previous studies have shown similar results (13, 14). High PP may lead to higher pulsatile stress on vessel wall and greater tendency to atherosclerosis and more rapid progression of coronary stenosis. So, more sever CAD is expected in patients with high PP. (8).

Our study has some limitations. Small number of patients has attenuated its power, and long term follow up was not available.

Conclusion

This study showed a negative relation of admission PP with mortality and crdiogenic shock in patients with first STEMI. The relation of admission PP with multi vessel coronary involvement was not statistically significant.

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