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Predictive Value of Central Arterial Pressure Pre-Primary Percutaneous Coronary Intervention in Patients with Acute ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Background: Despite advances in primary percutaneous coronary intervention (PPCI), ST-segment elevation myocardial infarction (STEMI) continues to be a significant cause of morbidity and mortality. Central arterial pressure (CAP) assessed during PPCI has emerged as a possible predictor of major adverse cardiovascular events (MACEs) after hospitalisation. **Objective:** To evaluate the predictive value of CAP measured at the initiation of primary PCI in STEMI patients for in-hospital MACE.

Patients and Methods: This study is a cross-sectional prospective study conducted in the Cath Lab and CCU Unit Cardiology Department of Menoufia University, Mataria Teaching Hospital, Mansoura International Hospital from June 2023 to June 2024. We studied 150 patients with STEMI underwent PPCI, they were divided into 2 groups: Group 1: Non-MACEs group includes (100 patients), and Group 2: MACEs group includes (50 patients).

Results: MACE occurred in 50 patients (33.3%) while there was no occurrence of MACE in 100 patients (66.7%). CAP parameters were strong predictors for MACE. Among them the low-pressure subgroup of CSP (<102 mmHg) had the highest relative risk (RR = 4.37, OR =2.14 and P value < 0.001) followed by the low-pressure subgroup of CMP (< 76 mmHg) with high relative risk (RR= 4.19 , OR= 4.2 and p value < 0.001) followed by the low-pressure subgroup of CDP < 61 mmHg (RR = 3.3 , OR = 1.39 and p value = 0.004) and the low-pressure subgroup of CPP< 29 mmHg was the lowest relative risk (RR = 2.29 , OR = 2.46 and p value = 0.002). **Conclusions:** CAP parameters acquired during PPCI provide easily quick information on risk stratification of STEMI patients, with a good predictive potential for the incidence of MACE. Its predictive value outperforms conventional risk score (TIMI risk score and Grace score).

Keywords: Central Arterial Pressure, STEMI, Primary PCI, MACE.

INTRODUCTION

Myocardial infarction (MI) is a significant contribution to morbidity and mortality associated with cardiovascular diseases. Although mortality rates have declined over the past few years, there remains a persistent risk for the occurrence of MACE [(1)].

The most effective therapy for STEMI is PPCI. Nevertheless, even if the infarct-related artery is successfully reopened, a small percentage of patients still have severe myocardial reperfusion damage [(2)].

The risk of MACE following acute myocardial infarction (AMI) has led to the development of various risk stratification tools and strategies. These tools have facilitated the accurate assessment of risk, contributed to reducing adverse outcomes, and guided management plans and treatment approaches [(3)].

Several risk ratings have been established and validated throughout the course of several clinical research and trials. These ratings have shown helpful in predicting MI outcomes by integrating a variety of clinical factors gathered during the first medical examination of patients presenting with AMI [(4)].

There is emerging data showing that hemodynamic measures taken during PPCI in STEMI patients can give useful insights into predicting outcomes and assessing risk [(5,6)].

The lateral pressure applied to the artery wall at the ascending aorta's root is known as CAP [(7)]. Because of its physical proximity to the heart and coronary arteries, CAP provides a clear indication of the cardiovascular system's hemodynamic state. According to recent research, CAP is a better predictor of the beginning, course, and clinical results of cardiovascular disease than peripheral arterial pressure [(8)].

Hemodynamics in AMI are intrinsically complicated, making it more difficult to estimate CAP and cardiac function from peripheral arterial pressure. With the widespread adoption of emergency coronary interventions, CAP can now be accurately measured during interventional procedures, providing real-time hemodynamic insights [(9)].

Therefore, this study aims to evaluate the predictive value of CAP measured at the initiation of primary PCI in STEMI patients for in-hospital MACE.

PATIENTS AND METHODS

This study is a cross-sectional prospective study conducted in the Cath Lab and CCU Unit, Cardiology Department of Menoufia University, and of Mataria Teaching Hospital, Mansoura International Hospital from June 2023 to June 2024 and included 150 patients presented with STEMI and underwent primary PCI. We excluded patients with malignant tumor, active systemic autoimmune or inflammatory disease, liver and renal failure, aortic dissection and pacemaker or intra-aortic balloon implanted before PCI.

METHODS

All patients in this study underwent the following:

- **Clinical and Risk Assessment**
  A comprehensive medical history was obtained, documenting personal details, cardiovascular risk

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factors (smoking, diabetes, hypertension, obesity, dyslipidaemia, chronic kidney disease), family history of premature CAD, prior CAD or CABG, cerebrovascular events, and current medical therapy. A general examination and cardiac assessment, including Killip classification, were performed. Risk stratification was conducted using the TIMI risk score for STEMI [(10)]. Diagnosis was confirmed with a 12-lead ECG, which also determined infarct location [(11)].

- **Laboratory and Echocardiographic Evaluation**

Routine laboratory tests were performed. Glycated Haemoglobin (HbA1c) levels were also measured. The peak troponin value within 72 hours post-PCI was recorded. Transthoracic two-dimensional echocardiography (TTE) was performed before PCI to assess left ventricular ejection fraction (LVEF) and detect mechanical complications such as acute mitral regurgitation, left ventricular free-wall rupture, ventricular septal rupture, papillary muscle rupture, pseudoaneurysm, and true aneurysm. A detailed TTE assessment was repeated after PCI [(12)].

- **Coronary intervention and CAP measurement**
- **Pre-PCI treatment and coronary angiography**

Patients were given a loading dose of 300 mg of aspirin and either 180 mg of ticagrelor or 600 mg of clopidogrel prior to PCI. The left and right coronary arteries were assessed from at least two to four projections during coronary angiography, which was carried out via the radial or femoral artery. The angiographic results were evaluated by two separate cardiologists to direct additional treatment.

- **CAP measurement**

An angiographic catheter was placed 1-2 cm above the ascending aortic valve through the radial or femoral artery during PCI. Before zeroing at the mid-axillary line, the catheter underwent several flushes with heparinized saline and was linked to a multi-channel physiological recorder via a transducer. Following the establishment of a steady pressure waveform, the average of several observations was used to calculate central systolic pressure (CSP) and central diastolic pressure (CDP). CAP values were computed.

- **Angiographic Thrombus Burden Classification and PCI Decisions**

The TIMI thrombus load scale [(13)], which ranges from Grade 0 (no angiographic evidence of thrombus) to Grade 5 (total thrombotic blockage), was used to measure thrombus load. Grade 1, which was defined by suggestive characteristics like haziness, reduced contrast density, irregular lesion contour, or a smooth convex meniscus in total occlusion, Grade 2, which had thrombus that measured less than half a vessel diameter, Grade 3, which had thrombus that ranged between half and two vessel diameters, and Grade 4, which had thrombus that was greater than two vessel diameters, were all considered intermediate grades. Thrombus aspiration, stent insertion (number and location), and/or balloon dilatation were carried out as clinically required based on angiographic results.

Outcome Measures and Follow-Up

The primary outcome was in-hospital MACE, including cardiac death, AHF, cardiogenic shock, malignant arrhythmias, and recurrent AMI. Secondary outcomes included heart failure (Killip II–III), cardiogenic shock (Killip IV), cardiac death, and ischemic stroke with neurological deficits [(14)]. All patients were monitored throughout hospitalization for MACE development.

Ethical approval:

Menoufia Faculty of Medicine's Ethics Committee accepted this work. After receiving all of the information, all the participants signed their permission. The Helsinki Declaration was followed throughout the course of the investigation.

Statistical analysis

Version 23 of SPSS was used to analyze the data. While categorical variables were displayed as numbers and percentages, continuous variables were represented as mean ±SD for normally distributed data and median and interquartile range (IQR) for non-normally distributed data. The Kolmogorov-Smirnov test was used to determine normalcy. Student's t-test for regularly distributed continuous variables, the Mann-Whitney U test for non-normally distributed data, and the X^2-test for categorical variables were used to compare groups. For continuous variables, multivariate logistic regression was utilized to find independent predictors. The ROC was used to evaluate diagnostic performance. Sensitivity and specificity were computed. Statistical significance was defined as a p-value <0.05.

RESULTS

We studied 150 patients with STEMI underwent PPCI, they were divided into 2 groups: Group 1: Non-MACEs group included (100 patients), and Group 2: MACEs group included (50 patients).

The 2 groups were compared as regard the demographic data, risk factors, clinical examination and invasive hemodynamics.

The studied patients included (128 males and 22 females) their ages ranged from 26.0-80.0 years with a mean age of 53.48 ± 9.99.

There was no statistically significant difference between groups as regards gender and age, as well as the prevalence of the risk variables; DM, HTN, smoking, dyslipidemia or history of IHD (Table 1).

Table (1): Demographic data and risk factors among patients

	Groups				Test of Significance	P-Value
	Non-MACEs group		MACEs group			
	N	%	N	%		
	100	66.7%	50	33.3%		
Age (years)						
Mean± SD	53.05 ± 9.58		54.34 ± 10.81		-0.735	0.485(a)
Min – Max	26 - 74		33 - 80			
Gender						
Male	87 (87.0%)		41 (82.0%)		0.666	0.415(b)
Female	13 (13.0%)		9 (18.0%)			
Diabetes mellitus						
No	82 (82.0%)		34 (68.0%)		3.727	0.054(b)
Yes	18.0 (21.2%)		16 (32.0%)			
Hypertension						
No	81 (81.0%)		36 (72.0%)		1.573	0.210(b)
Yes	19 (19.0%)		14 (28.0%)			
Smokers						
No	31 (31.0%)		12 (24.0%)		0.799	0.371(b)
Yes	69 (69.0%)		38 (76.0%)			
Dyslipidemia						
No	86 (86.0%)		39 (78.0%)		1.536	0.215(b)
Yes	14 (14.0%)		11 (22.0%)			
Ischemic heart disease						
No	88 (88.0%)		41 (82.0%)		0.997	0.318(b)
Yes	12 (12.0%)		9 (18.0%)			

MACEs: major adverse cardiovascular events, **N:** number**, (a):** Independent-Sample T Test, **(b):** Chi-Square Test, **P:** P-value between groups.

The mean SBP, DBP, and pulse pressure were statistically significantly higher in Non-Mace group than in MACE group. The mean heart rate was also statistically significantly lower in group I than in group II. Patients of Non-MACE group had significantly lower incidence of Killip class II, III, and IV and higher incidence of class I than those of MACE group (Table 2).

Table (2): Noninvasive hemodynamics and clinical examination among patients

	Groups				Test of Significance	P-Value
	Non-MACEs group		MACEs group			
	N	%	N	%		
	100	66.7%	50	33.3%		
SBP (mmHg)						
Mean± SD	135.0 ± 19.6		116.86± 47.14		2.199	**0.034*(a)**
Min – Max	90 - 150		60 - 230			
DBP (mmHg)						
Mean± SD	80.5 ± 9.92		68.29 ± 29.85		2.364	**0.023*(a)**
Min – Max	50 - 90		30 - 130			
Pulse pressure (bpm)						
Mean± SD	54.5 ± 9.68		48.57 ± 17.29		2.124	**0.041*(a)**
Min – Max	30 - 70		30 - 100			
Heart rate (beats/min)						
Mean± SD	82.03 ± 12.06		94.44 ± 20.15		-4.01	**<0.001**(a)**
Min – Max	40 - 120		40 - 133			
Killip class						
1	97 (97.0%)		26 (52.0%)		27.802	**<0.001**(b)**
2	3 (3.0%)		16 (32.0%)			
3	0 (0%)		3 (6.0%)			
4	0 (0%)		5 (10.0%)			

(a): Independent-Sample T Test, **(b):** Chi-Square Test, *: significant, **: Highly significant, **P:** P-value between groups

The mean ejection fraction was statistically significantly higher in patients of Non-MACE group than those of MACE group. There was a statistically significant difference between the 2 groups regarding mitral regurgitation and aortic stenosis and diastolic dysfunction and tricuspid regurgitation incidence (Table 3).

Table (3): Echocardiographic data among the studied groups

	Groups				Test of Significance	P-Value
	Non-MACEs group		MACEs group			
	N	%	N	%		
	100	66.7%	50	33.3%		
Ejection fraction						
Mean± SD	50.16 ± 8.44		42.18 ± 11.0		4.919	**<0.001**(a)**
Min – Max	25 - 73		20 - 67			
Mitral regurgitation						
No	28 (28.0%)		4 (8.0%)		29.19	**<0.001**(b)**
Mild	69 (69.0%)		30 (60.0%)			
Moderate	3 (3.0%)		13 (26.0%)			
Severe	0 (0%)		3 (6.0%)			
Tricuspid regurgitation						
No	42 (42.0%)		16 (32.0%)		1.778	0.411(b)
Mild	41 (41.0%)		22 (44.0%)			
Moderate	17 (17.0%)		12 (24.0%)			
Aortic stenosis						
No	94 (94.0%)		41 (82.0%)		5.333	**0.021*(b)**
Mild	6 (6.0%)		9 (18.0%)			
Diastolic dysfunction						
I	89 (89.0%)		34 (68.0%)		10.043	**0.007*(b)**
II	10 (10.0%)		14 (28.0%)			
III	1 (1.0%)		2 (4.0%)			

Grace: Global registry of acute coronary events, **(a):** Independent-Sample T Test, **(b):** Chi-Square Test, **(c):** Mann-Whitney U Test, *: significant, **: Highly significant, **P:** P-value between groups

No significant difference was found between the 2 groups regarding median CK-MB and median troponin and mean hemoglobin level and mean platelet count. However, the mean of TLC count, troponin on the second day, HBA1C, and serum creatinine were statistically significantly lower in patients of non-MACE group than those of MACE group (Table 4).

Table (4): Laboratory data among patients

	Groups				Test of sig.	P-Value
	Non-MACEs group		MACEs group			
	N	%	N	%		
	100	66.7%	50	33.3%		
Creatine kinase-MB (Ck-MB) (U/L)						
Median (IQR)	80 (123.25-39.25)		68 (139 – 32)			
Min – Max	13 - 523		8 - 1289		2471	0.908(c)
Troponin I (ng/mL)						
Median (IQR)	653.5 (2000 – 40)		929 (2000 – 62.5)			
Min – Max	30 - 2000		7 - 2000		2226	0.264(c)
Hemoglobin (g/dL)						
Mean± SD	13.61 ± 1.52		13.86 ± 1.47		-0.982	0.328(a)
Total leukocytic count (TLC) (×10⁹/L)						
Mean± SD	10.8 ± 2.58		14.58 ± 3.51		-3.671	<0.001**(a)
Platelet count (×10⁹/L)						
Mean± SD	267.75 ± 6.92		290.96 ± 72.48		-1.687	0.051(a)
Troponin on 2nd day (ng/mL)						
Min - Max	179 - 2000		254 - 2000		-3.952	<0.001**(a)
HBA1C (%)						
Mean± SD	6.23 ± 1.84		7.16 ± 1.46		-2.357	0.021*(a)
Serum creatinine (mg/dL)						
Mean± SD	1.11 ± 0.276		1.35 ± 0.33		-3.338	<0.001**(a)

(a): Independent-Sample T Test, **(b):** Chi-Square Test, **(c):** Mann-Whitney U Test, *: significant, **: Highly significant, **P:** P-value between groups.

Table (5) shows the mean values of invasive hemodynamics, CSP, CDP, CPP, and CMP, during primary PCI of the studied patients (Table 5).

Table (5): Invasive hemodynamics during 1ry PCI of the studied patients

Variables		Values
Central systolic pressure (CSP)	**Mean ± SD**	124.31 ± 33.0
	Min - Max	56 - 236
Central diastolic pressure (CDP)	**Mean ± SD**	70.53 ± 22.16
	Min - Max	28 - 139
Central pulse pressure (CPP)	**Mean ± SD**	53.43 ± 16.21
	Min - Max	23 - 108
Central mean pressure (CMP)	**Mean ± SD**	88.01 ± 24.96
	Min - Max	40 - 171

Table 6 shows the incidence rate of In-hospital MACE distribution in studied patients. There were notable differences in the rates of in-hospital outcomes, cardiac mortality, cardiogenic shock, acute left HF, and malignant arrhythmia across the studied patients (Table 6).

Table (6): In-hospital MACEs of the studied patients

Variables		Values	P-Value
Heart failure (NYHA class III-IV)	**No**	129 (86.0%)	**<0.001****
	Yes	21 (14.0%)	
Shock	**No**	121 (87.3%)	
	Yes	19 (12.7%)	
Malignant arrhythmia	**No**	137 (91.3%)	
	Yes	13 (8.7%)	
Ischemic stroke	**No**	141 (94.0%)	
	Yes	9 (6.0%)	
Re-infarction	**No**	131 (87.3%)	
	Yes	19 (12.7%)	
Cardiac death	**No**	142 (94.7%)	
	Yes	8 (5.3%)	

**: Highly significant.

CSP, CDP, CPP and CMP were each divided into 4 groups using the quartile method group. n3 was the least group of patients associated with MACEs and used as a control group (Table 7).

Table (7): Sub-groups according to CAP indicators

	Group n1	Group n2	Group n3	Group n4
Central systolic pressure (CSP)	**<102 mmHg**	**102 – 115 mmHg**	**116 – 137 mmHg**	**≥137.5 mmHg**
Central diastolic pressure (CDP)	**<61 mmHg**	**61 – 72 mmHg**	**73 – 84 mmHg**	**≥85 mmHg**
Central pulse pressure (CPP)	**<29 mmHg**	**29 – 40 mmHg**	**41– 54 mmHg**	**≥55 mmHg**
Central mean pressure (CMP)	**<76 mmHg**	**76 – 87 mmHg**	**88– 100 mmHg**	**≥101 mmHg**

Among them, compared to the incidence of in-hospital outcomes in group 3 (n3), the low-pressure subgroup of CSP had the highest relative risk (RR=4.37), followed by the low-pressure subgroup of CMP (RR =4.19) (Table 8).

Table (8): Incidence of outcomes in different sub-groups according to CAP indicators

Variables	Low-pressure	Relative risk (RR)
Central systolic pressure (CSP) (116 - 137 mmHg)	116 mmHg	4.37
Central diastolic pressure (CDP) (73 - 84 mmHg)	73 mmHg	3.30
Central pulse pressure (CPP) (41 - 54 mmHg)	41 mmHg	2.29
Central mean pressure (CMP) (88 – 100 mmHg)	88 mmHg	4.19

The results of binomial multivariate logistic regression revealed that the odds ratio of all the independent variables; CSP, CDP, CPP, and CMP of groups n1 and n4, were statistically significant (Table 9).

Table (9): Multivariate logistic regression analysis

Independent variables	Odds Ratio for n1 (95%) CI	P- value	Odds Ratio for n2 (95%) CI	P- value	Odds Ratio for n4 (95%) CI	P- value
Central systolic pressure (CSP)	**<102 mmHg 2.14 (0.91 – 3.98)**	**<0.001**	**102 – 115 mmHg 1.19 (0.39 – 1.87)**	**0.118**	**≥137.5 mmHg 1.28 (0.48 – 2.21)**	**0.018***
Central diastolic pressure (CDP)	**<61 mmHg 1.39 (0.65 – 2.19)**	**0.004***	**61 – 72 mmHg 1.2 (0.56 – 1.86)**	**0.11**	**≥85 mmHg 1.04 (0.45 – 1.63)**	**0.429**
Central pulse pressure (CPP)	**<29 mmHg 2.46 (1.44 – 3.48)**	**0.002***	**29 – 40 mmHg 1.28 (0.68– 1.91)**	**0.09**	**≥55 mmHg 1.79 (0.97 -2.56)**	**0.044***
Central mean pressure (CMP)	**<76 mmHg 4.20 (1.51 – 6.24)**	**<0.001**	**76 – 87 mmHg 1.73 (0.58 – 2.88)**	**0.189**	**≥101 mmHg 2.01 (1.05 – 2.95)**	**0.043***

*: Significant, **: Highly significant, CI: confidence interval, OR: odds ratio.

The capacity of several CAP variables to forecast hospitalization outcomes is shown in table 10. Regarding CSP at a cut-off value of ≥ 92.5; its sensitivity was 88.46% and its specificity was 70.3%. Regarding CDP at a cut-off value of ≥ 64.5; its sensitivity was 83.3% and its specificity was 54.05%. In terms of CPP at a cut-off value of ≥ 39.5; its sensitivity was 80.8% and its specificity was 89.2%. In terms of CMP at a cut-off value of ≥ 79.5 its sensitivity was 79.5% and the specificity was 54.05%.

Table (10): ROC curves analysis of CSP, CDP, CPP, and CMP to represent the capacity of different CAP indicators to predict outcomes during hospitalisation

Parameters	AUC	P	95% C.I.	Cut off	Sensitivity	Specificity	PPV	NPV
Central systolic pressure (CSP)	0.624	**0.033***	0.485–0.762	≥92.5	88.46%	70.3%	86.25%	74.28%
Central diastolic pressure (CDP)	0.660	**0.006***	0.537–0.783	≥64.5	83.3%	54.05%	79.27%	60.6%
Central pulse pressure (CPP)	0.703	**<0.001****	0.599–0.807	≥39.5	80.8%	89.2%	94.03%	68.75%
Central mean pressure (CMP)	0.690	**<0.001****	0.568–0.812	≥ 79.5	79.5%	54.05%	79.52%	62.5%

AUC: Area under a curve, **p-value:** Probability value, **CI:** Confidence Intervals, NPV**:** Negative predictive value, **PPV:** Positive predictive value, *: significant.

DISCUSSION

There is still a significant risk of MACE in this patient cohort, despite a ten-year drop in STEMI-related death rates [15]. For patients with STEMI, these incidents continue to be a leading cause of death and morbidity [16].

Although the rate of MACE recurrence after STEMI is somewhat unexpected, it might be reduced with the use of suitable risk stratification techniques and strategies that direct various therapeutic methods [17].

The first assessment of patients who present with STEMI has placed a great deal of emphasis on accurately determining their risk in order to make the right choices and increase the absolute benefit of using effective treatment modalities and hospital recovery time. A number of clinical characteristics, such as age, gender, co-morbidities, electrocardiographic criteria, multi-vessel coronary artery disease, post-procedural flow grade, lower LVEF, and greater Killip classification, are associated with MACE in patients with STEMI [18]. This led to the development of a large number of different risk scores with two major scores being the most widely used after many clinical studies and trials applied to them providing accurate evidence of their effectiveness; The thrombolysis In Myocardial Infarction (TIMI) risk score for STEMI and the Global Registry of Acute Coronary Events (GRACE) ACS risk score which incorporate clinical data available on admission to identify patients at highest risk for MACE [5]. Both of these risk ratings were not intended for use during PPCI, although they do include some non-invasive hemodynamic information (such as HR, SBP, and Killip class). A growing body of evidence from several earlier research indicates that CSP, CDP, CMP, and CPP measurements made at the time of PPCI are more predictive of outcomes in patients with STEMI than these risk scores [5].

Therefore, in contrast to the conventional GRACE and TIMI risk scores, the study's goals were to assess and compare the association between CAP, which was evaluated at the start of PCI, and hospital outcomes—including mortality—in patients who were diagnosed with STEMI.

The age of the studied group was 26.0 to 80.0 years with a mean age of 53.48 ± 9.99 years. There were 128 males (85.3%) and 22 females (14.7%) in the studied patients.

Invasive hemodynamic measures such as CSP, CDP, CPP, and CMP were assessed during the PPCI operation using 6F fluid-filled catheters. HR was obtained at the start of the PPCI process as well. Other information gathered included baseline comorbidities (diabetes, hypertension, smoking, dyslipidemia, positive family history) and admission creatinine. The fast physical examination done before to PPCI was used to obtain baseline vital signs (SBP, DBP, PP, HR), as well as Killip classification. Echocardiographic values were acquired via a transthoracic echocardiogram soon before PPCI and 24-48 hours later. Open-access internet tools were used to determine the GRACE and TIMI risk scores.

Patients were assessed and followed up for the occurrence of MACE during their hospital stay. There was no occurrence of MACE in 100 patients (66.67%) while MACE occurred in 50 patients (33.33 %) of the whole study population, including 21 patients had HF (14%), 19 patients had shock (12.7%), there were 13 patients had malignant arrhythmia (8.7%), 9 patients had ischemic stroke (6%), 19 patients had re-infarction (12.7%), and 8 patients had cardiac death (5.3%).

The quartile technique was used to separate CSP, CDP, CPP, and CMP into four groups. In-hospital outcomes such as cardiac mortality, cardiogenic shock, acute left HF, and malignant arrhythmia were substantially different among the four groups ($P < 0.001$).

The study discovered that among the CAPs, systolic CAP that was either lower (less than 102 mmHg) or higher (greater than 137.5 mmHg) was an independent risk factor for MACEs, diastolic CAP lower (less than 61 mmHg), higher (more than 55 mmHg), pulse CAP lower (less than 29 mmHg), and mean CAP higher (more than 101 mmHg) or lower (less than 76 mmHg).

The relationship between systolic CAP and mean CAP and in-hospital outcomes showed a “J”-shaped relationship, diastolic CAP and in-hospital outcomes showed an “L”-shaped relationship, and pulse CAP and in-hospital outcomes showed a “U”-shaped relationship.

Previous studies by **Chirinos *et al.* [16], Danchin *et al.* [19] and Weber *et al.*[20]** stated that The LV afterload and cardiac output are reflected in the CSP. Low CSP results in decreased cardiac output and a higher likelihood of inadequate perfusion of the major organs. Additionally, CSP has a stronger correlation with coronary artery stenosis than peripheral arterial SBP. It is also more accurate in forecasting the return of acute coronary syndrome and cardiac mortality following PCI.

In agreement to our study **Bao *et al.* [3], Huang *et al.* [12], Adamopoulos *et al.* [21] and Ather *et al.* [22]** who found that the significant risk factors for HF and cardiac mortality were reduced SBP and diminished LVEF. Low admission SBP was thought to be closely linked to death in AMI thrombolytic risk scores and other prognostic models, and the link between SBP and MACEs is similarly "J"-shaped with **Morrow *et al.* [23], Chin *et al.* [24] and Stebbins *et al.* [25]**.

According to our research, there was a "J"-shaped correlation between CSP and in-hospital outcomes. The greatest frequency of in-hospital outcomes was observed in the low-CSP group. The low CSP group's OR value was substantially greater than the high CSP group's after multivariate correction (2.14 vs. 1.28). CSP and LVEF had a significant correlation when CSP rose ($r=0.234$, $P<0.003$). Nonetheless, there was no discernible rise in the prevalence of malignant arrhythmias, cardiogenic shock, or cardiac mortality.

This is associated with elevated CSP, which raises blood perfusion in important organs while simultaneously increasing LV afterload. In the **Collins *et al.* [15]** and **Swedberg *et al.*[26]**, the ACEI group saw a considerably greater incidence of hypotension than the placebo group (10.3% vs. 4.8%, 12% vs. 3%), and in several individuals, the hypotension even led to the cessation of ACEI medication..

Based on clinical trial evidence, **Collins *et al.* [15] and Swedberg *et al.*[26]**, particularly in older individuals, ACEIs should not be taken if peripheral SBP is less than 100 mmHg or 30 mmHg below baseline. This aligned with our research, which found a robust and independent correlation between cardiovascular mortality and low CSP in STEMI patients. The analysis's findings also indicated that the probability of in-hospital outcomes rose progressively when CSP was less than 108.1 mmHg. Antihypertensive medication should be considered when CSP is greater than 137.5 mmHg in order to lower the risk of abrupt left HF.

Diastole is when coronary perfusion takes place, and DBP is the primary factor that determines coronary blood flow. **McEvoy *et al.* [27],** as well as **Tsujimoto and Kajio [28],** showed that patients who had low DBP (<60 mmHg, P<0.05) were more likely to experience cardiovascular events and die than those who had DBP between 80 and 89 mmHg. However, in contrast to our research, the aforementioned studies, along with **Denardo *et al.* [29]** were all long-term prognostic studies for peripheral DBP, and they were unable to reliably and directly represent coronary perfusion.

This analysis indicated that only the low CDP group (<61 mmHg) was an independent risk factor for in-hospital outcomes (OR = 1.39, P=0.004) after controlling for age, HR, and other confounding variables. An "L"-shaped link between CDP and in-hospital outcomes was shown in the findings of the limited cubic spline plot analysis, and the probability of in-hospital outcomes increased progressively when CDP was less than 61 mmHg (P value 0.004). In-hospital outcomes and acute left HF did not rise in the low group (62–84 mmHg) as compared to the high CDP group (85 mmHg), and the difference was not statistically significant (P>0.05). Furthermore, of the four groups, the high CDP group had the lowest incidence of malignant arrhythmia, cardiogenic mortality, and cardiogenic shock. This could be due to the fact that significant blood supply imbalance is the primary issue of individuals with STEMI. Raising CDP has the direct potential to ameliorate myocardial ischemia, decrease surgical outcomes, and boost coronary perfusion. Furthermore, our study found that central mean arterial pressure and SBP both had similar predictive capacities for in-hospital outcomes, and the nonlinear curve likewise had a "J"-shaped association. In-hospital complications were shown to be more likely in patients with CMP <76 mmHg and CMP >101 mmHg. Since CMP primarily measures the perfusion of major organs, there are no large randomized controlled trials available. The low-pressure subgroup of CMP had RR = 4.2.

Although several studies such as **Rosenwasser *et al.*[30], Vlachopoulos *et al.*[31] and Wu *et al.*[32]** have demonstrated that the long-term survival rate of patients with acute coronary syndrome is highly correlated with CPP, and that this association is "J" shaped. This was not consistent with our study, as we discovered that there was a "U"-shaped correlation between CPP and in-hospital outcomes, which was in line with the results of **Ndrepepa *et al.* [33].** CPP, on the other hand, was the least effective CAP indicator for predicting in-hospital outcomes. This might be because CPP primarily represents aortic compliance and is associated with the long-term prognosis of cardiovascular illness, while hemodynamic alterations are more directly linked to the short-term prognosis of patients with STEMI.

CONCLUSIONS

CAP parameters acquired during PPCI provide easily quick information on risk stratification of STEMI patients, with a good predictive potential for the incidence of MACE. Its predictive value outperforms conventional risk score (TIMI risk score and Grace score).

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