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ORIGINAL ARTICLE

Pulmonary Artery Stiffness Can Predict Contrast Induced Nephropathy in Acute Myocardial Infarction Patients Undergoing Primary Percutaneous Coronary Angiography

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ABSTRACT

Background: Acute myocardial infarction is a critical medical illness characterized by an elevated mortality rate. The WHO identifies heart attacks as a predominant cause of mortality globally, with a heightened incidence of serious adverse cardiovascular events in STEMI cases receiving primary percutaneous coronary angiography. The purpose of this study was to use an easy and noninvasive parameter as assessing pulmonary artery stiffness as a predictor for developing CIN in acute MI patients undergoing primary PCI.

Methods: This cross-sectional research was performed in the cardiology department of Zagazig University Hospitals commencing in 2024. The present research involved 133 cases with AMI who received primary percutaneous coronary angiography.

Results: A statistically significant difference was found between the two studied groups concerning both after PPCI s. creatinine and estimated GFR. Also, negative correlation between post PPCI serum creatinine, and EF by Simpson method which was highly statistically significant (p=≤0.001).

Conclusions: pulmonary artery stiffness and PSAP may be an independent indicator for the progress of CIN in acute MI cases participate in primary PCI. The simplicity of PSAP measurement using echocardiography makes it a potentially valuable tool in clinical practice for preventing CIN in acute MI patients undergoing primary PCI.

Keywords: Pulmonary Artery Stiffness ;CIN; AMI; Primary Percutaneous Coronary Angiography.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) is a critical medical illness related with an elevated mortality rate. Primary PCI is regarded as the optimal therapeutic modality for STEMI. **[1]**. Consequently, attention has shifted to CIN, a moderately frequent complications following PCI but with a high absolute incidence due to the prevalence of the disease and its treatment**[2]**.CIN accounts for 13% of all acute renal failure (ARF) cases, considering it one of the most prevalent reasons for in-hospital ARF and the third most common overall **[3]**. CIN results in prolonged hospitalizations , increased treatment expenses and in some cases, irreversible end-stage renal failure**[4].** The presence of high SPAP in STEMI cases has been associated with adverse outcomes**[5]**. Elevated SPAP may stem from increased elevated LV filling pressures attributed to LV systolic and diastolic dysfunction**[6]**. Systolic and diastolic dysfunction in STEMI patients with high SPAP may contribute to progress of CIN. The current

research aimed to use an easy and noninvasive parameter as assessing pulmonary artery stiffness as a predictor for developing CIN in acute MI patients undergoing primary PCI.

METHODS

This cross-sectional research was performed at cardiology department, Zagazig university hospitals starting from 2024. This study was conducted on 133 cases with AMI who were treated by primary percutaneous coronary angiography.

Inclusion criteria include cases with AMI candidate for primary PCI.

Exclusion criteria include moderate or severe valve stenosis or regurge, LVEF<50%, AF, other arrhythmias, Renal or hepatic failure.

This protocol was followed for all patients: Complete history-taking: Personal history, complaint & its duration, current history, past medical history, & past surgical history. Physical examinations, Laboratory Investigations: Troponin, CkMB, lipid profile, creatinine level.

We obtained a 12-lead electrocardiogram for all patients upon admission to determine the kind of MI. Blood samples were gathered at the time of admission and throughout the follow-up period. Echocardiography was conducted by experienced cardiologists in ICU immediately following PCI, and LVEF was computed via the modified Simpson's approach. Pulmonary hypertension was distinct as a resting average pulmonary artery pressure of > 25 mmHg, as established by right cardiac catheterization**[7]** .In cases without pulmonary valve stenosis, the right ventricular (RV) pressure was used for SPAP. SPAP by echocardiography was calculated utilizing Bernoulli equation ($4 \times TRVmax^2$) + RAP based on peak tricuspid regurgitation flow rate. The RAP was estimated from the subcostal view based on the respiratory change in the inferior vena cava (inspiratory collapse). In cases with ≥ 50% respiratory collapse, 5 mmHg was added, and in cases with < 50% collapse, 10-15 mmHg was added **[8]**.Right ventricular systolic pressure of < 30 mmHg was determined normal, 31 to 55 mmHg was classified as mildly to moderately raised, and > 55 mmHg was categorized as highly increased. Serum creatinine changes were monitored through daily blood tests. According to creatinine values, Cases were categorized into two groups: those with CIN and those without. CIN was characterized as a 0.5 mg/dL (44 μmol/L) absolute elevation in blood creatinine within 48-72 hours, or a rise of 25% in serum creatinine from baseline following PCI **[9]**. Doppler examination for evaluating Pulmonary arterial stiffness was determined using the formula: pulmonary artery flow (kHz/sec) = maximal frequency shift / acceleration time.

Coronary angiography and in-hospital follow-up

Every PCIs were achieved using the femoral approach by proficient cardiologists. Each procedure was conducted by just invasive cardiologist, employing a nonionic low-osmolality contrast medium (Omnipaque 350 mg/mL; GE Healthcare, Cork, Ireland). All cases were administered a 300 mg dosage of aspirin together with either a 180 mg dose of ticagrelor or a 600 mg dose of clopidogrel previous to the intervention. Following the visualization of the vascular architecture, 100 U/kg of heparin was injected. The administration of glycoprotein IIb/IIIa inhibitors was determined by the physician's judgment. Subsequent to the procedure, all cases were relocated to ICU and treatment was maintained with hundred milligram of aspirin, 75 mg of clopidogrel, or 90 mg of ticagrelor administered bi-daily. Decisions about the simultaneous administration of angiotensin-converting enzyme inhibitors, statins, and beta-blockers were made in accordance with the 2020 ESC Guideline recommendations **[10].** Patients with a good general condition were allowed oral fluid intake 90 minutes after the procedure. Electrocardiogram and blood pressure monitoring were conducted in ICU, and blood samples were obtained. Patients were observed with plasma creatinine levels for seventy-two hours post-procedure.

Ethical Consideration

Institutional Review Board (IRB) decided if the study to be approved. An informed consent was obtained. Confidentiality and personal privacy were upheld throughout the research, and the obtained data was not utilized for any other purpose.

Statistical Analysis

The acquired data was subsequently analyzed using software and presented in tables or appropriate graphics by computer applications. The collected data was entered into the Statistical Package for the Social Sciences (SPSS-20 Inc., Chicago, Illinois, USA) for subsequent analysis. Frequency was employed to summarize qualitative data, whereas descriptive data was systematically arranged by type, mean, SD, and range for continuous data. The significance level was established at 0.05. We considered results statistically significant if the p-value was below 0.05. Statistical parameters were expressed as the average plus standard deviation, whereas qualitative variables were represented as total counts and percentages. A variety of statistical tests were employed for comparison, involving the student's t-test, the Mann-Whitney test, the chi-square test (χ^2), the Z-test for proportions, and the odds ratio (OR).

RESULTS

Table (1) showed that there was no statistically significant difference between the studied groups regarding age and sex. However, patients in group B had significantly longer time to reperfusion (door to ballon time) compared to group A with no AKI. Patients in group (B) were liable for complications on admission (HF, Need for mechanical ventilation) compared to group (A).

Table (2) showed that there was no statistically significant difference among either group concerning before PPCI s. creatinine. However, a statistically significant difference was found among two groups concerning to both after PPCI s. creatinine and estimated GFR. **Table (3)** showed that patients ingroup B had lower ejection fraction by modified Simpson method and global 2D fraction ,higher PASP and showed difference regarding to three parameters between both group was statistically significant. **Table (4)** showed that also negative correlation between post PPCI serum creatinine, and ejection fraction by Simpson method which was highly statistically significant ($p=\leq 0.001$).

Table (5): Analysis of the ROC curve of the pulmonary artery systolic pressure (PASP): On plotting the ROC curve of the PASP it was observed that the ratio significantly predicted the risk of AKI development in cases with STEMI ($p=\leq 0.001$). The AUC of the ratio was 0.6377, with optimal cut-off value of 45mmhg and a sensitivity was 59%, specificity was 78%, PPV of 90%, and NPV of 36.7% for prediction of AKI development in patients with STEMI. **Table (6)** revealed that highly statistically significant difference was found regarding MFS, ACT and pulmonary artery stiffness between both groups. **Table (7) Analysis of the ROC curve of the pulmonary artery stiffness (PAS):** On plotting the ROC curve of the PAS, it was observed that the ratio significantly predicted the risk of developing CIN in acute MI cases suffering primary PCI. ($p=\leq 0.001$). The AUC of the ratio was 0.913 with sensitivity was 93% and specificity was 85% for prediction of CIN development in in AMI cases undergoing primary PCI.

Table (1): Comparison of demographic data, door to ballon time or chest pain window and complications(heart failure and need for mechanical ventilation) ,before PPCI and after PPCI serum creatinine and after PPCI estimated GFR between group (A) and (B):

Demographic variables	Group (A) (n=101) Mean ± S.D		Group (B) (n=32) Mean ± S.D		t-test	P
Age	55.740±10.57		57.78±9.39		-1.2136	0.22
Chest pain window (hours)	8.44±4.1		10.28 ±4.4		-2.087	0.04
	No (%)		No (%)			
Heart failure	7 (7)		9(8.75)			0.001
Mechanical ventilation	2(2)		6(18.75)			0.002
Sex	N	%	N	%		
Male Female	80 21	79% 21%	10 22	32% 68%	0.006	0.52
Before PPCI s. creatinine	0.75±0.0084		0.77±0.14		-0.7955	0.43
After PPCI s. creatinine	0.8±0.085		1.62±1.69		-2.7489	0.0098
After PPCI eGFR	102.04 ±17.2		65.09 ±21.2		8.9648	0.0000017

Table (2) :Comparison among group (A)and group (B) regarding echocardiographic variables (ejection fraction by biplane method and 2D fraction and PASP)

Variables	Mean±S.D		t-test	P
	Group(A) n =101	Group(B) n =32		
E.F by biplane method	44.8±7.06	37.9±8.56	7.0656	0.00006
E.F by 2 D global strain	48.4±7.06	37.94±8.56	6.5365	0.0005
PASP	29.4 ±5.39	43.8±10.2	-7.354	0.0002

Table (3): shows Pearson correlation between post PCI s. creatinine , eGFR and PASP indicate PASP as predictor of AKI in STEMI patients regardless contrast volume

Variable	Pulmonary artery systolic pressure (PASP)	
	R	P
After PPCI serum creatinine	0.73	0.00002
After PPCI e. GFR	0.58	0.0005
	EF-Simpson's method	
	R	P
Creatinine post	-0.6178846	≤0.001

Table (4): Cutoff value of pulmonary artery systolic pressure (PASP)

Cut-off	AUC	*Sensitivity*	*Specificity*	Standard Error a	Significance level	*PPV*	*NPV*
32	0.6377	*59.43*	*78.12*	0.2697	≤0.001	*90*	*36.76*

PV= positive predictive value, NPV= negative predictive value.

Table (5): Comparison between group (A) and group (B) regarding pulmonary artery stiffness

Variables	Mean ±SD		t-test	P
	Group(A) n =101	Group(B) n =32		
maximal frequency shift (MFS)	1.012± 0.1806	29.93± 9.87	29.67	≤0.001
acceleration time (ACT)	106.33± 14.15	72.37±3.61	13.40	≤0.001
pulmonary artery stiffness	0.0097± 0.0023	0.416 ± 0.143	28.78	≤0.001

Table (6): ROC curve of pulmonary artery stiffness (PAS)

AUC	Sensitivity	Specificity	Sig.	95% CI	
				Lower	Upper
.913	93	85	<0.001	.829	.906

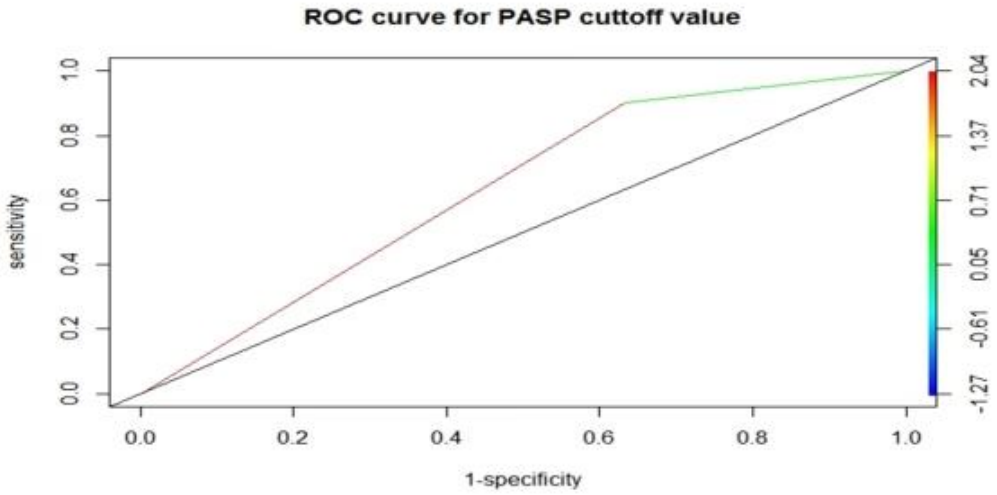

Fig (1): ROC of pulmonary artery systolic pressure for detection of cut off value.

DISCUSSION

The present study reported that no statistically significant differences was found among control and AKI group concerning age and sex.

Our findings reported that statistically significant differences were found among control and AKI group regarding mechanical ventilation, heart failure and chest pain window. As patients in group B had longer time to reperfusion (door to ballon time) and were liable for complications on admission compared to group A with no AKI.

This study was consistent with retrospective research of consecutive 930 STEMI cases who suffered primary PCI conducted by **Shacham et al.,[11]** who reported that cases with AKI were More likely to be older, own additional comorbidities, experience prolonged reperfusion time, and exhibit a lower baseline eGFR.

This study reported that no significant difference was found among two groups concerning before s. creatinine primary PCI however a statistically significant difference was found among both groups concerning s. creatinine after primary PCI and estimated GFR.

Similarly, this study agreed with **Bilen et al., [12]**who investigated whether the risk of CIN is heightened in STEMI cases with elevated SPAP on echocardiography. They reported that the first-day creatinine level after PCI and peak creatinine were significantly elevated in the CIN+ group in comparison to the CIN– group.

Our results showed that AKI group had significantly lower ejection fraction by modified Simpson method and global 2D fraction in comparison to control group. While AKI group had significantly higher PASP. This study reported that PASP showed a significant positive correlation with post primary PCI creatinine and estimated GFR. On the other hand, ejection fraction by Simpson method had significant negative association with post primary PCI creatinine.

Renal dysfunction in HF is due to diminished cardiac output, resulting in decreased blood flow and renal perfusion pressure**[13]**. Reduced cardiac output activates the renin–angiotensin–aldosterone system and the sympathetic nervous system, leading to congestion and constriction of afferent arterioles, which further diminishes renal perfusion pressure**[14].**

STEMI can lead to decreased LV pumping function, resulting in increased LV filling pressures. This, in turn, leads to an elevation in SPAP. Increased SPAP is frequently related to a reactive increase in pulmonary vascular resistance. further compounding the elevation in SPAP **[15].**

As a result, the pulmonary circulation following STEMI is marked by raised SPAP and

pulmonary vascular resistance, which augment RV afterload, potentially leading to RV dysfunction and ultimately RV failure **[16].**

Møller et al., [17] Examined 536 cases diagnosed with AMI who completed echocardiographic evaluations of LV systolic and diastolic function, as well as measurement of RV systolic pressure, and determined that an elevation in RV systolic pressure independently forecasted mortality following controlling for conventional risk factors and LV function.

Shacham et al., [11] revealed that cases with AKI had significant reduced LVEF, elevated SPAP, and increased RAP.

The current research revealed that the ideal cut-off value for PASP was 32 mmHg, exhibiting a sensitivity was fifty nine percent, specificity was seventy eight percent, PPV was ninety percent, and NPV was 36.7 percent for predicting AKI development in cases with STEMI.

Our findings in receiver operating characteristic curve analysis were consistent with **Bilen et al., [12].** It was observed that an SPAP over a threshold of 31.5 mmHg might indicate the existence of CIN, with a sensitivity was ninety-one percent and a specificity was ninety percent with p value < 0.001.

Furthermore, the current research were consistent with, **Shacham et al.,[11]** The best cutoff value of SPAP for predicting AKI was established as exceeding 32 mmHg, exhibiting a sensitivity was 72 percent and specificity was sixty-two percent (AUC 0.739, 95% CI 0.671–0.806 with P value < 0.001.

The current research reported that a highly statistically significant difference was found regarding MFS, ACT and pulmonary artery stiffness between both groups.

On plotting the ROC curve of the pulmonary artery stiffness, it was observed that the ratio significantly predicted risks related with progress of CIN in AMI cases suffering primary PCI. ($p=\leq 0.001$). The AUC of the ratio was 0.913 with sensitivity of 93% and specificity of 85% for prediction of CIN development in in acute MI patients undergoing primary PCI.

Similarly, **Ucar et al., [18]** It was determined that CIN was predicted by elevated aortic rigidity, detected by PWV.

RAAS activation, inflammation, endothelial dysfunction, and elevated vascular calcification are all related to arterial stiffness. A role is played by endothelin, angiotensin II, aldosterone, and nitric oxide in progress of AS and the pathophysiology of CIN **[19]** The impedance disparity among the central and peripheral arteries is diminished by raised arterial stiffness. This results in a high pulsatile pressure, elevated peripheral microcirculation, and vascular injury by disrupting the pressure buffering capacity of the arteries. This mechanism may explain the increased risk of developing CIN in AS with renal arteriole injury brought on by increasing pulsatile pressure **[20].**

CONCLUSIONS

We concluded that pulmonary artery stiffness and PSAP may be an independent indicator for the advancement of CIN in acute MI cases suffering primary PCI. The simplicity of PSAP measurement using echocardiography makes it a potentially valuable tool in clinical practice for preventing CIN in acute MI cases suffering primary PCI. Larger randomized studies are essential to further elucidate the correlation between PAS, PSAP and CIN, and to determine whether PSAP and PAS constitute a robust risk factor for the future development of CIN.

Conflict of interest:

The authors declare no conflict of interest**.**

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