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The Utility of Serum Procalcitonin Level as an Early Diagnostic Biomarker for COVID in Patients Presenting with ST-Elevation Myocardial Infarction Post Primary Percutaneous Coronary Intervention

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ABSTRACT

Objectives: The aim of the study was to detect if serum procalcitonin could be used as an early diagnostic marker for COVID in asymptomatic patients presenting with ST-segment elevation myocardial infarction and if procalcitonin is related to the extent of myocardial injury after the infarction.

Patients and Methods: The study was carried out on 150 patients (age 53.39 ± 13.16) presenting to Ain Shams University Hospitals with ST-segment elevation myocardial infarction (STEMI) and received the standard of care management, which is primary percutaneous coronary intervention (PCI). The patients were assessed regarding the demographic data, presence of any symptoms and signs of infection, risk factors, echocardiography and laboratory measures. COVID PCR and procalcitonin were assessed for all patients.

Results: Out of all the asymptomatic patients presenting with STEMI whose COVID PCR turned out to be positive, the procalcitonin was positive 24 and 48 hours post presentation with P value <0.001. There was high statistically significant relation between positive procalcitonin and total leucocytic count (TLC) where there was a drop from a median of 11/mm^3 with range (4.5– 19) /mm^3 in negative procalcitonin to a median of 3.7 /mm^3 with range (1.4 –12) /mm^3 in procalcitonin positive patients. There was high statistically significant relation between positive procalcitonin and rise of C-reactive protein (CRP) value where there was a rise from CRP median value from 12 mg/dL range (2–40) mg/dL in negative procalcitonin patients to 47 mg/dL range (14– 163) mg/dL in positive procalcitonin patients.

Conclusion: There was a strong relationship between procalcitonin and COVID infection in asymptomatic patients thus procalcitonin can be used as an early diagnostic biomarker for COVID infection in patients presenting with myocardial infarction.

Keywords: STEMI, COVID, Procalcitonin.

INTRODUCTION

The most prevalent type of cardiac illness is coronary artery disease (CAD). It arises from atheromatous modifications in the blood arteries feeding the heart. Acute coronary syndrome (unstable angina, NSTEMI, STEMI), asymptomatic atherosclerosis, and stable angina are all clinical diseases referred to as CAD [1].

One of the biggest causes of death globally is coronary artery disease. According to the World Health Organization (WHO), ischemic heart disease caused almost nine million fatalities in 2016. The mortality rate from CAD shows contrasting patterns in developed and developing nations. Ischemic heart disease-related death rates are declining in wealthy nations like the U.S. and the UK. Nevertheless, 16.5 million Americans over the age of 20 had coronary artery disease in 2018, with 55% of those affected being men. With rising mortality trends, the condition of CAD is getting worse in emerging nations [2].

The presence of acute myocardial injury indicated by abnormal cardiac biomarkers in the presence of evidence of acute myocardial ischemia indicated by the detection of an elevated cardiac troponins (cTn) value above the 99th percentile is what is referred to as myocardial infarction in clinical terms. If the values of cTn increase or decrease, the damage is deemed acute. Provided the fourth accepted definition of myocardial infarction (2018) [3].

It is standard procedure to classify patients with persistent chest pain or other symptoms indicative of ischemia and ST-segment elevation in at least two contiguous leads as STEMI in order to facilitate rapid treatment measures such reperfusion therapy [4].

COVID cases have been increasing significantly and it was associated with a lot of CAD cases. Most of the patients present asymptomatically but it's related to increased risk of morbidity and mortality in these patients.

In critically unwell patients, procalcitonin (PCT) is recognised as a biological diagnostic sign for severe sepsis or septic shock. The significance of procalcitonin in patients with acute myocardial infarction and those with acute coronary syndromes, that is, non-ST-elevation myocardial infarction or unstable angina, is still controversial due to conflicting evidence. There are conflicting findings regarding procalcitonin rise in COVID-19 subjects [5].

AIM OF THE WORK

Primary objective: To investigate the utility of serum procalcitonin as an early diagnostic biomarker for COVID in asymptomatic patients presented with STEMI.

Secondary objective: To evaluate the association between procalcitonin release in the acute setting of STEMI with the size and extent of myocardial infarction.

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PATIENTS AND METHODS

The study was conducted in Ain Shams University Hospitals on 150 patients presented with ST-segment elevation myocardial infarction and underwent primary PCI. All patients received the standard of care for acute STEMI patients, which is the primary percutaneous intervention in addition to the medical treatment according to the European Society of Cardiology (ESC) guidelines [6].

Proper history for demographical data, risk factors, analysis of chest pain and analysis of any symptom denoting infection was done. Proper examination for vital data, heart and chest auscultation and signs for any infection was done. Patients underwent echocardiography for assessment of extent of myocardial injury. General laboratory parameters, procalcitonin 24 and 48 hours post PCI and COVID PCR were assessed for all patients.

Ethical approval:

Before beginning the study, permission was sought from the Ain Shams University Ethics Committee. Each participants provided an informed written consent with consideration of adequate privacy and confidentiality. The Declaration of Helsinki for human beings, which is the international medical association's code of ethics, was followed during the conduct of this study.

Inclusion criteria:

- A patient with ST-elevation myocardial infarction is one who will have primary PCI within 24 hours of the onset of symptoms and who has symptoms of ischemia and ST-segment elevation of at least two contiguous leads with ST-segment elevation of ≥2.5 mm in men <40 years, ≥2 mm in men ≥40 years, ≥1.5 mm in women, and/or 1 mm in the other leads [7,8].
- Patients having Killip classification lower than III.
- Glomerular filtration rate estimated to be more than 30 mL/min/1.73 m^2.
- Both sexes were included.
- No age predilection, but children were not included.

Exclusion criteria:

- A history of coronary intervention or myocardial infarction.
- Patients with clinical evidence of infection including (cough, expectoration, sore throat, fever, dysuria, or skin infection).
- Patients with objective evidence of infection including (chest X-ray, CT chest, blood culture or urine analysis) were excluded.
- Patients having Killip III-IV classification.
- Patients with postponed primary PCI for any reason were excluded.

Methods

A checklist for evaluating all the clinical information that is pertinent to the patients was used. To create a data-based system for each patient, all of these sheets were gathered, a computerised system was used to enter the data, ensuring that the obtained information was kept secret.

All patients were subjected to:

Careful history taking including

a. Clinical, demographic data including age and sex, risk factors, co-morbidities including smoking, diabetes, hypertension, obesity, dyslipidemia and presence of family history.
b. Characterization of chest pain (onset, duration, site, radiation, character, pain to door, and residual pain).
c. Symptoms suggestive for any source of infection (nasal congestion, cough, expectoration, sore throat, dysuria, urgency, frequency, bloody urine, or diarrhea).

Comprehensive physical examination including:

a. Vital signals, such as heart rate and blood pressure.
b. A lung and heart auscultation.
c. Temperature measurement.
d. Measurement of oxygen saturation.
e. Meticulous inspection for any dermatological source of infection specially in diabetic patients.

Investigations:

a. 12 lead surface ECG.
b. CBC.
c. CRP.
d. Highly sensitive cardiac troponins.
e. Procalcitonin 24 and 48 hours post-PPCI.
f. COVID PCR.
g. CT chest.
h. Pan-cultures.
i. Trans-thoracic echocardiography.

Interventional phase:

- All patients received 300 mg of aspirin and either 180 mg or 600 mg of ticagrelor or clopidogrel.
- According to European Society of Cardiology 2017 recommendations for the therapy of myocardial infarction in patients presenting with ST-segment elevation myocardial infarction, the main PCI was carried out by a skilled interventional cardiologist [6].
- All patients were admitted to the CCU for follow up.
- The standard treatment was given to all patients according to the ESC guidelines including: ACEI or ARNi, a beta-blocker, and an MRA uptitrated according to the clinical state of the patient.

- COVID PCR was withdrawn from all the patients.
- Procalcitonin was withdrawn 24 and 48 hours post primary percutaneous intervention.

Imaging data [9,10]:

- Transthoracic echocardiogram post PPCI to establish a baseline LVEF was assessed by Modified Simpson's method.
- All echocardiographic studies were done in Echocardiography Unit of Cardiology Department Ain Shams University Hospitals, by the same Echo machine GE VIVED S5 (USA).
- All the measures were taken in left lateral decubitus and ranges for two-dimensional echocardiography obtained of LVEF, LVED and LVES were compared to normal reference ranges as per the American Society of Echocardiography and the European Association of Cardiovascular Imaging.
- Segmental wall motion abnormalities and the degree of mitral regurgitation were assessed.

Statistical analysis:

Data were gathered, reviewed, coded, and put into the Statistical Package for the Social Sciences (SPSS) IBM version 20 of. Quantitative information was provided as means, standard deviations, and ranges when parametric, and medians with interquartile ranges (IQR) when non parametric. Qualitative information was presented as numbers and percentages. The comparison between two groups utilising qualitative data was made using the Chi-square test or the Fisher exact test when the predicted count in any cell was less than 5. The Independent t-test was used to compare two groups' quantitative data with parametric distribution, whereas the Mann-Whitney test was used for data with non-parametric distribution. Wilcoxon rank test was used for comparison of paired data with a non-parametric distribution. The allowable margin of error was set at 5%, while the confidence interval was set at 95%. P value less than 0.05 was regarded as significant.

Description of the demographic data, risk factors, echocardiographic parameters and laboratory findings among all studied population:

The mean age of our study group was 53.39 ± 13.16 ranging from 29 to 82 with 69.3% males and 30.7% females. The most prevalent risk factor was smoking affecting 95 patients (63.3%), DM was the next affecting 75 patients (50%), the third was Hypertension affecting 73 patients (48.7%), Dyslipidemia (44%), Obesity (31.3%) and Family History (3.3%).

Echocardiography was used to assess degree of myocardial injury among our patients. The parameters assessed were ejection fraction (EF), LVEDV, LVESV, the extent of segmental wall motion abnormalities and degree of mitral regurgitation. EF was ranging from (20 to 70%) with mean of 45.62 ± 13.02, LV dimensions were obtained with range of (38 – 76 mm) and (28-63 mm) for end diastolic and end systolic volumes. 13.3% of our patients had no mitral regurgitation, 7.3% had trivial MR, 28.6 % has mild MR, 34.7% has moderate MR and 16% has severe MR.

Different laboratory parameters were assessed in the patients including a CBC (Hemoglobin, platelets and total leucocytic count), chemistry including serum creatinine and CRP, HbA1C and lipid profile including cholesterol, LDL and triglycerides.

Description of the Procalcitonin levels 24 and 48 hours post presentation and its relation to all parameters for all studied patients:

Procalcitonin was assessed 24 and 48 hours post presentation in all patients. The 24 hours values ranged from (0.01-0.8) ng/mL with a mean of 0.04 ng/mL and the 48 hours value ranged from (0-0.8) ng/mL with a mean value of 0.07 ng/mL. 43 patients were positive (28.6%) of whom 58.1% were males and 41.9% were females and 107 patients were negative (71.3%) of whom 73.8% were males and 26.2% were females.

There was no statistically significant relation between procalcitonin and demographical data of the patient [Table 1], risk factors and type of STEMI [Table 2], extent of myocardial injury [Table 3], and laboratory parameters (Hemoglobin, platelets, serum creatinine, HbA1C, lipid profile) [Table 4]

RESULTS

Table (1): Relation of procalcitonin to demographical data of the patients

		Procalcitonin 24 and 48 hours		Test value	P- value	Sig.
		Negative	Positive			
		No.= 107	No.= 43			
Age	Mean±SD	53.27 ± 13.82	53.70 ± 11.49	-0.179•	0.858	NS
	Range	29 – 81	34 – 82			
Sex	Female	28 (26.2%)	18 (41.9%)	3.552*	0.059	NS
	Male	79 (73.8%)	25 (58.1%)			

Table (2): Relation of procalcitonin 24 hours and 48 hours with risk factors, type of STEMI in our study population

		Procalcitonin 24 and 48 hours		Test value*	P- value	Sig.
		Negative	Positive			
		No.= 107	No.= 43			
Smoking	No	37 (34.6%)	18 (41.9%)	0.700	0.403	NS
	Yes	70 (65.4%)	25 (58.1%)			
Hypertensive	No	54 (50.5%)	23 (53.5%)	0.112	0.738	NS
	Yes	53 (49.5%)	20 (46.5%)			
Diabetic	No	57 (53.3%)	18 (41.9%)	1.597	0.206	NS
	Yes	50 (46.7%)	25 (58.1%)			
Dyslipidemia	No	62 (57.9%)	22 (51.2%)	0.572	0.449	NS
	Yes	45 (42.1%)	21 (48.8%)			
Family History	No	78 (72.9%)	25 (58.1%)	3.105	0.078	NS
	Yes	29 (27.1%)	18 (41.9%)			
STEMI	Anterior	43 (40.2%)	20 (46.5%)	0.648	0.958	NS
	Posterior	12 (11.2%)	5 (11.6%)			
	Inferior	25 (23.4%)	9 (20.9%)			
	Lateral	13 (12.1%)	4 (9.3%)			
	Inferoposterior	14 (13.1%)	5 (11.6%)			

NS: Nonsignificant

Table (3): Relation of procalcitonin 24 hours and 48 hours with degree of myocardial injury in our study population

		Procalcitonin 24 and 48 hours		Test value	P- value	Sig.
		Negative	Positive			
		No.= 107	No.= 43			
Ejection Fraction	Mean±SD	46.07 ± 44.42	44.49 ± 11.10	0.674	0.501	NS
LVEDV (mm)	Mean±SD	52.31 ± 9.19	53.30 ± 8.56	-0.611	0.542	NS
LVESV (mm)	Mean±SD	39.06 ± 9.16	39.58 ± 7.53	-0.333	0.739	NS
MR degree	None	16 (15.0%)	4 (9.3%)	5.014	0.286	NS
	Mild	35 (32.7%)	8 (18.6%)			
	Moderate	33 (30.8%)	19 (44.2%)			
	Severe	16 (15.0%)	8 (18.6%)			
	Trivial	7 (6.5%)	4 (9.3%)			

NS: Nonsignificant

There was high statistically significant relation between positive procalcitonin and drop in TLC. There was high statistically significant relation between positive procalcitonin and rise of CRP value [Table 4]. There was high statistically significant relation between the positive procalcitonin and positive PCR value in which all 43 patients with positive PCR had positive procalcitonin. [Table 4 and Figure 1].

Table (4): Relation of procalcitonin 24 hours and 48 hours with laboratory parameters in our study population

		Procalcitonin 24 and 48 hours		Test value	P- value	Sig.
		Negative	Positive			
		No.= 107	No.= 43			
Troponins (ng/ml)	Median (IQR) Range	28500 (8600 – 50000) 400 – 50000	39600 (17800 – 50000) 1200 – 50000	-1.697	0.090	NS
Hb (g/dl)	Mean±SD	11.74 ± 3.06	11.84 ± 2.89	-0.182	0.856	NS
TLC (/mm3)	**Median (IQR)** **Range**	**11 (7.9 – 13.6)** **4.5 – 19**	**3.7 (2.5 – 5)** **1.4 –12**	**-8.466**	**<0.001**	**HS**
PLT (/mm3)	Mean±SD	303.84 ± 132.55	344.05 ± 153.70	-1.603	0.111	NS
Creatinine (mg/dl)	Mean±SD	1.16 ± 0.41	1.15 ± 0.40	0.207	0.837	NS
HbA1c (%)	Mean±SD	6.49 ± 2.05	6.76 ± 2.02	-0.738	0.462	NS
CRP (mg/dl)	**Median (IQR)** **Range**	**12 (9 – 20.5)** **2 – 40**	**47 (34 – 89)** **14 – 163**	**-8.665**	**<0.001**	**HS**
Cholesterol (mg/dl)	Mean±SD	193.91 ± 38.81	196.93 ± 33.57	-0.448	0.655	NS
Triglycerides (mg/dl)	Mean±SD	251.80 ± 94.53	267.30 ± 106.08	-0.876	0.382	NS
LDL (mg/dl)	Mean±SD	99.54 ± 24.71	103.35 ± 29.17	-0.809	0.420	NS
PCR	**Negative** **Positive**	**107 (100.0%)** **0 (0.0%)**	**0 (0.0%)** **43 (100.0%)**	**150.000***	**<0.001**	**HS**
Obesity	No Yes	75 (70.1%) 32 (29.9%)	25 (58.1%) 18 (41.9%)	1.972	0.160	NS

Median (IQR), Range: Non-parametric data. NS: Nonsignificant, HS: Highly significant

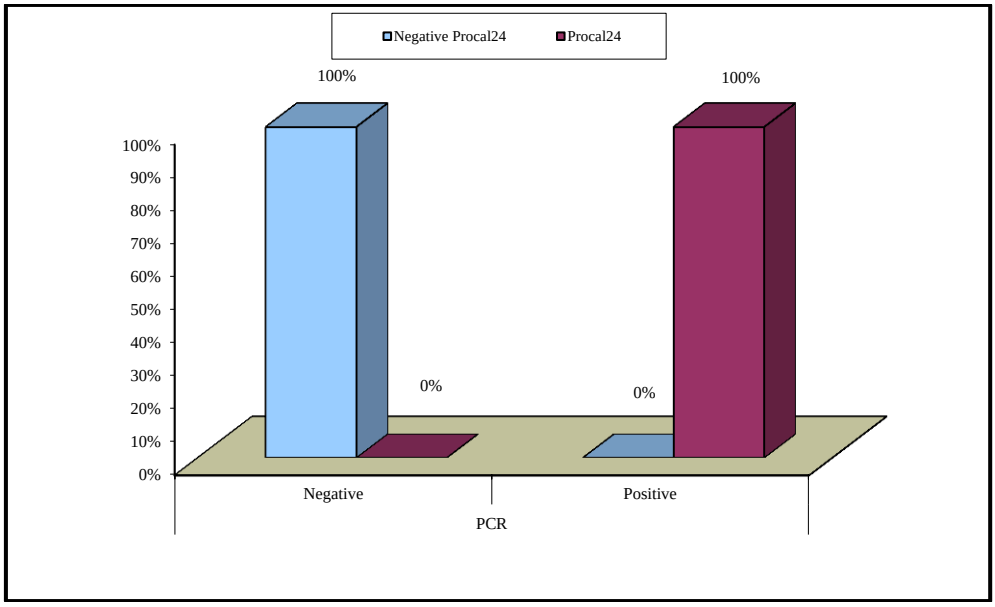

Figure (1): - A descriptive figure showing relationship between procalcitonin positivity at 24 and 48 hours and PCR

Description of the PCR and its relation with all parameters for all studied patients:

COVID PCR was assessed for all our study group. 43 were positive (28.7%) of whom 58.1% were males and 41.9% were females and 107 patients were negative (71.3%) of whom 73.8% were males and 26.2% were females [Table 5].

Table (5): - Table showing PCR values among our study population.

PCR	No.	%
Negative	107	71.3%
Positive	43	28.7%
Total	**150**	**100.0%**

There was no statistically significant relation between PCR positivity and demographical data of the patients, risk factors [Table 6].

Table (6): - Comparison between PCR positive and PCR negative among Cardiovascular risk factors in our study population

		Negative PCR		Positive PCR		Test value*	P-value	Sig.
		No.	%	No.	%			
Smoking	No	37	34.6%	18	41.9%	0.700	0.403	NS
	Yes	70	65.4%	25	58.1%			
Hypertensive	No	54	50.5%	23	53.5%	0.112	0.738	NS
	Yes	53	49.5%	20	46.5%			
Diabetic	No	57	53.3%	18	41.9%	1.597	0.206	NS
	Yes	50	46.7%	25	58.1%			
Dyslipidemia	No	62	57.9%	22	51.2%	0.572	0.449	NS
	Yes	45	42.1%	21	48.8%			
Family History	No	78	72.9%	25	58.1%	3.105	0.078	NS
	Yes	29	27.1%	18	41.9%			
Obesity	No	75	70.1%	25	58.1%	1.972	0.160	NS
	Yes	32	29.9%	18	41.9%			
STEMI	Anterior	43	40.2%	20	46.5%	0.648	0.958	NS
	Posterior	12	11.2%	5	11.6%			
	Inferior	25	23.4%	9	20.9%			
	Lateral	13	12.1%	4	9.3%			
	Inferoposterior	14	13.1%	5	11.6%			

NS: Nonsignificant

There is high statistically significant relation between procalcitonin and PCR positivity with a rise of median value of procalcitonin 24 and 48 hours post presentation [Table 7].

Table (7): Comparison between PCR positive and PCR negative with procalcitonin 24 and 48 hours post presentation

Procalcitonin (ng/mL)		Negative PCR	Positive PCR	Test value‡	P-value	Sig.
		No. = 107	No. = 43			
24 hours	Median (IQR)	0.03 (0 – 0.06)	1.4 (0.9 – 1.6)	-9.638	<0.001	HS
	Range	0 – 0.4	0.6 – 2			
48 hours	Median (IQR)	0.04 (0 – 0.09)	1.5 (1 – 1.9)	-9.645	<0.001	HS
	Range	0 – 0.3	0.5 – 2			
Wilcoxon Rank test		**-1.327**	**-3.359**			
P-value		**0.185 (NS)**	**0001 (HS)**			

NS: Nonsignificant, HS: Highly significant

Our study reached a conclusion that at a procalcitonin value of more than 0.4 ng/dL in 24 hours and 0.3 ng/dL in 48 hours, PCR is positive with high sensitivity reaching 100 % [Table 8 & Figure 2].

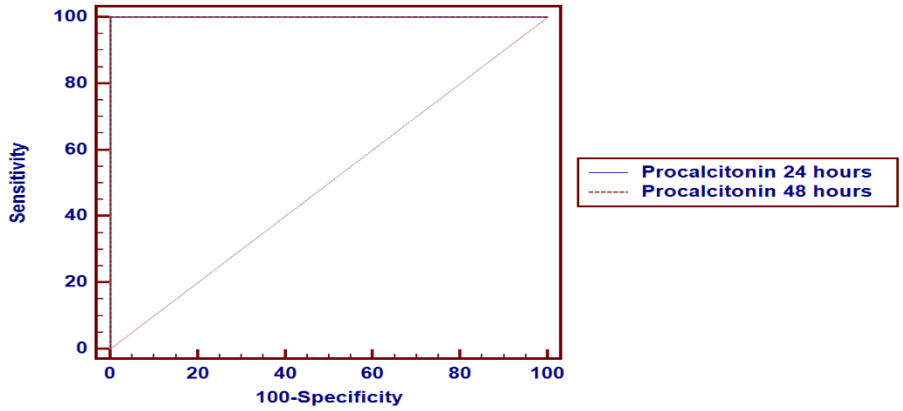

Figure (2): ROC curve of procalcitonin as a predictor of positivist of PCR

Table (8): Cut off point values for suspecting COVID infection

Procalcitonin	AUC	Cut of Point	Sensitivity	Specificity	PPV	NPV
24 hours	**1.000**	**>0.4**	**100.0**	**100.0**	**100.0**	**100.0**
48 hours	**1.000**	**>0.3**	**100.0**	**100.0**	**100.0**	**100.0**

DISCUSSION

The number of COVID patients is still increasing, and this increase is being accompanied by an increase in hospitalisation. When the COVID-19 pandemic broke out, healthcare institutions underwent a massive reorganisation that restricted access to emergency care and decreased or even stopped elective treatments[11].

The best course of treatment for ST-segment elevation myocardial infarction (STEMI) requires rapid referral for primary percutaneous coronary intervention (PPCI) [12].

The first wave of the COVID-19 pandemic, in particular, had a significant influence on how acute disorders like acute myocardial infarction (AMI) and stroke were treated. Because of resource shortages brought on by the excessive frequency of hospitalisations connected to COVID-19, catheterization labs were transformed into COVID-19 critical care units. Additionally, delays in providing acute STEMI treatment might have been brought on by extended triage while awaiting the results of the COVID-19 test [13].

A sensitive and specific biomarker for bacterial sepsis is procalcitonin. The amount and timing of the rise can be directly correlated with the patient's result and the bacterial load. The increase in plasma level increases in direct proportion to the severity of the illness. There are currently little and conflicting data on serum PCT levels in individuals with cardiogenic shock and acute coronary syndromes (ACS). When ACS patients are admitted, some studies claim that PCT levels rise; nevertheless, other research show that plasma PCT concentrations are within the normal range [14].

Our study was conducted on 150 patients aiming at finding an early predictive marker for COVID in patients presenting asymptomatically with ST elevation myocardial infarction to facilitate faster triaging, to avoid any revascularization delay and then provide the proper management for these patients regarding the later on accommodation into a COVID free or a COVID positive area and starting the COVID regimens earlier for better prognosis and outcomes in the ischemic patients.

For this purpose, we need to exclude any relationship between procalcitonin and extent of myocardial injury.

Procalcitonin and myocardial injury:

There are few and conflicting data on PCT levels in individuals with cardiogenic shock and ACS. PCT appears to be more sensitive to a larger degree of inflammatory activation across the whole range of ACS, from unstable angina to cardiogenic shock following STEMI, being positive in patients with cardiogenic shock.

Our study showed no elevation in procalcitonin in any of the 107 uncomplicated STEMI cases either 24 nor 48 hours post- revascularization.

The extent of myocardial impairment was assessed in our study by drop in left ventricular function and increase in left ventricular diameters.

Study population included in our study had no previous PCI nor a strong ischemic history to avoid any previously existing impaired functions.

The procalcitonin value was negative in all uncomplicated STEMI cases regardless of ejection fraction ranging from 20-70% in our population with a mean value 46.07 ± 13.17. When compared to patients with positive procalcitonin values, the EF ranged from 20-66% with a mean value 44.49 ± 12.71, thus the P-value was 0.501 showing non-significant correlation between procalcitonin and ejection fraction. Other parameters were used to assess extent of myocardial injury in our study; the increase in LVEDV and LVESV. The mean value for LVEDV in procalcitonin negative patients was 52.31mm ± 9.19 as compared to

53.30 mm ± 8.56 in procalcitonin positive patients with a P-value 0.542 showing non-significant correlation. The same goes for LVESV with a mean value 39.06 mm ± 9.16 in procalcitonin negative patients as compared to 39.58 mm ± 7.53 in procalcitonin positive patients with a P-value 0.739 proving non-significant correlation. Also, the degree of mitral regurgitation was used as a method for assessment of degree of myocardial injury; the P-value between positive and negative patients was 0.286 proving non-significant correlation.

This was supported by a study by **Claudio *et al.*** [14] on 52 patients who presented with acute ischemia and came to the conclusion that C-reactive protein, which was positive in the majority of acute cardiac care patients of all their subgroups, rather than PCT, which appears more sensitive to a higher degree of inflammatory activation, is a better indicator of the degree of myocardial injury following acute ischemia and the related inflammatory-induced response. The study only included 15 uncomplicated STEMI cases and focused on relationship between procalcitonin and CRP with shocked status. It didn't perform a serial procalcitonin measurement. Procalcitonin was assessed on admission and it was correlated to severity of ischemia at time of presentation as unstable angina, NSTEMI or STEMI. Procalcitonin wasn't correlated to any of the parameters assessing the degree of myocardial injury.

Sentürk *et al.* [15] carried out a study on 50 patients with ACS including unstable angina, NSTEMI and STEMI patients; correlating CRP and procalcitonin to severity of CAD. They used coronary angiography as a method to evaluate the severity of CAD reaching the conclusion that except in the group of patients with unstable angina who underwent revascularization surgery, there was no connection between procalcitonin and hsCRP levels at admission and after 48 hours and the primary end points after 3 months.

On the other hand, **Kafkas *et al.*** [5] investigated PCT levels in 60 patients who were admitted with STEMI and found that PCT concentrations are higher than those of CK-MB or troponin I in the majority of patients. Procalcitonin levels were tested at admission, 3, 6, 12, 24, 48, and 72 hours, as well as on the seventh day. Their findings demonstrated an increase in PCT levels that peaked after 12–24 hours and then declined to a plateau by the seventh day. The release of PCT in AMI is most likely caused by the inflammatory process that takes place during AMI, the scientists found, and as such, PCT might be thought of as a novel sensitive myocardial indicator. In this study, the PCT levels were correlated to the IL-6 and CRP only. No parameters for functional assessment were included.

Another study carried out by **Verma *et al.*** [16] focused-on use of procalcitonin as a prognostic value for the acute ischemic events, predicting the long-term outcomes of the patients regarding morbidity and mortality. This study was carried out in India on 250 patients admitted with STEMI and was followed up regarding ischemic complications; arrhythmia, shock and heart failure. It showed that procalcitonin at the time of admission is a good predictor for the long-term outcomes of the patients and a good predictor of mortality.

Procalcitonin and COVID infection:

COVID-19, despite being a viral infection, many studies are being conducted to detect usefulness of procalcitonin in predicting and stratifying the severity of COVID. It has been known that procalcitonin is effective as an inflammatory marker in detecting bacterial infection and for suspecting superadded infection in virally infected patients. It is believed that bacteria and viruses regulate procalcitonin production through interferon signaling[17].

There is mounting evidence that many COVID-19 individuals are asymptomatic or only have minor symptoms, yet they can still spread the virus to other people. It is challenging to screen for asymptomatic infections, which makes it more challenging to prevent and control this epidemic nationally[17].

When SARS-CoV-2 nucleic acid is positively identified in patient samples by RT-PCR, an infection is considered asymptomatic if there are no conventional clinical symptoms or signs, and there are no obvious abnormalities in imaging, including lung computed tomography. Numerous studies have demonstrated that they are a significant source of illness dissemination and have the same infectivity as symptomatic individuals [17]. Many studies focused on correlation between procalcitonin and severity of COVID infection and its efficacy as a prognostic value, but no studies tested the efficacy of procalcitonin as a predictive marker for asymptomatic COVID infection.

All the patients chosen to be included in our study were thoroughly investigated. They all reported no symptoms of infection and all laboratory and imaging investigations came out free.

Our study showed high correlation between procalcitonin and asymptomatic COVID infection. All of the 43 COVID patients were asymptomatic at presentation and the infection was confirmed by a positive PCR. The value was confined between 0.5 ng/dL and 2 ng/dL; a value for systemic infection not reaching the septic range; above 2 ng/dL.

A procalcitonin cut-off point for suspecting COVID in STEMI patients being above 0.4 ng/dL in 24 hours and above 0.3 ng/dL in 48 hours was supported by our statistics.

By this we can limit the COVID PCR use in asymptomatic patients to only those meeting the procalcitonin cut-off points.

The relationship between procalcitonin and COVID was supported in a study carried on 95 COVID patients conducted by **Hu *et al.*** [18] and it

focused mainly on relation between the procalcitonin levels and severity of infection in which higher procalcitonin indicated worse prognosis and higher mortality. All patients included in the study were symptomatic and were stratified at time of admission according to the severity of infection into moderate, severe and critical. According to the study, in severe patients compared to moderate patients, the mean blood procalcitonin levels were over four times higher, and in critical patients compared to moderate patients, they were over eight times higher.

CONCLUSION

Our study reached the conclusion that procalcitonin doesn't relate to myocardial infarction nor degree of myocardial injury as TLC and CRP do. This was assessed using ejection fraction, changes in left ventricular dimensions and degree of mitral regurgitation.

Despite being a viral infection, procalcitonin can be used as an early predictive marker for COVID in early presenting asymptomatic patients and can be used later on to stratify the patients according to severity.

In procalcitonin levels above 0.4 ng/mL after 24 hours and above 0.3 ng/mL after 48 levels, COVID should be suspected, patients should be isolated, a PCR should be assessed to confirm the diagnosis and COVID protocol should be started as early as possible for better prognosis for the STEMI patients.

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REFERENCES

1. **Lopez E, Ballard B, Jan A (2021):** Cardiovascular Disease. StatPearls Publishing; Treasure Island (FL). https://pubmed.ncbi.nlm.nih.gov/30571040/
2. **Nowbar A, Gitto M, Howard J *et al.* (2019):** Mortality from ischemic heart disease. Circ Cardiovasc Qual Outcomes, 12(6): e005375. doi: 10.1161/CIRCOUTCOMES.118.005375.
3. **Benjamin E, Virani S, Callaway C *et al.* (2018):** American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2018 Update: A Report from the American Heart Association. Circulation, 137(12): 67-492.
4. **Almdal T, Scharling H, Jensen J *et al.* (2004):** The independent effect of type 2 diabetes mellitus on ischemic heart disease, stroke, and death: a population-based study of 13,000 men and women with 20 years of follow-up. Arch Intern Med., 164: 1422-26.
5. **Kafkas N, Venetsanou K, Patsilinakos S *et al.* (2008):** Procalcitonin in acute myocardial infarction. Acute Cardiac Care, 10: 30-36.
6. **Ibanez B, James S, Agewall S *et al.* (2018):** 2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J., 39(2):119-177.
7. **Eitel I, Stiermaier T, Rommel K *et al.* (2015):** Cardioprotection by combined intrahospital remote ischaemic preconditioning and postconditioning in ST-elevation myocardial infarction: the randomized LIPSIA CONDITIONING trial. Eur Heart J., 36(44):3049-3057.
8. **Thygesen K, Alpert J, Jaffe A *et al.* (2018):** Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol., 72(18):2231-2264.
9. **Behnia M, Powell S, Fallen L *et al.* (2013):** Correlation of stroke volume measurement between sonosite portable echocardiogram and Edwards Flotrac sensor-Vigileo monitor in an intensive care unit. Clin Med Insights Circ Respir Pulm Med., 7:45–51.
10. **Fred A, Mettler J, Milton J *et al.* (2012):** Essentials of Nuclear Medicine Imaging. 6th ed., Publisher: Elsevier/Saunders, Philadelphia, https://www.worldcat.org/title/essentials-of-nuclear-medicine-imaging/oclc/778785848
11. **WHO (2022):** Rapidly escalating COVID-19 cases amid reduced virus surveillance forecasts a challenging autumn and winter in the WHO European Region. https://www.who.int/europe/news/item/19-07-2022-rapidly-escalating-covid-19-cases-amid-reduced-virus-surveillance-forecasts-a-challenging-autumn-and-winter-in-the-who-european-region
12. **De Luca G, Verdoia M, Cercek M *et al.* (2020):** Impact of COVID-19 pandemic on mechanical reperfusion for patients with STEMI. J Am Coll Cardiol., 76: 2321–30.
13. **Alvarez M, Boucher T, Terre J *et al.* (2021):** Surge-in-place: conversion of a cardiac catheterization laboratory into a COVID-19 intensive care unit and back again. J Invasive Cardiol., 33: 71–6.
14. **Picariello C**, **Lazzeri** C, **Valente** S ***et al.* (2011):** Procalcitonin in acute cardiac patients. Int J Cardiol., 72(1):3-10.
15. **Sentürk T, Cordan J, Baran I *et al.* (2007):** Procalcitonin in patients with acute coronary syndrome: correlation with high-sensitive C-reactive protein, prognosis and severity of coronary artery disease. Acta Cardiol., 62(2):135-41.
16. **Verma S, Pratap M, Mathur S (2021):** A study of serum procalcitonin levels as a prognostic marker in patients admitted with ST Elevation Myocardial Infarction. International Journal of Scientific Research, 10: 78-81.
17. **Chen Y, Wang A, Yi B *et al.* (2020):** The epidemiological characteristics of infection in close contacts of COVID-19 in Ningbo city. Chin J Epidemiol., 41: 667-71.
18. **Hu R, Han C, Pei S *et al.* (2020):** Procalcitonin levels in COVID-19 patients. International Journal of Antimicrobial Agents, 56(2): 106051. doi: 10.1016/j.ijantimicag.2020.106051.