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Original Article

Evaluation of intracoronary blood from obstructive vessel in patients of ST-elevation myocardial infarction undergoing PPCI☆

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ABSTRACT

Objective: Information available on acid–base imbalance in ST-elevation myocardial infarction (STEMI) submitted to primary percutaneous intervention is limited and no data were present on intracoronary blood analysis, extracted from obstructed artery.

Methods: This was a prospective study conducted over 12 months in which STEMI patients presenting in emergency and undergoing primary percutaneous coronary intervention were included. Blood gas analysis of intracoronary arterial blood from obstructed vessel and peripheral arterial blood was performed. Patients in whom adequate intracoronary sample could not be obtained were excluded. Intracoronary and peripheral arterial blood gas measurements were correlated and relationship of intracoronary parameters were compared with clinical parameters, investigational markers and short-term outcome.

Results: The mean age of study population was 54.8 years and average symptom onset to door time was 162 min. On comparing intracoronary blood with peripheral blood arterial obtained, pH (95% confidence interval [CI] −0.01 to 0.02;$p = 0.44$), lactate (95% CI 0.03–0.1;$p = 0.28$), bicarbonate (95% CI 0.6 −1.5;$p = 0.64$), pCO_2 (95% CI 1.1–2.4;$p = 0.79$) and pO_2 (95% CI 3.2–47.5; $p = 0.06$) were all found to be statistically insignificant. Intracoronary hyperlactatemia was present in patients presenting with higher symptom onset to door time ($p = 0.025$). Systolic blood pressure (SBP) ($p = 0.03$) was also significantly lower in patients who had high intracoronary lactate levels.

Conclusion: The evaluation of intracoronary blood provides no additional information regarding the prognosis and short-term (30-day) outcome of the patients when compared with peripheral blood. However, there was a significant intracoronary hyperlactatemia in patients presenting late after symptom onset. SBP was also significantly less in patients with high intracoronary lactate, which signifies that predominant cause of hyperlactatemia was systemic hypoperfusion rather than local increase in lactate levels.

1. Introduction

Coronary artery disease (CAD) is currently the most common, noncommunicable disease in India, affecting over 65 million people. CAD is a major cause of morbidity and mortality in India. Approximately, 8–10% of Indians in urban areas and 3–4% in rural areas have CAD.[1] CAD tends to occur at a younger age in Indians, with more extensive angiographic involvement contributed by genetic, metabolic, conventional and nonconventional risk factors.[2,3]

Primary percutaneous coronary intervention (PPCI) is the treatment of choice in ST-elevation myocardial infarction (STEMI), as in comparison with thrombolysis, PPCI guarantees a higher rate of reperfusion. However, in patients presenting late it does not

☆ All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

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prevent complete myocardial necrosis, which is related to the duration of occlusion, particularly in higher-risk patients.

The present study attempts to describe the correlation between arterial and intracoronary blood values (pH, bicarbonate, lactate levels, partial pressures of carbon dioxide (pCO_2) and oxygen (pO_2) in patients of STEMI undergoing PPCI and also whether these parameters correlate with clinical data and short-term outcome of the patients.

2. Methods

2.1. Study design

We performed a single-centre, prospective study over a period of 12 months conducted in Department of Cardiology, Dayanand Medical College and Hospital, Unit-Hero DMC Heart Institute, Ludhiana. All patients of STEMI presenting in emergency and undergoing PPCI and in whom adequate intracoronary sample could be obtained were included in the study.

2.2. Study population

Patients with STEMI who were referred for PPCI within 12 h after the onset of symptoms, in whom adequate intracoronary arterial sample could be obtained were eligible to participate in the trial. All patients provided written informed consent. Patients with chronic kidney disease, chronic liver disease and patients who had received fibrinolytic therapy were not eligible.

2.3. Procedure and laboratory methods

Patients underwent the procedure according to the operator's usual technique. After cannulating the involved coronary artery by guiding catheter, the lesion was crossed with a guidewire and the Export (Medtronic) aspiration catheter was advanced and suction started before it crossed the lesion. The first intracoronary blood suctioned was sent for arterial blood gas (ABG) analysis. It was made sure that the guide catheter be fully engaged with the coronary ostium during removal of the aspiration catheter to avoid embolizing thrombus to the systemic vasculature. The percutaneous coronary intervention procedure was performed after aspiration was completed. At the very same time, blood from peripheral artery was also sent for ABG analysis. ABG analysis was performed by using instrument, GEM Premier 3000. Heparinized 2 ml of Intracoronary and peripheral blood sample was used for analysis. Parameters analysed included pH, lactate, bicarbonate levels and partial pressures of carbon dioxide (pCO_2) and oxygen (pO_2).

2.4. Study outcomes

Intracoronary and peripheral ABG samples were compared among themselves for analysed parameters and in addition, intracoronary blood parameters were also correlated with clinical data, investigational data and short-term (30-day) outcome.

2.5. Statistical analysis

Categorical variables were presented as numbers and percentage, continuous variables as mean ± standard deviation (SD) or median and 95% confidence interval (95% CI). Comparisons were made using the Student's *t*-test for numerical parameters and the chi-squared test for categorical data where appropriate. Categorical variables were compared by chi-squared analysis. Continuous variables were compared by Student's *t*-test. A *p*-value of <0.05 was considered to be statistically significant. All the data were recorded on Microsoft Excel database and statistical analysis was performed using SPSS software (version 23.0, SPSS Inc., Chicago, Illinois, USA).

3. Results

The study involved a total of 27 STEMI patients. The baseline characteristics of the study population are listed in Table 1. The average symptom duration to door time was 162 min and average door to balloon time was 27.5 min. There were 4 patients with Killip class ≥II. Anterior infarct was seen in 77% and 23% had inferior infarction. Intra-aortic balloon pump was used in single patient (3.7%). Coronary angiogram revealed single-vessel disease in 66% of subjects. Double-vessel disease was present in 26% of subjects and 8% had triple-vessel disease. Diabetes and hypertension were seen in 26% of subjects. Dyslipidaemia was present in 66%, 22% were obese and 11% were smokers. No patient in our study population died or had 30-day readmission or revascularization.

Serum markers of metabolic acidosis are often used as a part of critical care assessment to determine the severity of injury and provide prognostic information. The most common of these markers include lactate, bicarbonate and pH. On comparing intracoronary and peripheral blood (Table 2), our results revealed nearly correlating pH (95% CI −0.01 to 0.02; $p = 0.44$), lactate (95% CI 0.03–0.1; $p = 0.28$), bicarbonate (95% CI 0.6–1.5; $p = 0.64$), pCO_2 (95% CI 1.1–2.4; $p = 0.79$) and pO_2 (95% CI 3.2–47.5; $p = 0.06$).

Intracoronary lactate levels (Table 3) were found to be significantly higher in patients with more symptom onset to door time ($p = 0.025$) (Fig. 1). Systolic blood pressure (SBP; $p = 0.03$) was also significantly lower in subjects with increasing lactate levels. Hyperlactatemia did not show association with estimated

Table 1
Baseline characteristics and key time intervals ($N = 27$).

Parameter	Total ($N = 27$)
Age (y)	54.8 ± 9.4
Males (*n* and %)	21 (77%)
Symptom duration to door (min)	162 ± 72
Door to balloon (min)	27.5 ± 9
Killip class ≥ II (*n* and %)	4 (15%)
HR (bpm)	82 ± 15
SBP (mm Hg)	125 ± 15
DBP (mm Hg)	76 ± 11
BMI (kg/m^2)	27.2 ± 4.8
Infarct location	
Anterior (*n* and %)	21 (77.7%)
Inferior (*n* and %)	6 (22.3%)
Vessel involved	
LAD	21 (77.7%)
LCX	1 (3.7%)
RCA	5 (18.5%)
Haemoglobin (gm%)	14 ± 1.9
Urea (mg/dl)	27.7 ± 6.7
Creatinine (mmol/l)	0.85 ± 0.16
eGFR (ml/min per 1.73 m^2)	
SGOT (IU/l)	87 ± 116
SGPT (IU/l)	37 ± 18
Lipid profile	
TC (mg/dl)	206 ± 57
LDL (mg/dl)	143 ± 50
TG (mg/dl)	177 ± 93
HDL (mg/dl)	38 ± 8
LVEF (%)	38 ± 4

All values are presented as mean ± SD or number (%).
SD, standard deviation; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery; eGFR, estimated glomerular filtration rate; SGOT, serum glutamic–oxaloacetic transaminase; SGPT, serum alanine aminotransferase; TC, total cholesterol; LDL, low-density lipoprotein; TG, triglycerides; HDL: high-density lipoprotein; LVEF, left ventricle ejection fraction.

Table 2
Comparison of intracoronary and peripheral blood.

Variable	Intracoronary blood (mean ± SD)	Peripheral blood (mean ± SD)	95% CI	*p*-Value
pH	7.37 ± 0.05	7.37 ± 0.06	−0.01 to 0.02	0.44
Lactate (meq/l)	1.63 ± 0.79	1.73 ± 1.16	0.03–0.1	0.28
Bicarbonate (meq/l)	21.60 ± 3.0	21.42 ± 3.1	0.6–1.5	0.64
pCO_2 (mm Hg)	37.26 ± 5.7	37.48 ± 6.4	1.1–2.4	0.79
pO_2 (mm Hg)	117.04 ± 39.3	140.85 ± 64.3	3.2–47.5	0.06

CI, confidence interval; SD, standard deviation; pCO_2, partial pressure of CO_2; pO_2, partial pressure of O_2.

Table 3
Parameters according to intracoronary lactate levels.

	Intracoronary lactate		
	≤2 meq/l ($n = 20$)	>2 meq/l ($n = 7$)	*p*-Value
Symptom to door time (min)	140 ± 58	224 ± 75	0.025
SBP (mm Hg)	128 ± 13	114 ± 17	0.03
DBP (mm Hg)	79 ± 9	70 ± 11	0.08
Killip class			
I	18	5	0.38
≥II	2	2	
LVEF (%)	38 ± 4	37 ± 2	0.56
eGFR (ml/min per 1.73 m^2)	104 ± 26	105 ± 32	0.37

SBP, systolic blood pressure; DBP, diastolic blood pressure; LVEF, left ventricle ejection fraction; eGFR, estimated glomerular filtration rate.

glomerular filtration rate (eGFR; $p = 0.93$), Killip class ($p = 0.38$) or left ventricular ejection fraction (LVEF; $p = 0.56$). As there was no mortality or short-term readmission or revascularization, the significance of these parameters could not be assessed. Intracoronary pH showed no association with symptom onset to door time, systolic blood pressure, eGFR, Killip class or LVEF.

4. Discussion

Limited information is available on acid–base disturbances in the course of myocardial infarction; and at present, there is no study to the best of knowledge have compared the intracoronary and peripheral blood in patients of STEMI undergoing PPCI.

The hypothesis was that because of stasis of blood in culprit intracoronary artery, it has lower pH and bicarbonate, more deoxygenated blood and higher lactate levels, and whether it will help to provide more clinical information and prognostication than the information given solely by peripheral blood.

The acid–base balance provides clinical information and is useful for in-hospital risk stratification in STEMI patients because the detection of acidosis characterizes patients with a more severe myocardial dysfunction who therefore have higher morbidity and mortality.

In our study, it was seen that the blood from culprit intracoronary vessel was no different when compared to peripheral blood and none of the parameters of the two samples, like pH, bicarbonate, lactate, partial pressures of carbon dioxide and oxygen reached statistical significance. Because of absence of literature on intracoronary blood in STEMI, our findings could not be correlated with any of the previous studies.

Intracoronary hyperlactatemia was present in patients presenting with higher symptom onset to door times, also these patients had lower systolic blood pressure on presentation. However, pH of intracoronary blood, when compared with clinical and investigational parameters, was not significant with any parameter.

The prognostic significance of lactate in acute coronary syndrome has been investigated in observational, mainly single-centre studies.[4,5] Lactate concentrations, measured in the early phase of STEMI are influenced by the degree of hemodynamic impairment (as indicated by Killip class) and myocardial ischemia (as inferred by Troponin I). In 1176 STEMI patients, hyperlactatemia measured at arrival in the catheterization laboratory was associated with worse outcome measures[6] (increased 30-d mortality, larger enzymatic infarct size and increased use of intra-aortic balloon pump]. In 807 STEMI patients treated with percutaneous coronary intervention, lactate values were independently associated with early mortality only in the subgroup of patients in advanced Killip class.[7]

MacKenzie et al[8] observed that patients with cardiogenic shock complicating myocardial infarction show significant metabolic acidosis and a poor prognosis. Pilcher and Nagle[9] documented that

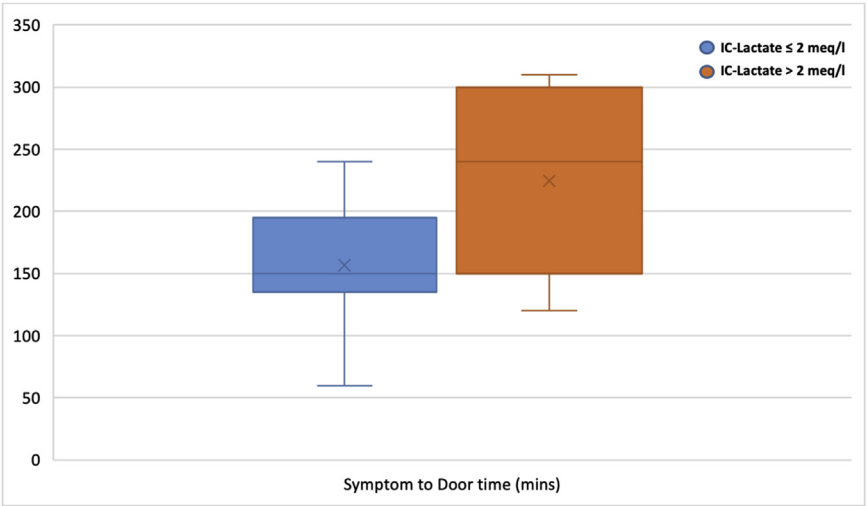

Fig. 1. Boxplot showing relationship of IC-lactate levels and symptom onset to door time. IC, intracoronary.

there was no relationship between acid–base status (assessed by pH) and ventricular dysrhythmias in myocardial infarction without cardiogenic shock. In both these studies, patients with an acute myocardial infarction were medically treated. In a small study by Gandhi and Akholkar[10], on 50 patients of STEMI and the use of thrombolytic therapy, mortality in patients with pH level <7.35 was 60% in those with pH level more than 7.45 was 11.11% ($p = 0.03$). In above studies, parameters of peripheral blood gas analysis were used, but our study used intracoronary blood.

Results of our small, single-centre study showed that no extra prognostic information can be obtained from intracoronary blood when compared with peripheral blood. Hyperlactatemia, however, was more in patients presenting late after symptom onset, and systolic blood pressure on presentation was also significantly lower in such patients.

In-hospital mortality and short-term outcome could not be evaluated as an outcome because none of our patients had in-hospital mortality or had 30-day readmission or revascularization. Although this finding is not surprising as our study population was small, it would be interesting to evaluate in a larger cohort of patients, where intracoronary blood analysis might hold a prognostic role.

We acknowledge several limitations in our study. First, this study comprises small sample size. Second, patients in whom adequate intracoronary sample could not be obtained were excluded from the study, so not all STEMI patients were included. Third, for the analysis of intracoronary blood, crossing the occluded coronary artery with a guidewire may result in partial restoration of epicardial blood flow. This may result in changes of the local metabolites and thus the results. Fourth, in-hospital mortality and short-term (30-day) outcome could not be evaluated as an outcome because of the absence of mortality and readmission or revascularization in our population.

5. Conclusions

We did this study with the premise that parameters like acidosis, hyperlactatemia and hypoxia will be more in the intracoronary blood of the culprit vessel as compared with peripheral blood in patients undergoing PPCI for STEMI. The results of our study showed that the evaluation of intracoronary blood provides no additional information regarding the prognosis and short-term (30-day) outcome of the patients when compared with peripheral blood. However, there was a significant intracoronary hyperlactatemia in patients presenting late after symptom onset. SBP was also significantly less in patients with high intracoronary lactate, which signifies that predominant cause of hyperlactatemia is systemic hypoperfusion rather than local increase in lactate levels.

Declaration of competing interest

All authors have none to declare

What is already known?

Peripheral blood lactate levels are influenced by the degree of hemodynamic impairment (as indicated by Killip class) and myocardial ischemia (as inferred by Troponin I), and is independent predictor of mortality.

What this study adds?

- The evaluation of intracoronary blood in STEMI provides no additional information regarding the prognosis of the patients when compared with peripheral blood.
- Intracoronary hyperlactatemia is more in patients presenting late after symptom onset.
- The predominant cause of hyperlactatemia is systemic hypoperfusion rather than local rise.

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