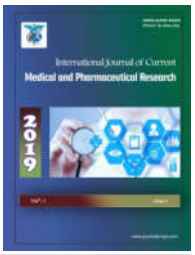
Research Article

INTERNATIONAL JOURNAL OF CURRENT MEDICAL AND PHARMACEUTICAL RESEARCH

ISSN: 2395-6429, Impact Factor: 4.656
Available Online at www.journalcmpr.com
Volume 5; Issue 01(A); January 2019; Page No. 3991-3993
DOI: http://dx.doi.org/10.24327/23956429.ijcmpr201901606



METABOLIC ACIDOSIS: A RISK IN ACUTE MYOCARDIAL INFARCTION

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ARTICLE INFO

Article History:
Received 13th October, 2018
Received in revised form 11th November, 2018
Accepted 8th December, 2018
Published online 28th January, 2019

Key words:
Myocardial infarction, metabolic acidosis, serum bicarbonate.

ABSTRACT

Background: Metabolic changes are known to occur in early stages of an acute myocardial infarction. It is rarely severe and could contribute to the development of dysrhythmias. The objective of the present study was to ascertain the acid base status in patients of acute myocardial infarction admitted in intensive care unit.
Method: 50 patient of acute myocardial infarction diagnosed by Physician & confirmed by the Physician were selected and investigated by collection of blood sample for arterial blood gas analysis. Informed written consent was taken from each patient in their known language.
Results: Metabolic acidosis was seen in majority of cases i.e. P^H level < 7.35 was 50 % (3 out of 6), while pH level 7.35 to 7.45 was 19.4(7 out of 36) and pH level > 7.45 was 12.5% (1 out of 8) & P value was significant (0.3). Whereas mortality in patients with bicarbonate level less than 22meq/lit seen in 33.33% while in bicarbonate level 22-26meq/lit was 20.58% bicarbonate level > 26meq/lit was 14.28% and showed significant p value (0.13).
Conclusion: There is a definite metabolic disturbance i.e. in acid-base balance early stages of AMI as metabolic acidosis.

INTRODUCTION

A change in pH causes gain or loss of protons by proteins and alters charges distribution on them and hence, changes molecular conformation, structure & function. Therefore, strict regulations of pH and acid-base homeostasis are crucial for normal cell, tissue and organ performance. Acid Base disorders are very commonly encountered in critically ill patients [1].

Few data are available on the acid-base imbalance in acute myocardial infarction (AMI) submitted to coronary revascularization, and earlier studies on this topic differ with respect to patients' selection criteria, treatment and evaluated parameters. Anion gap acidosis on admission is a powerful predictor of short-term mortality, independent of other biochemical, historical and electrocardiographic data available at the time of admission [2]. In AMI, the combination of a fall in cardiac output and arterial hypoxemia leads to tissue hypoxia, metabolic acidosis and fall in plasma bicarbonate due to rise in lactic acid. Elevation in carbon dioxide not only increases the acidosis but also reduces the arterial oxygen tension that is particularly a dangerous combination. The dominant mechanism for the reduction of contractility with acidosis is competitive inhibition of the slow calcium current by hydrogen ions. The slow calcium current initiates cardiac contraction and contributes to the action potential. The major effect of acidosis on the action potential of ventricular muscle is to cause a lengthening owing to inhibition of potassium exchange and as a consequence of reduced calcium current. The reduced contractility is dependent upon the severity of the acidosis.

Acid-base disorders are diagnosed from stepwise interpretation of reports of Arterial Blood Gas (ABG) analysis [3]. The disorder may be single or primary or may be mixed acid-base disorder. An ABG is one of the main tools for decision making in patients with dyspnea [4].

MATERIALS & METHOD

This is a descriptive study carried out in wards of Department of Medicine from June 2015 to December 2017 at Government Medical College & Hospital , Aurangabad, Maharashtra, India. Clearance from Ethical Committee of the institution was obtained.

By finding confidence of interval, fifty patients of AMI clinically diagnosed by ECG and enzyme biomarkers were included in this study.

Blood samples were collected from radial artery or femoral artery. Blood gas analysis was done by Cobas b 121 system blood gas analyser (Roche). Analyser contains the probe for testing of the sample. The values of PO_2, PCO_2, pH, HCO_3- were studied.

Inclusion Criteria

50 patients of AMI clinically diagnosed by physician and confirmed by ECG and enzyme biomarkers were selected for this study.

Exclusion Criteria

Patients with anaemia, significant hepatic, renal and pulmonary disease, diabetes mellitus, infection, hypo and hyperthyroidism were excluded from this study.

Statistical analysis

The observed clinical outcome was analysed by chi square test 'p' value less than 0.05 was taken as statistically significant.

RESULTS

In total study population of 50 patients, 62% (n=31) were male and 38% (n=19) were female. Normal pH ranges from 7.35 - 7.45

Table I shows distribution of study subjects as per pH level. Majority of subjects (n=36) are in pH range of 7.35 - 7.45 that consists 72% of the study subjects. 12% of study subjects have pH < 7.35 and 16 % of study subjects had pH > 7.45. In this study mean pH of patients who survived (n=40) was 7.40 ± 0.56 & who died (n=11) was 7.32 ± 0.152 P = value was 0.03, which is statistically significant.

Table I Subjects as per pH level

PH	Total No. n=50	Male	Female
<7.35	6	4	2
7.35 - 7.45	36	22	14
>7.45	8	5	3

Table 2 shows mortality according to pH level. Mortality in patients with pH level <7.35 was 50% (3 out of 6) .While in pH level 7.35-7.45 was 20.59% (7 out of 34) and mortality in pH level more than 7.45 was 12.5% (1 out of 8) ,p value was significant (p=0.031) . It is evident from the data that as pH value decreases the mortality rate increases (12.5%→20.59%→50%→ 100%). Two subjects in our study group had pH level <7.2 and both these subjects didn't survive, mortality was 100%.

Table 2 Mortality as per pH level

PH	Total patients n=50	Mortality	Male	Female
<7.2	2	2	1	1
<7.35	6	3	2	1
7.35 - 7.45	34	7	5	2
>7.45	8	1	1	0

Table 3 shows distribution of study subjects according to bicarbonate level. Majority of patients 66% (n=34) had bicarbonate level in the normal range of 22-26Meq/lit 24% (n=12) patients had bicarbonate < 22meq/lit. 10% (n=5) had bicarbonate level > 26Meq/lit.

Table 3 Subjects as per HCO3 level

HCO3 level Meq/L	Total n=50	Male	Female
<22	12	8	4
22 - 26	33	23	10
>26	5	3	2

Table 4 Shows distribution of mortality according to bicarbonate level. Normal bicarbonate level is 22-26 meq/dl. Mortality in patients with bicarbonate level less than 22 was 33.33 %, while in those with level 22-26 meq/l was 20.58% and in those with level >26 was 14.28%. Thus there is higher rate of mortality in patients with bicarbonate level <22 meq/l (p value= 0.12).

Out of 8 subjects with bicarbonate level <22meq/l, 3 subjects in this study had bicarbonate level <20 and all 3 subjects didn't survive. So mortality was 100% in patients level <20 meq /litre.

Table 4 Mortality as per HCO3 level

HCO3 level Meq/L	Total No. n=50	Mortality	Male	Female
<20	3	3	2	1
<22	8	3	2	1
22 - 26	32	7	5	2
>22	7	1	1	0

DISCUSSION

In this study, we hypothesized that metabolic acidosis might be associated with mortality in the initial phase in AMI. Acidosis detecting in AMI in the initial stage is very crucial. Significant metabolic acidosis as primary abnormality as indicated by pH < 7.35 and serum bicarbonate < 22mEq/dl. Injury to myocardium in AMI is responsible for lock of vascular resistance or maintenance. Metabolic acidosis itself can cause myocardial depression and blunts the pressure response to adrenaline and noradrenalin, both reversed by correction of metabolic acidosis.

Mackenzie GJ et al studied 15 patients with one day old myocardial and the metabolic profile. Patients with uncompleted myocardial infarction had only mild hypoxemia and complete lack of metabolic disturbances [5].

In the study of 50 patients by Neaverson et al 64% were males and 36% were female. Out of 50 cases, 33 had significant base deficit, 15 were within normal range & 2 were alkalotic of the 33 acidotic cases, 13 had low PH > 7.35, among which 12 died. Similarly in this study also higher mortality was seen in AMI patients who had metabolic acidosis [6].

Kirby HJ studied 123 patients of AMI diagnosed by ECG & cardiac enzymes. ABG was done on their patients. They found 58% patients had low bicarbonate level and 22% patients showed fall in PH. However correlation of acidosis had no effect on mortality [7].

Anderson R, et al studied the relationship between metabolic acidosis & cardiac arrhythmia in 21 patients with AMI. They found that metabolic acidosis occurs in early stages of AMI [8].

In similar study by Lazzeri et alfound that acidosis was present in 4.2% and HCO_3<22meq/l in 24% patients [9]. They concluded that evaluation of existence of acidosis in early phase provides bedside clinicians with useful tools for early risk stratification and base excess proved independent marker for initial complications in infarction [10] [11]. Below certain pH level, the protective stimulant to cardiac depression by

acidosis i.e. catecholamine release fails [12] [13]. Similarly in our study we considered mortality in patients during hospitalization and it showed trend in higher mortality in patient who had metabolic acidosis. The dominant mechanism for the reduction of contractility with acidosis is competitive inhibition of the slow calcium current by hydrogen ions. The slow calcium current initiates cardiac contraction and contributes to the action potential. The major effect of acidosis on the action potential of ventricular muscle is to cause a lengthening owing to inhibition of potassium exchange and as a consequence of reduced calcium current. The reduced contractility is dependent upon the severity of the acidosis [14]. The acid-base balance provides clinical information, useful for in-hospital risk stratification even in uncomplicated AMI patients [15].

CONCLUSION

As the blood pH was decreasing mortality was increasing, definite correlation was found between metabolic acidosis & mortality in acute myocardial infarction . Thus study of Arterial Blood Gas analysis in the initial stage of onset of infarction help as a marker to assess the acid base imbalance.

Recommendation

From the finding of this study we can recommend that the basic metabolic profile should be done in all patients with AMI as a part of the routine laboratory assessment for correction of acidosis and incidence of early complications of AMI if present.

Acknowledgement

We acknowledge Dr Tambse & Dr Mehraj, Lecturers, Department of Biochemistry, GMC, Jalgaon for their valuable suggestions & discussion while preparing this manuscript.

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How to cite this article:

Varghese M.V *et al* (2019) 'Metabolic Acidosis: A Risk in Acute Myocardial Infarction', *International Journal of Current Medical And Pharmaceutical Research,* 05(01), pp. 3991-3993.

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