International Journal of Advances in Medicine
*Gandhi AA et al. Int J Adv Med. 2015 Aug;2(3):260-263*
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20150556

**Research Article**

# Metabolic acidosis in acute myocardial infarction

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**Received:** 11 July 2015, **Revised:** 13 July 2015
**Accepted:** 19 July 2015

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## ABSTRACT

**Background:** Metabolic acidosis is known to occur in the early stages of an acute myocardial infarction but it is rarely severe except in the presence of profound cardiogenic shock; nevertheless, it could contribute to the development of dysrhythmias. The objective of present study was to ascertain the acid base status of patient of acute STEMI at the time of hospitalization.
**Methods:** Fifty patients of acute myocardial infarction were interrogated for history of presenting illness examined for vitals systemic examination and investigated by collection of blood sample and ECG.
**Results:** Mortality in patients with pH level <7.35 was 60% (3 out of 5**)**. While in pH level of 7.35-7.45 was 16.6% (6 out of 36) and mortality in pH level more than 7.45 was 11.11% (2 out of 9) p value was significant (0.03). Mortality in patients with bicarbonate level less than 22 meq/l was 40 %, while in those with level 22-26 meq/l was 17.14% and in those with level >26 meq/l was 20%. Thus there is higher rate of mortality in patients with bicarbonate level <22 meq/l (p value 0.12).
**Conclusions:** The definite correlation was found between metabolic acidosis and mortality (p value was 0.03). As the blood pH was decreasing mortality was increasing and 100% mortality was found in pH level <7.3.

**Keywords:** Myocardial infarction, Metabolic acidosis, Serum bicarbonate

## INTRODUCTION

In AMI (acute myocardial infarction), 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. Metabolic acidosis is compensated by hyperventilation. Those who are not able to compensate the metabolic disturbances by respiration are at risk of higher mortality. Elevation in carbon dioxide not only increases the acidosis but also reduces the arterial oxygen tension that is particularly a dangerous combination. Corrections of metabolic acidosis and respiratory compensation have showed different effect on prognosis of patient in different studies. Various rhythm disturbances which are even refractory to electrical cardioversion are found to be spontaneously responding to correction of metabolic acidosis.

## METHODS

Fifty patients of acute myocardial infarction were examined and investigated during their admission in hospital. History of presenting illness Systemic examination and vitals were noted. ECG and collection of blood sample were done for laboratory investigations including ABG, blood urea, serum creatinine, serum electrolytes, blood sugar, total protein A:G.Treatment of the patients was initiated with standard anti ischaemic therapy and serial ECG of the patient were done.

Blood gas analysis was done by Cobas b 121 system blood gas analyzer (Roche). Blood sample were collected

from radial artery of non dominant arm or from femoral artery. Analyzer contains the probe for testing of the sample. The valuation of the $PO_2$, $PCO_2$, pH, $HCO_3^-$ was done by comparison of the value with the individual control for the each of the above parameter.

***Inclusion Criteria***

Fifty patients of STEMI as diagnosed clinically, by ECG and by biomarkers. All patients of AMI were medicated with angiotensin converting enzyme inhibitors or angiotensin receptor blockers over and above the standard thrombolytic, anti-ischaemic and antiplatelet therapy.

***Exclusion Criteria***

Anemia, significant hepatic, renal and pulmonary disease, diabetes mellitus, infection, hypo and hyperthyroidism.

***Statistical Analysis***

The observed clinical outcome was analysed by Chi square test. P value of less than 0.05 was taken as statistically significant.

## RESULTS

In total study population of 50 patients 64% (n=32) were male and 36% (n=18) were female. Normal pH ranges from 7.35-7.45. Table 1 shows the distribution of study subjects as per pH level. Majority of subjects (n=36) are in the pH range of 7.35-7.45 that consists 72% of the study subjects. 10% of study subjects have pH <7.35 and 18% of study subjects had pH >7.45. In our study, mean pH of STEMI patients who survived (n=39) was 7.40±0.057 & who died (n=11) was 7.32±0.15. p value was 0.03. It was statistically significant.

**Table 1: Subjects as per the pH level (male and female).**

| pH | Total Patients | Male | Female |
|---|---|---|---|
| <7.35 | 5 | 3 | 2 |
| 7.35-7.45 | 36 | 23 | 13 |
| >7.45 | 9 | 6 | 3 |

**Table 2: Mortality as per pH level.**

| pH level | Total Patients | Mortality | Male (Mortality) | Female (Mortality) |
|---|---|---|---|---|
| <7.35 | 5 | 3 | 2 | 1 |
| 7.35-7.45 | 36 | 6 | 4 | 2 |
| >7.45 | 9 | 2 | 1 | 1 |

Table 2 shows mortality according to pH level. Mortality in patients with pH level <7.35 was 60% (3 out of 5). While in pH level of 7.35-7.45 was 16.6% (6 out of 36) and mortality in pH level more than 7.45 was 11.11% (2 out of 9). p value was significant (p=0.03).

It is evident from the data that as pH value decreases, the mortality increases (11.11% → 16.6% → 60%). Three subjects in our study group had pH level <7.3 and all these subjects didn't survive, mortality was 100%.

**Table 3: Subjects according to bicarbonate level.**

| $HCO_3$ level (Meq/L) | Total | Male | Female |
|---|---|---|---|
| <22 | 10 | 7 | 3 |
| 22-26 | 35 | 22 | 13 |
| >26 | 5 | 3 | 2 |

Table 3 shows distribution of study subjects according to bicarbonate level. Majority of patients 70% (n=35) had bicarbonate level in the normal range of 22-26 meq/l. 20% (n=10) patients had bicarbonate <22 meq/l. 10% (n=5) had bicarbonate level >26 meq/l.

**Table 4: Mortality as per bicarbonate level.**

| $HCO_3$ level (Meq/L) | Total | Mortality | Male | Female |
|---|---|---|---|---|
| <22 | 10 | 4 | 2 | 2 |
| 22-26 | 35 | 6 | 4 | 2 |
| >26 | 5 | 1 | 1 | 0 |

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 40%, while in those with level 22-26 meq/l was 17.14% and in those with level >26 was 20%. Thus there is higher rate of mortality in patients with bicarbonate level <22 meq/l (p value= 0.12).

Out of 10 subjects with bicarbonate level <22meq/l, 3 subjects had bicarbonate level <15 and all 3 subjects didn't survive. So mortality was 100% in patients with level <15 meq/l.

## DISCUSSION

Acidosis in a patient of STEMI at the time of presentation is of ominous significance. Significant metabolic acidosis as primary abnormality as indicated by pH <7.35 and Serum bicarbonate <22 meq/dl was present in 5 patients. Out of which 3 patients died (p value=0.03) and 3 out of 5 patients had developed ventricular arrhythmias. Out of which 3 patients which died 2 had developed ventricular arrhythmias.

Change in the systemic vascular resistance is important for maintenance of cardiac output in the patient with AMI. Injury to myocardium is responsible for lack of vasoconstriction by neurogenic and humoral influences but severe acidosis and hypoxia are equally important

factors in the impairment of this fundamental reflex. Metabolic acidosis itself can cause myocardial depression and blunts the pressure response to adrenaline and noradrenaline, both reversed-by correction of metabolic acidosis.

Mackenzie GJ et al[1] studied the 15 patients with one day old myocardial infarction and their metabolic profile, arterial blood gas tension and effect of oxygen therapy. Patients with cardiogenic shock had very low cardiac output, and inadequate compensatory rise in systemic vascular resistance and severely impaired stroke volume. Patients with uncomplicated myocardial infarction had only mild hypoxemia and complete lack of metabolic disturbances. Patients with cardiogenic shock had considerable degree of arterial blood hypoxemia as well as metabolic acidosis, lactic acidosis and hyperglycemia. Associated hypoxemia did not significantly improve with oxygen therapy. Impairment of response was shown to be due to shunting of around 25% of the cardiac output through the vessels which are inaccessible to pulmonary gas exchange.

In the study of 50 patients by Neaverson et al[2] 64% (n=32) were males & 36% (n=18) were females. 12 were selected for intensive therapy unit treatment of whom seven died (14%). Of the 50 cases they investigated 33 cases who had a significant base deficit, 15 were within the normal range & 2 were alkalotic. Of the 33 acidotic cases, 13 had an abnormality low pH of less than 7.35. After conclusion of 28 days the patients were divided into those who were alive with complication (congestive cardiac failure) and those who died only 1 of the 17 non acidotic patients died, where 12 of the 33 acidotic patient died the death rate was greater with ($P<0.05$) an abnormality low pH. Similarly, we also found a trend towards higher mortality in our study amongst patients who had metabolic acidosis.

Kirby BJ[3], studied the 123 patients of acute myocardial infarction diagnosed by history ECG and cardiac enzymes. Arterial blood gas analyses were done on these patients. They found fall in plasma bicarbonate in 58% of the patients was compensated in majority of patients and fall in pH was found in 22% of the case. They did not find any relation plasma bicarbonate and arterial oxygen tension. Metabolic changes were profound in the patients with hypotension and left ventricular failure, and were associated with very high mortality (85%). Correlation of acidosis had no effect on mortality.

Anderson R et al[4] studied the relationship between metabolic acidosis and cardiac arrhythmia in 21 patients with clinical ECG and biochemical evidence of acute myocardial infarction. They found that metabolic acidosis occurs in early stages of acute myocardial infarction but it is rarely severe except in the case of cardiogenic shock. Still it could contribute to development of dysarrhythmia. Experimental and clinical situations have been described where extreme metabolic acidosis has been associated with a tendency to develop arrhythmias.

In the similar study by Lazzeri et al[5] which was most similar study with us (only difference that they considered in patients who had undergone primary angioplasty instead of thrombolysis) they assessed 445 patients with STEMI submitted to primary percutaneous coronary intervention in identifying the patients at higher risk for in hospital complications (e.g. acute pulmonary edema & arrhythmias) they found that acidosis was present in 4.2% (n=11) patients. $HCO_3$ <22 meq/l in 24% (n=62) patients. They concluded that evaluation of base excess and lactate i.e. evaluation of existence of acidosis in the early phase of STEMI provides the bedside clinicians with useful tools for early risk stratification and base excess proved to be independent marker for intra ICCU complication and thus acidosis in STEMI patients even who underwent primary coronary angioplasty was associated with higher rate of complications. Similarly in our study we considered mortality in patients during hospitalization and it showed trend in higher mortality in patient who had metabolic acidosis.

Acidosis is a cardiac depressant but a stimulant to catecholamine release in its own right.[6] Catecholamine release protects the heart from acidotic depression[7], but below a certain pH level this protection probably fails.[8]

## CONCLUSIONS

The definite correlation was found between metabolic acidosis and mortality (p value was 0.03). As the blood pH was decreasing mortality was increasing and 100% mortality was found in pH level <7.3.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

## REFERENCES

1. MacKenzie GJ, Taylor SH, Flenley DC, McDonald AH, Staunton HP, Donald KW. Circulatory and respiratory studies in myocardial infarction and cardiogenic shock. Lancet 1964;2:825.
2. Neaverson MA. Metabolic acidosis in acute myocardial infarction. BMJ 1966;2:383.
3. Kirby BJ, McNicol MW. Acid-base status in acute myocardial infarction. Lancet 1966;2:1054.
4. Anderson R, Gardner FV, Honey H, Noble M, Woodgate DW. Relation between metabolic acidosis and cardiac dysrhythmias in acute myocardial infarction. British Heart Journal 1968;30:493.
5. Lazzeri C, Valente S, Chiostri M, Picariello C, Gensini GF. Acid-base imbalance in uncomplicated ST-elevation myocardial infarction: the clinical role of tissue acidosis. Intern Emerg Med. 2010;5:61-6.
6. Ng ML, Levy MN, Zieske HA. Effects of changes of pH and of carbon dioxide tension on left

ventricular performance. Am J Physiol. 1967 Jul;213(1):115-20.
7. MaIm JR, Manger WM, Sullivan SF, Papper EM, Nahas GG. The effect of acidosis on sympatho-adrenal stimulation; particular reference to cardiovascular bypass. Journal of the American Medical Association 1966;197:121.
8. Rocamora JM, Downing SE. Preservation of ventricular function by adrenergic influences during metabolic acidosis in the cat. Circulation Research 1969;24:373.

**Cite this article as:** Gandhi AA, Akholkar PJ. Metabolic acidosis in acute myocardial infarction. Int J Adv Med 2015;2:260-3.