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METABOLIC ACIDOSIS: A RISKFACTOR INACUTE KIDNEY INJURY

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Abstract:

Background: Metabolic acidosis has been proved to be a risk factor for the progression of chronic kidney disease, but its relation to acute kidney injury (AKI) has not been investigated. The objective of this study was to ascertain the acid base balance status of patients of acute kidney disease patients.

Methods: Fifty patients those who developed acute kidney disease during hospitalization in government medical college, Aurangabad were included in this study.

Results: Mortality in patients with pH level <7.35 was 50% (3 out of 5**)**. While in pH level of 7.35-7.45 was 29.4% (10 out of 34) and mortality in pH level more than 7.45 was 20% (2 out of 10) p value was significant (0.031). Mortality in patients with bicarbonate level less than 22 meq/l was 50 %, while in those with bicarbonate level 22-26 meq/l was 33.3 % and in those with level >26 meq/l was 25%. Thus there is higher rate of mortality in patients with bicarbonate level <22 meq/l (p value 0.11).

Conclusion: The results show that acidosis especially metabolic acidosis with decreased bicarbonate levels were contributors to the incidence of AKI and found that they can be the predictors of hospital mortality. As the blood pH level decreases <7.3 and bicarbonate level<22 meq/l mortality was increasing and 100% mortality was found below pH 7.2 and bicarbonate below 20 meq/l.

Key words: Acute kidney injury, Acid –base, Metabolic acidosis, serum bicarbonate.

INTRODUCTION

The kidney is a principally responsible organ for retention and excretion of electrolytes and maintaining acid-base homeostasis in healthy individuals **[1].** Arterial blood gases (ABG) results reflect underlying pathology and interpretation of the results which are often compounded by ongoing disease processes and clinical interventions. Recent studies showed that metabolic acidosis is associated with high mortality and increased the length of stay in the hospital and ICU **[2],[3].** An arterial blood gas result can help in the assessment of a patient's gas exchange, ventilator control and acid–base balance. The diagnosis of metabolic acidosis is based on arterial blood gas (ABG) . The components of an ABG analysis are PaO2, SaO2, hydrogen ion concentration (pH), PaCO2, HCO3, base excess, and serum levels of hemoglobin, lactate, glucose and electrolytes (sodium, potassium, calcium, and chloride). A recent experimental study proved that metabolic acidosis exacerbates AKI. However, limited data exists

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about the harmful effect of metabolic acidosis on the development of AKI **[4].** Corrections of metabolic acidosis have showed different effect on prognosis of patient in different studies. This study was aimed at studying the acid-base status at admission which was associated with the occurrence of renal dysfunction and hospital mortality.

$P^{H:}$ A low pH indicates acedemia while a high pH indicates alkalemia.

The parameters most frequentlyused—PaO2, SaO2, pH, PaCO2, HCO3_, and lactate—often are adequate in diagnosing and managing mostclinical situations. To quantify the Concentration in blood, a simplifiedmathematical expression, called pH, is used. In health, the normal range for pH is 7.35–7.45 . PH is a negative logarithm, which means that the higher the H+ concentration, the lower the pH and vice versa.

Maintaining acid–base balance: The three systems that regulate the acid–base balance are the buffer system (metabolic), kidneys (metabolic) and the lungs (respiratory) **[1].** The lungs regulate carbon dioxide (CO2) and the renal system regulates bicarbonate (HCO3), one ofthe body's buffers. Therefore, to maintain the tight balance both the respiratory and metabolic system work together in an attempt to compensate for any abnormalities **[5] [6].**

Renal buffer: The renal system acts as a buffer through its ability to excrete or retain bicarbonate (HCO3). Bicarbonate is considered alkaline and although takes a little longer than the respiratory system to respond, is considered a powerful buffer. As the blood pH decreases (more acidic), the kidneys will compensate by retaining HCO3 and likewise, as the blood pH increases, the kidneys excrete HCO3 **[2].** When the lungs and kidneys are working together, they are able to maintain the pH of the blood within its narrow range of 7.35–7.45. It is when one or both of these buffer systems fail that the patient's status is compromised reflecting in abnormal arterial blood gases.

MATERIAL & METHODS:

This is a descriptive study carried out in the Dept. of Biochemistry in collaboration with 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. Informed written consent was taken from each patient in their known language.

By finding confidence of interval , fifty patients who developed AKI during the following hospitalization with an increase in serum creatinine (SCr) ≥0.3 mg/dl (≥26.5 µmol/l) within 48 hours , or an increase in SCr to ≥1.5 times baseline were included in this study. Collection of arterial blood samples in heparinized syringe for blood gas analysis was done on Cobas b 121 system blood gas analyzer (Roche). Blood samples were collected from radial artery or femoral artery. Analyzer contains the probe for testing of the sample. The values of PO2, PCO2, pH, HCO3- were studied.

Inclusion Criteria:

50 patients of acute kidney disease clinically diagnosed by Improving Global Outcomes criteria as an increase in serum creatinine more than 0.3 mg/dL within 48 hrs or increase in serum creatinine more than 1.5 times baseline after hospitalization, were selected for this study.

Exclusion Criteria

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

Statistical analysis:

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

Results:

In total study population of 50 patients 60 % (n=30) were male and 40 % (n=20) were female. Normal pH ranges from 7.35-7.45. Table 1 shows the distribution of study subjects as per pH level.

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Majority of subjects (n=34) are in the pH range of 7.35-7.45 that consists 68 % of the study subjects. 12 % of study subjects have pH <7.35 and 20% of study subjects had pH >7.45.

In our study, mean pH of AKI patients who survived (n=40) was 7.40±0.06 & who died (n=10) was 7.32±0.18. p value was 0.31. It was statistically significant.

Table1: Subjectsas per the pH level (male and female)

PH	Total No. (n=50)	Male (n=30)	Female (n=20)
<7.35	6	4	2
7.35 – 7.45	34	20	14
>7.45	10	6	4

Table 2: Mortality as per pH level

PH	Total patients (n=50)	Mortality (n=15)	Male (n=9)	Female (n=6)
<7.30	3	3	2	1
<7.35	6	3	2	1
7.35 – 7.45	31	10	6	4
>7.45	10	2	1	1

It is evident from the table no.2 that as pH value decreases, the mortality increases (20% → 29.4% → 50%→100%). Three subjects in our study group had pH level <7.30 and all these subjects didn't survive, mortality was 100%.

Table 3: Subjects as per HCO3 level

HCO3 level Meq/L	Total (n=50)	Male (n=35)	Female (n=15)
<22	12	9	3
22 – 26	30	20	10
>26	8	6	2

Table 3 shows distribution of study subjects according to bicarbonate level. Majority of patients 60% (n=30) had bicarbonate level in the normal range of 22-26 meq/l. 24% (n=12) patients had bicarbonate <22 meq/l. 16% (n=8) had bicarbonate level >26 meq/l

Table 4: Mortality as per HCO3 level.

HCO3 level Meq/L	Total No.(n=50)	Mortality (n=18)	Male (n=10)	Female (n=6)
<20	2	2	1	1
<22	10	5	3	3
22 - 26	30	10	8	2
>26	8	2	1	1

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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 50 %(5 out of 10), while in those with level 22-26 meq/l (10 out of 30) was 33.3% and in those with level >26 was 20%(2 out of 8). Thus there is higher rate of mortality in patients with bicarbonate level <22 meq/l (p value= 0.1). 3 subjects had bicarbonate level <20 and all 3 subjects didn't survive. So mortality was 100% in patients with level <20 meq/l.

Discussion:

The present study focuses on acid base balance and metabolic acidosis associated with the development of AKI and hospital mortality.

Whether acid base disturbances are contributors to acute kidney injury has rarely been discussed in previous studies **[1].** The role of serum bicarbonate level as a risk factor for renal outcomes has been evaluated in patients with chronic kidney disease **[4].** However it is not known whether metabolic acidosis affects the development of AKI in clinical settings. In this study it was found that acidosis especially metabolic acidosis was contributor to the incidence of AKI **[7] [8]**. Besides the previous study showed that patients with acidosis are 4 to 5 times as likely to die as patients who do not die, mortality among patients with metabolic acidosis is highest [8]. We found that acidosis and low bicarbonate levels were predictors of hospital mortality and were observed in AKI **[9] [10].**

CONCLUSION:

Measuring arterial blood gases can be a useful adjunct to the assessment of patients with either acute or chronic diseases. The results show that acidosis especially metabolic acidosis were contributors to the incidence of AKI and found that they can be the predictors of hospital mortality. The definite correlation was found between metabolic acidosis and mortality (p value was 0.031). Hence when combined with a patient's clinical features, blood gas analysis can facilitate in diagnosis and management.

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