PATTERN OF ACID BASE ABNORMALITIES IN CRITICALLY ILL PATIENTS

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ABSTRACT

Objective: To find out the pattern of acid base abnormalities in critically ill patients in a tertiary care health facility.

Study Design: A descriptive study.

Place and Duration of Study: The study was carried out in the department of pathology, Combined Military Hospital Kharian from January 2013 to June 2013.

Patients and Methods: Two hundred and fifty patients suffering from various diseases and presenting with exacerbation of their clinical conditions were studied. These patients were hospitalized and managed in acute care units of the hospital. Arterial blood gases were analysed to detect acid base status and their correlation with their clinical condition. Concomitant analysis of electrolytes was carried out. Tests related to concurrent illnesses e.g. renal and liver function tests, cardiac enzymes and plasma glucose were assayed by routine end point and kinetic methods. Standard reference materials were used to ensure internal quantify control of analyses.

Results: Two hundred and fifteen patients out of 250 studied suffered from acid base disorders. Gender distribution showed a higher percentage of male patients and the mean age was 70.5 ± 17.4 years. Double acid base disorders were the commonest disorders (34%) followed by metabolic acidosis (30%). Anion gap was calculated to further stratify metabolic acidosis and cases of diabetic ketoacidosis were the commonest in this category (47%). Other simple acid base disorders were relatively less frequent. Delta bicarbonate was calculated to unmask the superimposition of respiratory alkalosis or acidosis with metabolic acidosis and metabolic alkalosis. Though triple acid base disorders were noted in a small percentage of cases (05%), but were found to be the most complicated and challenging. Mixed acid base disorders were associated with high mortality.

Conclusion: A large number of critically ill patients manifested acid base abnormalities over the full spectrum of these disorders. Mixed acid base disorders were commonest and were bad prognostic indicators, most often associated with high mortality. This warrants a high index of suspicion, a thorough clinical assessment of patient and a structured approach to analyze the relevant laboratory data in the given clinical setting. Only with prompt detection of an acid base disorder, clinician can formulate an appropriate management strategy for the patient.

Keywords: Arterial blood gases (ABGs), Acid base disorder (ABD), Anion gap (AG).

INTRODUCTION

Acid base homeostasis is vital to the normal body physiology. If an acid base disorder is not detected timely, it may lead to serious or potentially fatal outcome. The appropriate diagnosis and subsequent management of an acid base disorder, in acutely ill patient, requires accurate and timely interpretation of the specific acid base disorder[1]. Appropriate interpretation requires simultaneous measurement of plasma electrolytes and arterial blood gases as well as an appreciation by the clinician of physiologic adaptations and compensatory responses that occur with specific acid base abnormality[2]. An early diagnosis of acid base disorder in critically ill patients would definitely improve prognosis[3].

It is natural to expect a high incidence of acid base disorders in critically ill patients. However the pattern of acid base disorders in critically ill patients is not commonly analyzed. A descriptive study, therefore, was performed in critically ill patients managed in intensive care units of this

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hospital with the aim to analyze the pattern of acid base abnormalities.

PATIENTS AND METHODS

This descriptive study was carried out at CMH Kharian from 1st January 2013 to 30th June 2013. All critically ill patients who developed acid base abnormalities were included in this study. Exclusion criteria were (1) those patients who were brought in dead or died shortly after arrival (2) patients transferred out within two hours of arrival (3) the patients with "Do Not Resuscitate" orders. The essential data regarding age, gender, primary disease, any complication and duration of stay were collected from medical record.

Arterial and venous blood samples were taken simultaneously from all patients for blood analyses including electrolytes estimation. Standard guidelines of International Federation of Clinical Chemistry (IFCC) and National Committee on Clinical Laboratory Standards (NCCLS) for arterial blood sampling were followed. Blood gases analysis was carried out on Arterial Blood Gases Analyser Nova Biomedical (pHOx Plus) UK. Electrolytes estimation was carried out on Easylyte Medica USA. Blood glucose, urea, creatinine and alanine aminotransferase were estimated by using routine end point and kinetic assays using commercial kits manufactured by M/S Linear (Spain) on fully automated chemistry analyzer Selectra ProM Netherlands. The ketone bodies in the urine were detected qualitatively. Appropriate control materials were used to ensure quality control. Descriptive statistics were used to express and analyze the data.

Acid base homeostasis or imbalance was judged according to the sample taken upon arrival by taking into consideration expected compensatory response. Anion gap (AG) was calculated by using the equation $Na^+ — (HCO_3^- + Cl^-)$ and the delta bicarbonate by $HCO^-_3 — 24$. A pH < 7.35 combining with increase of partial pressure of arterial carbon dioxide ($PaCO_2$) or decrease of bicarbonate is defined as respiratory or metabolic acidosis respectively. A pH > 7.45 combining with decrease of $PaCO_2$ or increase of HCO_3^- is defined as respiratory or metabolic alkalosis respectively.

Anion gap was used to further categorize the metabolic acidosis and delta HCO_3^- and delta Cl^- were used to define/characterize the superimposition of a high anion gap metabolic acidosis on a preexisting acid base disorder.

RESULTS

Various acid base disorders were observed in 215 critically ill patients. Double acid base disorders were present in 73 (34%) cases followed by 64 (30%) cases of metabolic acidosis, 20 (9%) cases of metabolic alkalosis, 30 (14%) cases of respiratory acidosis and 18 cases (8%) of respiratory alkalosis. Triple acid base disorders were found in 10 (05%) cases. Cases of simple acid base disorders along with their frequency and percentages are shown in table-1. Frequency and percentages of various combinations of disorders are depicted in table-2. Mixed acid base disorders were associated with high mortality. Among mixed acid base disorders metabolic acidosis with respiratory alkalosis occurred more commonly as compared to other mixed disorders.

DISCUSSION

Assessment of acid base status of critically ill patients is an integral component of diagnostic workup of these cases as various acid base disorders are present in such clinical scenarios. However the pattern of acid base disorders among critically ill patients being managed in acute care facilities is seldom reported. Our findings have shown that the incidence of Acid base disorders in such cases is very significant (86%) and was comparable with the previously reported figures[3,4].

In the category of simple acid base disorders, metabolic acidosis was the commonest (48%) and diarrhea constituted the major underlying disease entity (16%). A metabolic acidosis with normal

Anion gap (hyperchloremic) suggests that HCO_3^- has been effectively replaced by Cl^-. Loss of HCO_3^- from body through gastro intestinal or renal excretion with subsequent retention of Cl^- is one of the major under lying mechanism leading to hyperchloremic metabolic acidosis[5].

Table-1: Frequency and percentages of simple acid base disorders (n=132).

S.No.	Acid base disorders	Disease entities	Frequency	Percentage
1.	Metabolic acidosis (n=64)			
	a. Hyperchloremic metabolic acidosis	1. Diarrhoea	10	16%
		2. ACE Inhibitors	06	09%
	b. High anion gap metabolic acidosis	3. K^+ Sparing diuretics	08	12%
		1.Diabetic ketoacidosis	30	47%
		2.Chronic renal failure	10	16%
2.	Metabolic alkalosis (n=20)	1. Vomiting	14	70%
		2. Thiazide diuretics	06	30%
3.	Respiratory acidosis (n=30)	1. Stroke/CVA	04	13%
		2. Asthma	08	27%
		3. ARDS	04	13%
		4. Hypoventilation due to mechanical ventilation	04	13%
		5. COPD	10	34%
4.	Respiratory alkalosis (n=18)	1. Encephalitis	02	11%
		2.Congestive cardiac failure	08	45%
		3. Septicaemia	02	11%
		4. Hepatic failure	04	22%
		5. Mechanical ventilation	02	11%

Table-2: Frequency and percentages of double acid base disorders (n=73).

S. No.	Acid base disorders	Disease entities	Frequency	Percentage
1.	Metabolic acidosis and respiratory acidosis	a. Cardio pulmonary arrest (12) b. Respiratory failure with anoxia (04)	16	22%
2.	Metabolic acidosis and respiratory alkalosis	a. Septic shock (4) b. Congestive cardiac failure and renal failure (28)	32	44%
3.	Metabolic alkalosis and respiratory alkalosis	a. Congestive cardiae failure and vomiting (1) b. Diruetic therapy and hepatic failure (1) c. Diruetic therapy and pneumonia (1)	03	04%
4.	Metabolic alkalosis and respiratory acidosis	a. Diruetic therapy and chronic obstructive lungs disease (12) b. Vomiting and chronic obstructive lung disease (4)	16	22%
5.	Metabolic alkalosis and metabolic acidosis	a. Diruetic therapy and and keto acidosis (2) b. Vomiting and renal failure (2) c. Vomiting and kertoacidosis (2)	06	08%

Hypokalemia may accompany gastrointestinal loss of HCO_3^-. Diarrhea results in the loss of large quantities of HCO_3^- and K^+ from the stools[6]. The resulting volume depletion causes activation of renin, angiotensin and aldosterone system causing sodium and water retention along with increased renal K^+ excretion[7,8]. Different studies have highlighted the same mechanisms and disease entities in their studies[9].

In this study, 30 (47%) cases of metabolic acidosis due to diabetic ketoacidosis were noted. Though the ketoacids are excreted but are rapidly reabsorbed and cause high anion gap[10]. Diabetic ketoacidosis is caused by increased fatty acid metabolism and accumulation of ketoacids as a result of insulin deficiency or resistance[11]. These patho-physiological changes leading to high anion gap metabolic acidosis were also elaborated in different studies[12]. Chronic renal failure was the second most important disease entity in this subgroup. Ten (16%) cases of chronic renal failure presented with high anion gap metabolic acidosis. Poor filtration, together with continued reabsorption of poorly identified uremic organic anions contribute to the pathogenesis of this metabolic disturbance[13]. Acid base abnormalities in case of renal failure were also studied by adopting Figge's methodology and mechanism leading to acidosis as elaborated by Rocktaeschel et al[14].

Next simple acid base disorder observed was metabolic alkalosis. Patients who reported with history of vomiting (70%) followed by patients on thiazide diuretics (30%) were two important conditions. The same pattern has earlier been also observed in hospitalized patients developing metabolic alkalosis by Hodgkin et al[15]. Incidence of metabolic alkalosis and associated morbidity and mortality have also been extensively studied[16,17] and abnormalities highlighted in these studies were of same pattern as noted in our study. In assessing a patient with metabolic alkalosis two questions must be considered (1) What is the source of alkali gain (or acid loss)[18] and (2) what renal mechanisms are operating to prevent the excretion of excess HCO_3^- thereby maintaining, rather than correcting the alkalosis[19]. Both the above mentioned conditions lead to effective extracellular volume contraction, K^+ deficiency and secondary hyper-reninemic hyperaldosteronism leading to low urinary Cl^- and are hence saline responsive[18].

Out of 215 cases of acid base disorders 14% were due to respiratory acidosis. Incidence, etiologies and outcome/mortality related to respiratory acidosis in patients managed in acute care have extensively been studied[20] and the same pattern of respiratory abnormalities have been highlighted as noted in this study. Eighteen cases of respiratory alkalosis (8%) also presented in critical condition. Routpe et al[20] and Doyelela et al[28] studied the etiologies, morbid conditions and mortality of critically ill patients with respiratory alkalosis and the spectrum of clinical pathological changes observed was same as detailed in this study. Plasma potassium concentration is often reduced and chloride concentration is increased[20].

In many clinical situations, however there may be a mixture of acid base disorders. If the arterial acid base values fall outside the 95% confidence limits for simple acid base parameters this implies that a mixed disorder exists and a tentative diagnostic category can be assigned[21]. Different methodologies have been suggested to define limits of compensation which is a predictable physiologic consequence of the primary disturbance and does not represent a secondary disorder[21]. A significant number (34%) of patients showed mixed acid base disorders. Different combinations manifested are depicted in table-2. Many complicated clinical situations, especially in severely ill patients may give rise to mixed acid base disorders[22]. The same has been observed by Mc Curdy in patients who were critically ill[23].

A high anion gap acidosis has two identifying features: a low HCO_3^- concentration and an elevated anion gap. The elevated AG will remain evident even if another disorder coincides

to modify the HCO_3^- concentration independently[24]. The combination of (high anion gap) metabolic acidosis with metabolic alkalosis is not uncommon and is recognized when the anion gap is elevated but HCO_3^- and pH are near normal, that is, the change in anion gap is out of proportion to the change in HCO_3^- concentration (Δ AG > Δ HCO_3^-). Conversely when hyperchloremic metabolic acidosis and metabolic alkalosis occur concomitantly, increase in Cl^- concentration is out of proportion to the change in HCO_3^- concentration (Δ Cl^- > Δ HCO_3^-)[24].

Respiratory acidosis or respiratory alkalosis occurring with a mixed disorder of metabolic acidosis and metabolic alkalosis is another critical situation one may encounter in acutely ill patients. In this study 10 cases (5%) of total studied, manifested this spectrum of abnormality with respiratory alkalosis in 89% and respiratory acidosis in 11% cases. This has also been observed by Hu in his study carried out to identify / characterize triple acid base disorders and different approaches to analyze these abnormalities[25]. Triple acid base disorders are a serious metabolic derangement not to be missed in critical care. Some studies suggested that inappropriate intake of potassium wasting diuretics with inadequate potassium intake, excessive bicarbonate intake / prolonged gastrointestinal suction, excessive gastric lavage and mechanical ventilation are common iatrogenic factors inducing triple acid base disorder[26]. In triple acid base disorder combinations, respiratory alkalosis is more common than respiratory acidosis which reflects that respiratory compensation mechanism is major way leading to acid base disorder[27]. Triple acid base disorder is most complicated and challenging and may lead to serious morbidity and mortality. The prognosis is influenced by age and severity of disease. Its prompt detection and correction would therefore contribute to improve prognosis of the patient.

CONCLUSION

Critically ill patients manifest full spectrum of acid base abnormalities i.e simple acid base disorders as well as mixed. However, mixed acid base disorders are the commonest. Calculating anion gap and delta bicarbonate help in detecting triple acid base disorder promptly. These disorders warrant close monitoring as these are bad prognostic indictors in critically ill patients. Timely detection and appropriate correction of these abnormalities would have a profound effect on the outcome of such cases.

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