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Original Research Article

A short study of acid base disorders in CCU patients

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Abstract:

Introduction: Mixed acid base disorder in CCU patients can be defined as independently coexisting disorder characterised by varied proportions of metabolic acidosis (decreased bicarbonate, decreased pH), metabolic alkalosis (increased bicarbonate, increased pH), respiratory acidosis (increased $PaCO_2$) and metabolic alkalosis (decreased $PaCO_2$) depending on specific diagnosis of the patient.

AIM (Objective): To identify incidence of acidosis and alkalosis (respiratory and metabolic) in CCU patients of SCCGMCH, Uluberia, based on ABG parameters considering their normal range.

Method: Cases comprised of 100 CCU patients whose blood samples were tested in ABG analyser of the institutional CCU within a period of six months. Diagnosis of acid base disorder was made considering normal ranges of the parameters ascertained by prevailing worldwide standards.

Result: Incidence of metabolic acidosis in our sample of CCU patients was found to be higher followed by respiratory alkalosis, while incidence of metabolic alkalosis was much lower followed by respiratory acidosis.

Conclusion: The early diagnosis of acid base imbalance and its monitoring has been helpful in the diagnosis and treatment of the terminally ill patients. Improved arrangement for more running ABG analysers in peripheral medical colleges like SCCGMCH will surely help to reduce the mortality and morbidity of rural population.

Keywords: Acidosis, Aetiology, Metabolism, Alkalosis, Aetiology, Metabolism, Anionic Gap, Electrolyte Derangement, Arterial Blood Gas Analysis.

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Introduction

Mixed acid base disorders in critical care unit can be defined as independently coexisting disorder and not merely a compensatory response [1]. Metabolic acidosis can occur because of increasing endogenous acid production (Ex: Lactic Acid, Ketoacid), loss of bicarbonate (Ex: Diarrhoea) or accumulation of endogenous acid (Ex: Renal failure) [1]. The fall in pH is accompanied by characteristic increasing ventilation (Ex: Kussmaul respiration) [1,2,3]. CNS function is depressed with headache, lethargy, stupor, coma [1]. Acid base disorder can be evaluated by simple calculation of Anionic Gap $AG=[Na^+ + K^+]-[Cl^- + HCO_3^-]$ = 10- 12 mmol/L. An increase in Anionic Gap can be due to decrease in unmeasured cations (Ca^{++}, Mg^{++}, K^+) or increase in unmeasured anions (phosphate, sulphate) [1]. Serum for electrolytes and arterial blood for gases drawn simultaneously show decrease in $[HCO_3^-]$ in metabolic acidosis and respiratory alkalosis while increase in $[HCO_3^-]$ occurs in metabolic alkalosis along with hypochloraemia and hypokalaemia [1,2]. Respiratory acidosis with increase in $PaCo_2$ can be due to compensatory hypoventilation [5,6] in severe pulmonary disease (Ex: COPD,), respiratory muscle fatigue (Ex: Chest wall disorder), abnormality in ventilatory control due to 1) decreased stimulation of respiratory centre (Ex: CVA, meningitis, brain Injury [5], sleep apnoea, coma, drug (narcotic, barbiturate) or 2) due to peritonitis (2). It is accompanied by increase in $PaCo_2$ [8,9] leading to increase in H_2CO_3 causing increase in H^+ and HCO_3^- ---> Na^+-H^+ exchange through kidney increases and H^+ is buffered by Hb and protein. This leads to increase in $[HCO_3^-]$ causing metabolic alkalosis [2,6]. Respiratory alkalosis due to alveolar hyperventilation[10] occurs due to increased stimulation of respiratory centre (e.g. in anxiety, fever, gram negative septicaemia, hypoxia due to severe anaemia or in high altitude, drugs, catecholamines, hyperthyroidism, pulmonary

disorders like pneumonia ,interstitial lung disease, pulmonary emboli) leading to decrease in $PaCO_2$ [7,9]and increase in [HCO_3^-/ $PaCO_2$] ratio .This leads to shift in normal equilibrium of bicarbonate carbonic acid buffer system causing decreased formation of H^+ from carbonic acid thereby increasing the pH.]. Our present study aims to diagnose acidosis (metabolic and respiratory) and alkalosis (metabolic and respiratory) in CCU patients based on ABG parameters.

Material and Method

The study was hospital based, retrospective study conducted at a tertiary care centre in West Bengal, India. The study was approved by Institutional Ethical Committee of Sarat Chandra Chattopadhyay Government Medical College & Hospital, Uluberia, Howrah. Before enrolment for the study, written informed consent were obtained from all the participants. The duration of study was six months and cases comprised of 100 patients in CCU. Diagnostics criteria for acid base disorder was based on normal standardised values of respective parameters The bio-chemical parameters were assayed by arterial blood gas analysis in ABG analyser in CCU. The quality of results was validated with internal quality control (IQC) procedure participation to an external quality assessment scheme (EQAS). The study was carried out in accordance with declaration of Helsinki and with the term of the local legislation. Statistical analysis was done in Microsoft excel 2013.

Following data was obtained:

Acidosis (21)		Alkalosis (28)	
Respiratory (2)	Metabolic (19)	Respiratory (19)	Metabolic (6)

*3 patients showed alkalosis which cannot be categorised either as respiratory or metabolic alkalosis.

Discussion

Among 100 CCU patients, 21 patients developed acidosis of which 19 patients had metabolic acidosis with their pH varying between 6.94 – 7.34(normal pH: 7.35 -7.46) due to fall in [HCO_3^-] level [6]ranging between 4.8 -21.9 mmol/L (normal [HCO_3^-] 22 -29 mmol/L) along with increase in AG [6]varying between 11.5 – 24.7 (normal AG 10 – 12 mmol/L) .There was associated increase in lactate varying between 1.55-9.12 mmol/L (normal lactate .56 – 1.39 mmol/L) in patients suffering from uterine rupture, ectopic pregnancy ,post LUCS, placenta previa , MI , PIH , PPH or RTA. So, patients were either in shock leading to increase in respiratory rate [11] and thereby decrease in [HCO_3^-] level or patients were in respiratory distress and so there was rapid breathing to have proper oxygenation of blood leading to decrease in [HCO_3^-] level. [1, 3].

1 case of metabolic acidosis pH (6.94) presented with decreased [HCO_3^-] 4.8 mmol/L, increase in [Cl^-] 112 mmol/L [12] (normal [Cl] 98 -117 mmol/L) with increased AG 30.4 mmol/L (in diabetic ketoacidosis). Another case of post cholecystectomy presented with metabolic acidosis (pH 7.28), decreased [HCO_3^-] (21.9) and increased [Cl^-] 110 mmol/L. with normal AG 11 mmol/L. 1 case of chronic kidney disease showed metabolic acidosis (pH 7.3) with decreased [HCO_3^-] 11.1 mmol/L and increased anionic gap 16.4 mmol/L but not with increased lactate or not with increased [Cl^-] (13). 1 case of septic shock with increased respiratory rate presented with metabolic acidosis (pH 7.28) with decreased [HCO_3^-] 13 mmol/L and increased anion gap 27.5 mmol/L with increased lactate 2.3 mmol/L and increased [Cl^-] 131 mmol/L

2 patients of COPD presented with respiratory acidosis, one of them having increased $PaCO_2$ 98.7 mm of Hg (Normal $PaCO_2$: 35 -45 mm of Hg), decreased pH 7.25 and normal [HCO_3^-] level while another with pH 7.23 and increased $PaCO_2$ 84.3 mm of Hg and increased [HCO_3^-] 34.8 mmol/l . The study done by Mohammed Tinawi et al [8] supports the outcome of the study.

28 patients developed alkalosis with pH ranging between 7.46-7.62 of which 19 patients with either shock or hypoxia had respiratory alkalosis with decreased PaCO2 ranging between 24.2 – 34.4 mm of Hg and increased pH (7.46-7.58). These patients suffering from acute myeloid leukaemia, anaemia, chronic kidney disease, hypotension or hyperemesis gravidarum, cerebrovascular accident, postpartum ecclampsia, lower respiratory tract infection, hanging or ruptured uterus. ultimately lead to either shock or hypoxia causing increased stimulation of respiratory centre. This lead to decreased $PaCO_2$, with increased [HCO_3^-/ $PaCO_2$] ratio causing shift in normal equilibrium of bicarbonate carbonic acid buffer system resulting in decreased [H^+] ion from carbonic acid. This shift also caused decrease in [HCO_3^-] thus improving pH. Richard J. Heber et al supported this study [2,7]. Of these 19 respiratory alkalosis patients,1 patient with pH 7.48 and decreased $PaCO_2$ 29.8 mm of Hg and increased anionic gap 16.6 mmol/L and increased lactate 5.22 mmol/L was suffering from respiratory distress, hypertension and diabetes mellitus. Respiratory distress lead to decreased $PaCO_2$ with increased pH while due to diabetes mellitus there was increased anionic gap with increased lactate. Among 6

patients with metabolic alkalosis, 3 patients with COPD presented with increased pH, increased [HCO_3^-] and decreased [K^+] or chloride. This lead to hypoventilation with increased $PaCO_2$ [5,6].

3 patients with LRTI, COPD and LVF developed metabolic alkalosis with increased pH, increased [HCO_3^-] (29.5- 42.4 mmol/l), decreased [K^+] (1.92-2.5 mmol/L) and decreased [Cl^-] (78-96 mmol/L) due to hypoventilation. 3 Cases of alkalosis with pH 7.46, normal PaCo2 & normal HCO_3^- suffering from CVA, AMI, LVF with CKD could not be categorised as respiratory or metabolic alkalosis.

The study has certain limitations. Since the number of cases was not large enough so our study result might not reflect the true picture of entire population. Hence multicentric study on larger population for longer duration should be required to derive a proper conclusion.

Conclusion

This study truly provides a statistic of the incidence of acid base imbalance among the common CCU patients in a peripheral medical college like SCCGMCH, Uluberia. Early detection and prompt correction of the imbalance had helped in the treatment of these patients. This study therefore truly reflects the necessity of instalment of larger number of running ABG analysers in peripheral setups. However, a more detailed study with larger sample size and morbidity and mortality data would have been more beneficial in providing a clear picture of the scenario.

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