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A Preliminary Study of Acid Base Balance Status In Intensive Care Unit Non Vegetarian Malayalee Patients Admitted with Metabolic Encephalopathy.

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ABSTRACT

To study the acid-base status of the malayalee patients admitted in Medical Intensive care Unit with metabolic encephalopathy, to know about the most common underlying disease, to understand the prevalent metabolic disorder among nonvegetarian patients in the Malappuram region of Kerala state, India. Estimation of acid base parameters and electrolytes (Na^+, K^+, and Cl^-) in intensive care unit patients with metabolic encephalopathy were done in a total of 43 adults admitted with metabolic encephalopathy. Simultaneously, age and sex matched 25 non vegetarian malayalee of control group were also studied. Most of the ICU admitted nonvegetarian malayalee patients showed significant acid base and electrolyte balance disorders. Most cases of metabolic encephalopathy had an identifiable precipitating factor. Disorders like Hyponatremia require timely recognition and can often be reversed with appropriate intervention and treatment of underlying predisposing factors. Early diagnosis of hyponatremia, and electrolyte imbalances, in the ICU admitted patient may prove life saving in a nonvegetarian malayalee patient.

Keywords and phrases: ICU (intensive care unit), metabolic encephalopathy, hyponatremia

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INTRODUCTION

Advances in investigative acid base chemistry have led the intensive care medicine field over the past century [1, 2]. It has been appreciated time and again that the human body religiously controls the relative concentrations of hydrogen and hydroxyl ions in the extra cellular and intra cellular spaces and that a significant alteration in this "balance" disrupts human transcellular ion pumps [1-4]. Deviations of systemic acidity in either direction can impose adverse consequences and such deviations, when severe can threaten human life itself [3, 4]. It is well established that characterization of acid –base balance in the body is of central importance in emergency medicine, since it may provide valuable information about the status of a patient or provide clues about the underlying pathophysiology of a patient's disease process or possible outcomes [5-7].

Acid base balance is maintained by chemical buffering and by pulmonary and renal elimination of H^+ [8-10]. Disturbances of these mechanisms can have serious clinical consequences. Disturbances of the acid base equilibrium occur in a wide variety of critical illnesses and are among the most commonly encountered disorders in the ICU [10-11]. Acid base equilibrium is closely tied to fluid and electrolyte balance and disturbances in one of these systems often affect another [11]. The sum of cation and anion in ECF is always equal ,so as to maintain the electrical neutrality .Anion gap is calculated as the difference between ($Na^+ + K^+$) and ($HCO_3^- + Cl^-$).The alteration in anion gap is extremely useful in the clinical assessment of patients with acid base disorders [7,8,11].

Kinnier Wilson in 1912 coined the term "metabolic encephalopathy" [7] to describe a clinical state in which the brain's integrated activity is impaired in the absence of structural abnormalities. Metabolic encephalopathy can vary in clinical presentation from mild executive dysfunction or agitated delirium, to deep coma with decerebrate posturing. There are also some differences in presentation depending on the underlying metabolic disorder [7, 8]. Those underlying disorders causing encephalopathy can be due to vital organ failure, nutritional deficiencies, electrolyte imbalances, hypoglycemia, hyperglycemia, endocrine disorders, and systemic sepsis, cardiac arrest and anoxic-ischemic encephalopathy, direct CNS infections, exogenous toxins (including drugs, alcohol, and poisons), hematological conditions, immune-mediated CNS diseases, and direct and indirect effects of cancer on the nervous system [9].

MATERIALS AND METHODS

Our study was conducted in the tertiary referral centre, M.E.S Medical College Hospital; Perinthalmanna, situated in Malappuram Region of Kerala, where a majority of population consume a mixed diet which is predominantly non vegetarian. We studied a total of 43 cases of adults admitted in intensive care unit with metabolic encephalopathy and 25 cases of control group over a period of three months from October 2009 to December 2009. Medical Intensive care unit Patients with age group between 33 and 80 were included while patients with structural coma were excluded.

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All the acid base abnormalities seen could be easily interpreted and evaluated directly and quantitatively, as abnormalities of the variables pCO_2, HCO_3^-, anion gap and pH. The data required for such evaluation and the concentrations of the electrolytes (Na^+, K^+, and Cl^-) were available from arterial blood gas measurements and serum electrolyte panel. Permission for the conduct of the exploratory pilot study was obtained from the hospital authorities. After taking a written consent to study, diet history,patient data of the admitted patient was collected using a structured proforma containing relevant clinical details of patients.

TEST SPECIMEN:

An arterial whole blood specimen was obtained from each patient by his/her ICU nurse or intensivist under aseptic precautions. A portion of that specimen was placed directly in to a heparinized (Arterial bold gas) ABG gas syringe, while the reminder was placed in an EDTA vacuum collection tube. The arterial blood in the heparinized ABG gas syringe was transported on ice to the hospital core laboratory and analyzed using a calibrated ABG analyzer [12-14]. The sealed syringe is taken to a blood gas analyzer for measurement of the blood pH, pO_2 and pCO_2 [13, 15, 18]. The bicarbonate concentration was also calculated [15, 16-18].

ESTIMATION OF ELECTROLYTES (SODIUM, POTASSIUM, and CHLORIDE):

Serum obtained from blood collected in an EDTA vacuum collection tube was used for the sodium, potassium and chloride assay. The estimations are done in VITROS 250/350 Dry Chemistry System, which automatically measures the concentration of electrolytes by dry slide technology and the results were noted as mmol/L.

Figure 1: VITROS 250/350 DRY CHEMISTRY SYSTEM

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Figure 2: ABG ANALYSER

RESULTS AND DISCUSSION

Out of the 43 ICU malayalee non vegetarian metabolic encephalopathy patients 29 (67 %) were males and 14 (33 %) were females (Graph.1) (diagram.1).A net of 79.31% of males were smokers and 34.48 % had a habit of alcohol intake. Our finding has significance in preventive and public health care setting, and could be of use to health planners of Kerala state in particular.

Graph -1: Frequency distribution of metabolic encephalopathy in Different age groups

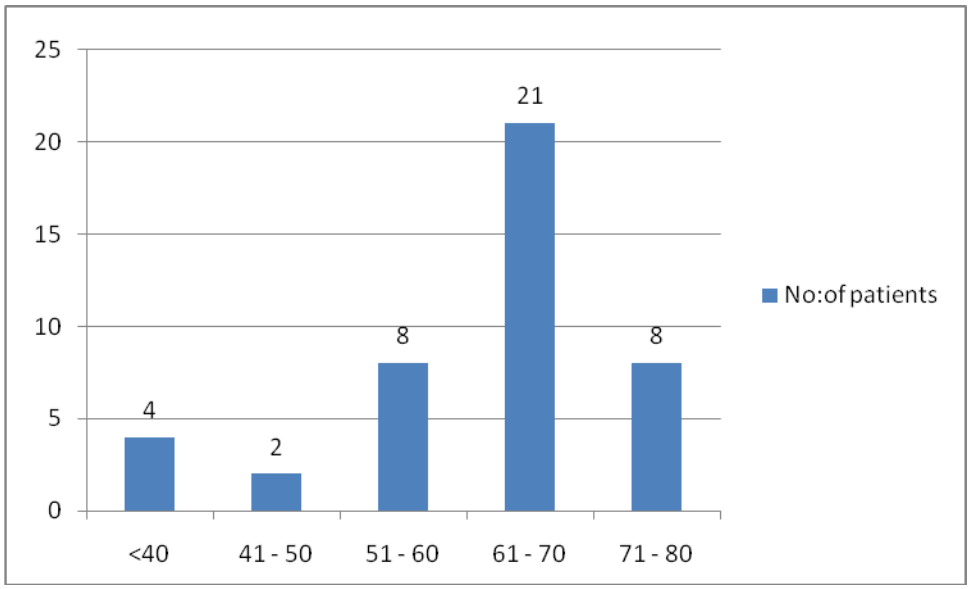

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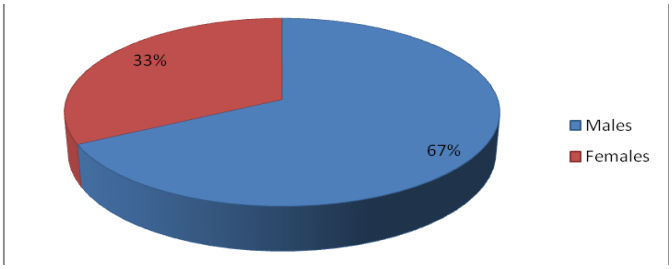
Diagram -3 showing: Patient's distribution -Gender wise percentage

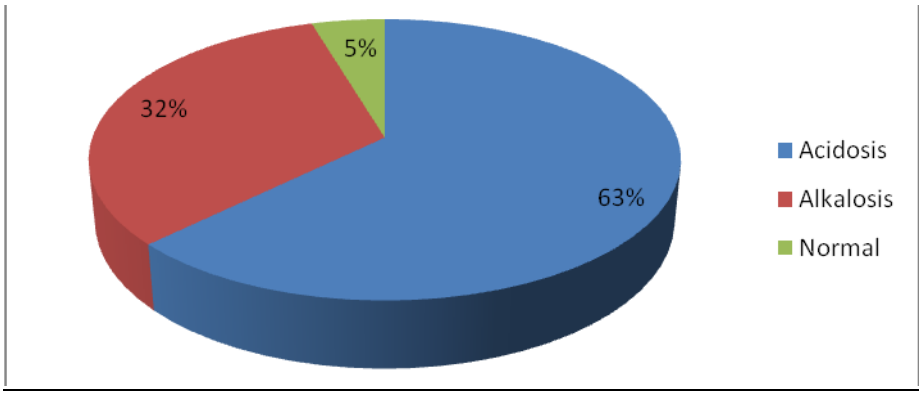


A total of 25 age and sex matched, malayalee non vegetarians, without any signs or symptoms of metabolic encephalopathy were used as controls. In these normal subjects, $HCO3^-$ and anion gap were within the range of established normal values. Among the acid base abnormalities, in our study, some of the ICU patients had primary metabolic cause (acidosis or alkalosis) and others have mixed acid base disturbances with primary metabolic cause. In fact, nearly two thirds i.e 27 were having acidosis, while about one thirds i.e,14 had alkalosis and 2 patients were within the normal pH range (diagram.2). The measured values were expressed as average with standard deviation. (Table .1)

Table -1

Parameters	Mean ± Standard deviation
Sodium	127.21 ± 10.19
Potassium	4.08±1.12
Bicarbonate	20.19± 6.63
Chloride	92.9±9.93
Anion gap	18.43±5.35
pH	7.28 ± 0.29
pCO2	. 35.53±7.3
pO2	87±18.102

Diagram-2: Distribution of patients based on pH



Hyponatremia has been described as a major electrolyte derangement in hospitalized patients especially in intensive care units and we too agree with the finding, and note its significance [19, 20]. In our study within ICU admitted metabolic encephalopathy patients, overall 83.72 % were hyponatremic.

In this study, 14 were hepatic encephalopathy patients (32.56 %) and out of this 11 with portal hypertension and liver cirrhosis. All these 14 had a mixed acid base disorder, where the primary cause was a metabolic one with a high anion gap. Males outnumbered females in our study as in the western study. The male preponderance in west is explained by patterns of alcohol consumption where 77% cases of chronic liver disease are related to alcoholism. Interestingly 75(94%) patients had viral etiology of cirrhosis in this study [21, 22]. In our study 13 (30.23% of metabolic encephalopathy patients) were Chronic obstructive pulmonary disease (COPD) patients. In a study conducted at Gaffrée e Guinle University Hospital (HUGG) between 2000 and 2006, the results of the study, although admittedly preliminary, suggest that the concept of sub clinical encephalopathy might be extended to patients with COPD [23]. In another study conducted at Yang University Hospital, Korea, in COPD patients the $PaCO_2$ was 39 ± 7 mm Hg, and the Pao_2 was 89 ± 18 mm Hg, and results demonstrate that the cerebral metabolism is significantly altered in symptomatic COPD patients [24].

Again, in our study, only 2 patients with encephalopathy were admitted for sepsis, so 4.7% have infectious etiology. This finding is in agreement with studies by Souheil who have reported infections responsible in only 3% of cases of metabolic encephalopathy [25]. Our findings are also in agreement with another study by Conn where infections were responsible in only 4% of the ICU cases [26]. In a study at admission to the ICU, septic patients presented with a severe metabolic acidosis scenarios with an average pH of 7.29; with average pCO_2 = 36 mmHg values [26, 27]. We observed that in our studies that, the average pH values within the ICU patients with metabolic encephalopathy among malayalee non vegetarians were in the range of 7.28 ± 0.29.These values are also in agreement with studies by Conn & Leiberlhal (1980) and Noritomi et al (2007) [26,27].

Again, 8 of our 43 ICU patients with metabolic encephalopathy have type 2 diabetes mellitus. It has been noted that diabetic patients have severe hepatic encephelopathy at earlier stages of biochemical decompensation and portal hypertension compared with nondiabetic patients [33]. In our study the 'p value' is less than 0.05 (5% level of significance). So there is a significant difference between the values of the two groups, ie normal persons and the patients [30-33].

Again, while 28 patients in the study showed a mixed acid base disorder (65 %) the remaining 13 showed evidence of a metabolic disorder (30%) (Diagram 3).It was seen notably and conspicuously that there is a high serum anion gap disorder predominantly (74.42 %) while normal serum anion gap disorder was 20.93 % and low serum anion gap disorder was a meager 0.02 %. Again this biochemical finding has gross clinical implications in intensive care unit outcomes.

Graph-2: Distribution of electrolyte imbalance

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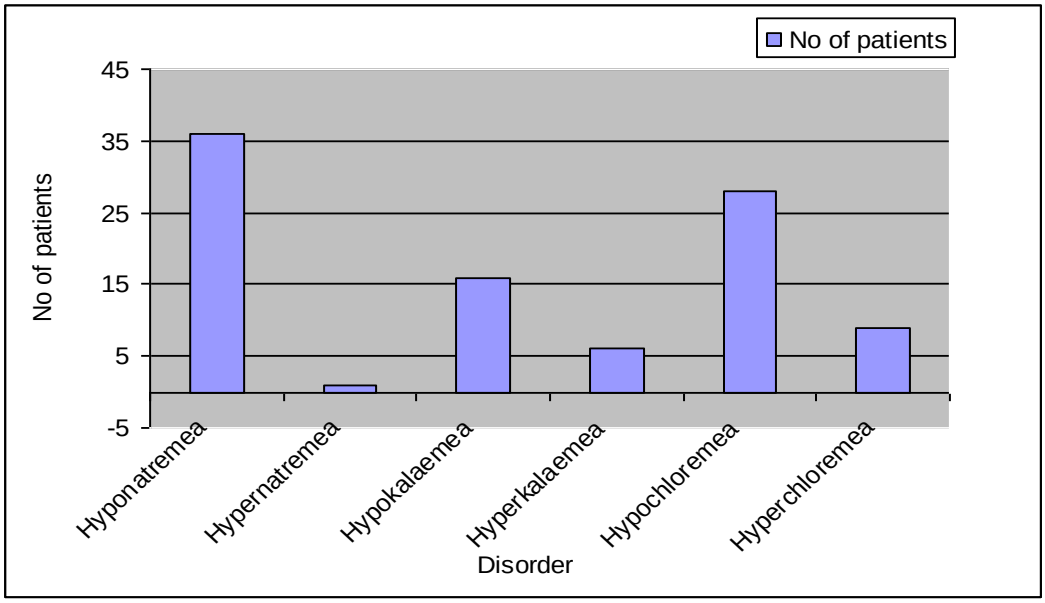


Diagram- 3: Distribution of Acid-Base disorder

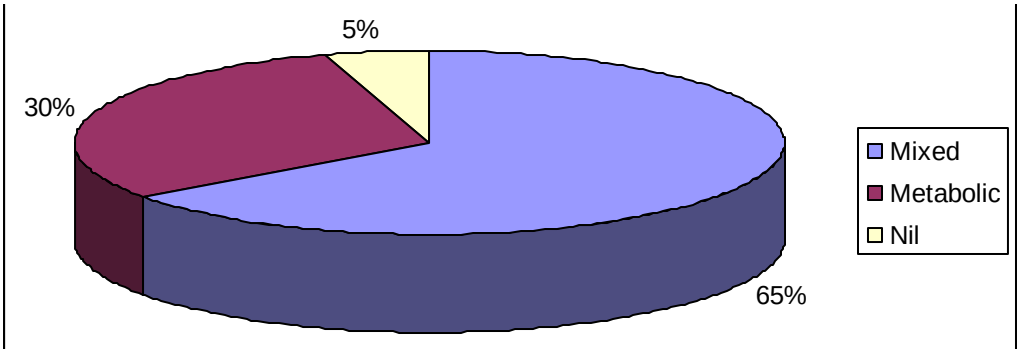
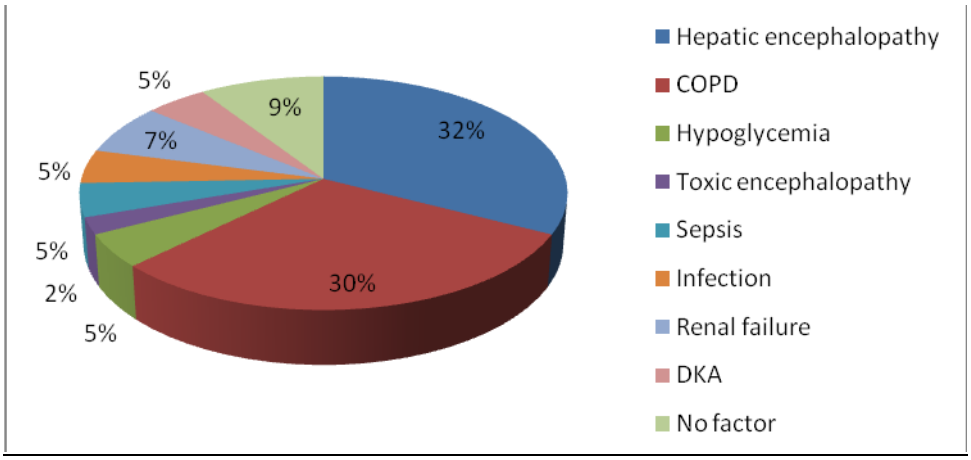


Diagram-4: Distribution of precipitating factors



It was seen that all the patients in the intensive unit were non vegetarian. Precipitating factors leading to encephalopathy were ascertained. In majority of cases, we found out that encephalopathy was secondary to a precipitating factor. Some of them have more than one factor. They are shown in Diagram.4. The level of statistical significance of the study was measured by calculating the 'p value' for 5 %.i.e., (p value <0.05) .The 'p value' for different parameters are shown in the table 2.

Table-2: 'p Value' for 5% level of significance

Measured parameters	t value	p value
Sodium	4.5	<0.0001
Potassium	2.01	0.0243
Bicarbonate	3.26	0.0018
Chloride	4.29	<0.0001
Anion gap	3.97	0.0001
pH	2.09	0.0202
pCO2	3.19	0.0022
pO2	2.99	0.0020

The most commonly encountered causes of metabolic acidosis in the ICU are renal insufficiency, sepsis, and Diabetic ketoacidosis [29]. Alkalosis, on the other hand, is less common in the ICU. Fluid status derangements and, especially, gastric fluid depletion are the usual underlying causes of metabolic alkalosis [30].

In 4 (9.3%) patients in this study, no precipitating factor was found for metabolic encephelopathy. This agrees with similar finding in a Pakistani study conducted at Lahore, in 2007, where, in 8.75% of patients no precipitating factor was found [21]. From a Islamabad study, Maqsood reported no precipitating factor of hepatic encephalopathy in 10% of cases [28]. In our study, 2 patients had hypoglycemia (5%) which can be surmised to be the cause of Hypoglycemic coma (neuroglucopenia) [31].

In our study we had only 1 of the 43 patients with toxic encephalopathy (2.3%).3 of the 43 metabolic encephalopathy sufferers were having renal disease (7%), out of which one had thrombocytopenia. 2 of our 43 patients had chronic kidney disease with diabetic nephropathy (DKA) (4.7%).studies have reported that serum sodium and potassium, and arterial bicarbonate, concentrations are frequently abnormal in Acute renal failure patients [32-34].

We also found out that, amongst the malayalee non vegetarians with metabolic encephalopathy, hyponatremia is the most predominant disorder noticeable, and that nearly 4 out of every 5 intensive care unit patients admitted with metabolic encephalopathy (83.72 %) were hyponatremic and a mere 0.02 % were hypernatremic . Simultaneously, we can also usefully note that 65.12 % were hypochloremic and 20.93 % were hyperchloremic (Graph 2). While our study results are of a small size, and larger studies at different locations across Kerala among non vegetarians may be needed, it would not be premature to conclude that early recognition of hyponatremia and hypochloridemia may indeed save many a life in the ICU

setting. So, if alert intensivist clinicians could easily spot hyponatremia and hypochlorideemias biochemically, and treat aggressively, while keeping a close watch on hypokalemia, it could go a long way in securing a successful early exit from metabolic encephalopathy and the intensive care unit, and its costs.

Also important was another find that nearly 37.21 %(more than one third) of non vegetarian metabolic encephalopathy patients had hypokalaemic values and 13.95 % (more than one tenth) turned out to be hyperkalaemic.We have to interpret these findings in conjunction with aforesaid sodium levels, as well as chloride status of the patients. This could be due to the partial supplementation of the intensive care unit patients with Coconut water, in the diet of ICU patients, which happens to be rich in potassium [35-37].

CONCLUSIONS

Most cases of metabolic encephalopathy within the tertiary care unit ICU had an identifiable precipitating factor. Hepatic encephalopathy, Chronic obstructive pulmonary disease, sepsis, infection, Hypoglycemia, renal insufficiency and Diabetic ketoacidosis were the most common precipitating factors in our setting. These disorders require timely recognition and can often be reversed with appropriate intervention and treatment of underlying predisposing factors. Most of the ICU admitted nonvegetarian malayalee patients showed significant acid base and electrolyte balance disorders. Our study showed that acidosis is most common than alkalosis. Hyponatremia was the predominant electrolyte imbalance according to us in the ICU admitted non vegetarian malayalee. Biochemical disorders like Hyponatremia require timely recognition and can often be reversed with appropriate intervention and treatment of underlying predisposing factors. Early diagnosis of hyponatremia, and electrolyte imbalances, in the ICU admitted patient may prove life saving in a nonvegetarian malayalee patient. Also 65 % were with mixed acid base disorder having primary metabolic cause and 30 % with metabolic acid base disturbances. 79.31% of males were smokers and 34.48 % having a habit of drinking. Long term measures like smoking cessation and anti-alcoholism initiatives could help this part of Kerala avoid unpleasant intensive care unit outcomes.

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