(3) EVALUATION OF HYPONATREMIA IN HEART FAILURE PATIENTS ADMITTED IN CRITICAL CARE UNIT: SINGLE CENTRE EXPERIENCE.

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Abstract :

Background & Objectives :

Hyponatremia is a very common electrolyte disturbance in hospitalized patients of heart failure. Clinical profile of such patients with hyponatremia and common co-morbidities are subjects of current study where we made an attempt to evaluate severity, symptoms and in hospital outcome of such patients.

Methods :

All patients presented to intensive coronary care unit (ICCU) with decompensated congestive cardiac failure (CCF) during three months period in 2012 were included in the study.

Result :

Out of total 1184 ICCU admissions, 650 were presented with decompensated heart failure. Serum sodium levels less than 135 mmol/L was found in 334 patients while severe hyponatremia less than 110 mmol/L was seen in 5.3%. Symptomatology ranged from mild irritability and intractable vomiting to seizures and unresponsiveness on neurologic evaluation. Male preponderance (64.40%) was noted. Common co-morbidities were hypertension (64.22%), Diabetes mellitus (32.41%), Ischemic Heart Disease (67.42%) and valvular heart disease (10.5%). Two diuretic molecules were co-administered in (39.70%). Females tolerated different grades of hyponatremia better than males with fewer symptoms and less mortality.

Conclusion :

Hyponatremia is more common in male patients presenting with CCF however, female patients with CCF have less mortality and fewer symptoms. There is a direct relation of serum sodium concentration and in hospital mortality.

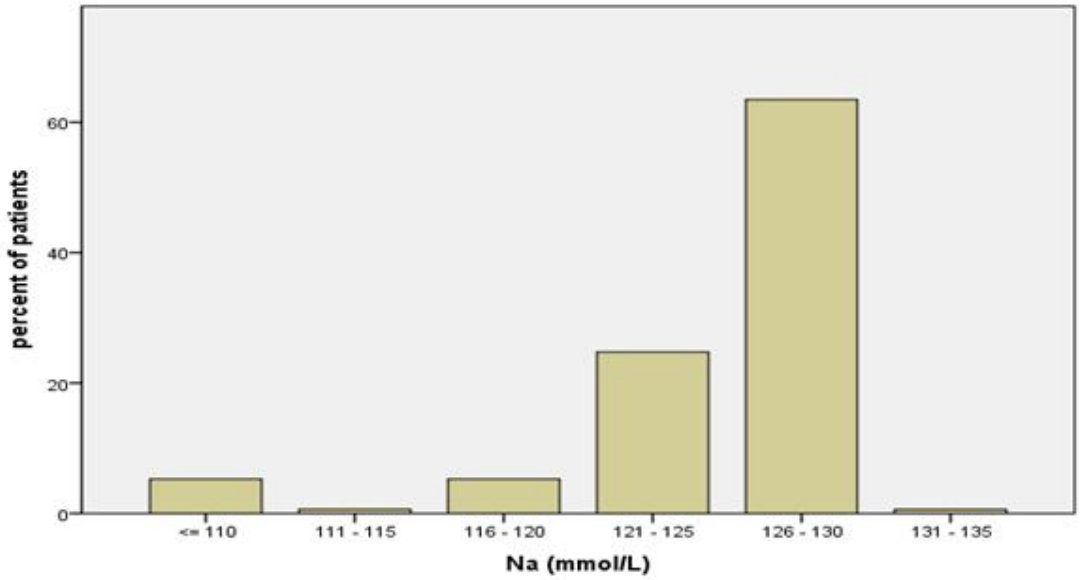
Key words :
Hyponatremia, congestive cardiac failure, diuretics.

Introduction :
Hyponatremia is found to be fairly common biochemical abnormality in CCF patients. Primary mechanism is dilutional hyponatremia where arginine vasopressin (AVP) is secreted without any triggering signals of osmolality change. Severe heart failure causes hormonal release of AVP via baroreceptor mediated signals. Studies have shown that prevalence of hyponatremia (defined as less than 135mmol/L) ranges from 15 to 30% in different chronic care hospital settings. The incidence is much common in elderly and female patients with some impairment of renal or cardiovascular functions. The symptoms of hyponatremia develop according to severity and the speed of fall in sodium concentration. Reducing plasma concentration of sodium generates an osmotically driven water movement into brain cells resulting into brain oedema, raised intracranial pressure and neurologic symptoms. When hyponatremia builds up slowly brain cells have regulatory mechanism operating in few hours to days which prevents brain oedema and hence fewer symptoms. Organic solutes like glutamate, taurine, myoinosilol and glutamine are involved in counter regulatory transport across the cell membrane, thus ameliorating cellular oedema in brain during chronic stages of hyponatremia. Hyponatremia predicts higher mortality in congestive cardiac failure; community acquired pneumonia and hospitalized patients in general. Hyponatremia has wide variety of causes, commonest being overzealous diuretic usage combined with salt restriction in hypertensive individuals & CCF patients. While acute hyponatremia symptoms may progress to death, the treatment for hyponatremia is also carrying serious risk of Central Pontine Myelinolysis (CPM). This complication is result of rapid correction of hyponatremia. Brain cells require time to adapt to changes in osmotic environment. Pons have very tight grid arrangement of glial tissue and axons hence, Pons is the seat of major injury of osmotic imbalance.

Data regarding incidence of hyponatremia in heart failure patients and ICCU patients are very scanty in our country. The present study was aimed at getting clinical features, aetiology and in hospital outcome of variable digress of hyponatremia in CCF patients admitted in ICCU.

Methods :
1184 Consecutive patients admitted in our hospital with symptoms of decompensated congestive cardiac failure between 1st June, 2012 to 30th Sept., 2012 were evaluated. Relevant history and clinical features were recorded including past medical history, drug history, examination findings and volumic status was recorded. Skin turger, pulse rate, postural blood pressure, oedema legs, basal craps in lung fields, jugular venous pressure etc. were subjectively evaluated to record volumic status of patients and accordingly, hypervolaemic, euvolaemic and hypovolaemic status categories were designated to classify patients of CCF. Routine blood and urine tests including complete blood counts, renal function tests, electrolytes, liver function tests, chest radiographs, transthoracic echocardiography, troponin I levels, CT scan (if clinically indicated) were performed in all these patients. All patients were treated for hyponatremia as per hospital protocol.

Investigations were repeated during hospital stay as per clinical judgment of caretaker physician. Serum sodium concentration was measured on 'Cobas Integra 400' fully automated 'Roche Diagnostics' machine.
Normal laboratory range is 136 to 145 mmol/L.



Results :

Total 1184 patients were admitted to ICCU of our hospital between 1st June, 2012 upto 30th September, 2012. Out of these, 650 patients presented with clinical features of congestive cardiac failure. Total of 334 patients have serum sodium concentrations less than 135mmol/L.

The mean age of patients having hyponatremia and CCF was 44.25 years. Male preponderance was noted 64.40%. The mean sodium level on admission was 122.95 mmol/L and pre-discharge repeat levels were done with mean value of 131 mmol/L. Hypervolemic status was seen in 71.42% and 26.20% patients were found euvolemic.

Mild hyponatremia was asymptomatic. However, in CCF patients even levels below125 mmol/L were asymptomatically tolerated. Nausea and malaise were frequent below 120 mmol/L and headache, lethargy, confusion, disorientation prevail after levels reach below115 mmol/L. Most common presentation was drowsiness at 110 mmol/L and seizures, coma was found at 105 mmol/L levels.

Drowsiness, irrelevant talk and poor response to verbal stimuli without any focal neurologic deficit were the (68.71%) most common central nervous system (CNS) presentations. Three cases had significant loss of fluid with intractable vomiting. All patients with neurological symptoms were evaluated for structural damage on CT Scan. No structural neurologic abnormalities were detected in CT Scan.

The common co-morbid conditions were hypertension (64.22%) and Diabetes mellitus (32.41%) and ischemia heart disease (67.42%), renal failure (13.60%). Co-existing hypokalemia was noted in 7.10%. Two different molecules for diuretics were used simultaneously in 39.70%. Metolazone use was seen in 3.7%. 28 patient succumbed to their heart failure. Out of them, 16 had sodium levels below 105 mmol/L. Female patients had lower mortality of

18.48% in severe hyponatremia patients (< 105 mmol/L Na^+) while mortality in males with severe hyponatremia was 57.14%.

Discussion :

Heart failure patients frequently present with hyponatremia as a part of water retention and oedema. This study shows higher prevelance of this biochemical abnormality (51.38%) in patients presenting with CCF. However, these patients tolerate it better upto 120 mmol/L with minimal symptoms attributable to hyponatremia. The prevalence is more in males as compared to females. This reflects the sex distribution of CCF patients presenting in critical care unit. Chronic hyponatremia is common in postmenopausal women. However, in those patients having CCF as presenting syndrome male dominance is seen. Sodium concentration less than 135 mmol/L was associated with increased 30 day mortality or recurrence of myocardial infarction in non ST segment elevation myocardial infarction (NSTEMI). Similarly, risk of death and heart failure are higher among ST segment elevation myocardial infarction (STEMI) patients who had hyponatremia on admission. Activation of arginine vasopressin (AVP) may impair water excretion and result in dilutional hyponatremia in STEMI patients with acute left ventricle (LV) dysfunction.

Morality rates have been reported in wide range from 33% to 86% in elderly patients and 20% in an Indian report by Rao MY. However, our study reports 8.38% mortality rates in CCF patients with hyponatremia. Previous mortality data in hyponatremia has included syndrome of inappropriate antidiuretic hormone secretion (SIADH), cirrhosis and renal causes also. In fact, MY Rao et al reported SIADH and old age (> 72 yrs) as major demographic factors in their group and hence higher mortality. The present study focused only heart failure patients with hyponatremia where mortality is lower as compared to other studies possibly because mean age was less (younger population) and slow development of chronic hyponatremia in CCF patients is better tolerated with fewer symptoms and less mortality as compared to other group containing older patients and SIADH patients. Severe hyponatremia less than 105 mmol/L has very high mortality rates (57.14%) and seizures were seen in 8 cases (50% of those who succumb to hyponatremia).

Drug induced hyponatremia needs special mention here. Many studies have documented thiazide diuretics as a major cause. In present study two different molecules of diuretics were used in 30.70% cases. All of them received furosemide by intravenous route. Co-existing hypokalemia (7.10%) also complicates the course of the disease. Though joint national committee (JNC) VII recommended diuretics as first line of drugs for treatment of hypertension, a word of caution is expressed here because of frequent occurrence of hypernatremia following its indiscriminate usage. CCF patients continue to receive diuretics however, dose modification or withdrawal of such drugs may be done when systemic congestion comes down to bare minimum levels and angiotensin converting enzyme (ACE) inhibitors reach up titration to their optimal recommended dosages.

This study does not look into the methods of correction of hyponatremia and the responses to treatment. It highlights very common association of dilutional hyponatremia in CCF and emphasizes more usage of vasodilators like ACE inhibitors and minimum use of diuretics because diuretics may add to the burden of hyponatremia in CCF. In Congestive Cardiac

Failure (CCF), higher mortality is expected due to ongoing disease severity or possibly due to mismanagement.

Further studies are needed with controlled prospective design so as to include all severity grades of hyponatremia patients to find out mortality rates linked with severity of hyponatremia, specific subsets of cirrhosis, CCF etc.

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