STUDY OF INCIDENCE OF HYPONATRAEMIA IN EMERGENCY SURGICAL PATIENTS AND ITS MANAGEMENT

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ABSTRACT

INTRODUCTION

Disorders of sodium and water metabolism are common in hospitalised patients and are occasionally encountered in outpatients. Both hyponatraemia and hypernatraemia can cause substantial morbidity and mortality, and ironically, incorrect treatment can add to the problem. But hyponatraemia is the most commonly seen electrolyte abnormality in surgical wards. The symptoms of hyponatraemia vary from anorexia, headache, nausea, vomiting and lethargy to convulsion, coma and death. Treatment of hyponatraemia must be individualised considering the aetiology, rate of development, severity and clinical signs and symptoms.

PURPOSE

1. The study was aimed to find the incidence of hyponatraemia in emergency surgical patients, to correlate the impact of hyponatraemia in morbidity and mortality.
2. To diagnose hyponatraemia in patients early and proper management to avoid the unwanted complication.

MATERIALS AND METHODS

The present study was carried out in 72 emergency surgical patients admitted to Department of General Surgery of M.K.C.G Medical College and Hospital, Berhampur, during the period from September 2013 to August 2015.

RESULTS

1. During the period of study, 16 cases (21.81%) were detected of hyponatraemia from 72 patients in their hospital stay.
2. 4 female (23.53%) patients developed hyponatraemia and 12 male (22.22%) patients developed hyponatraemia during their hospital stay.
3. Out of 72 cases, 10 cases (13.89%) developed hyponatraemia in preoperative period. 6 cases (8.33%) developed hyponatraemia in postoperative period.
4. 14 cases (87.5%) manifested with symptoms of altered mental status. 7 cases (43.7%) manifested with nausea and vomiting. 10 cases (62.5%) presented with headache. Only 3 cases (18.7%) had convulsion due to hyponatraemia.
5. In the age group 0-20, no cases of hyponatraemia was observed while in the older age group (60-80) we observed 5 cases (50%) out of 10. In the age group 40-60, (25.00%) and in the age group 20-40, (13.33%) of cases developed hyponatraemia in their hospital.
6. 6 cases (2 females and 4 males presented with mild hyponatraemia (37.50%). 4 cases (1 female and 3 males) presented with moderate hyponatraemia (25.00%) and 6 cases (1 female and 5 males) found to have severe hyponatraemia out of total 16 cases (37.50%).
7. Out of 16 cases, 11 cases were grouped into hypovolaemic hyponatraemia and 5 cases into euvolaemic hyponatraemia according to their extracellular volume status.

KEYWORDS

Hyponatraemia, Hypernatraemia.

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INTRODUCTION: Disorders of sodium and water metabolism are common in hospitalised patients and are occasionally encountered in outpatients. Both hyponatraemia and hypernatraemia can cause substantial morbidity and mortality, and ironically, incorrect treatment can add to the problem. But hyponatraemia is the most commonly seen electrolyte abnormality in surgical wards. Although most of the studies show the incidence of about 1-2% in hospitalised patients but in the surgical ward, approximately 4.4% of postoperative patients developed hyponatraemia within 1 week of surgery. Hyponatraemia has also been observed in approximately 30% of patients treated in the intensive care unit (Upadhyay A, Jaber BL, Madias NE 2006).[1] Although hyponatraemia is very common

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in hospitalised patients, but is rarely seen in an ambulatory patient (if present, reflects a chronic disease status).

Hyponatraemia is defined as a decrease in the serum sodium concentration to a level below 136 mmol per litre (NEJM 2000).[2] Whereas hypernatraemia always denotes hypertonicity, hyponatraemia can be associated with low, normal, or high tonicity (Griinfeld J.P 1998).[3] As sodium is the major ECF cation and 80-90% is extracellular, it is responsible for more than 90% of total osmolality of extracellular fluid. So ECF volume is a reflection of total body sodium content. Hyponatraemia can be caused either by a solute driven shift of fluid from cell to the extra cellular compartment or by water ingestion in excess of the ability to excrete it. In the former setting, the osmolality of the body fluid is typically elevated despite the presence of hyponatraemia. In the latter setting, water retention causes a dilution body solute and a reduction in osmolality that is proportional to the severity of the hyponatraemia.

Hyponatraemia is physiologically significant when it indicates a state of extracellular hypoosmolarity and a tendency for free water to shift from the vascular space to the intracellular space. Although cellular oedema is well tolerated by most tissues, it is not well tolerated within the rigid confines of the bony calvarium. Therefore, clinical manifestations of hyponatraemia are related primarily to cerebral oedema. The rate of development of hyponatraemia plays a critical role in its pathophysiology and subsequent treatment. When serum sodium concentration falls slowly, over a period of several days or weeks, the brain is capable of compensating by extrusion of solutes and fluid to the extracellular space. Compensatory extrusion of solutes reduces the flow of free water into the intracellular space, and symptoms are much milder for a given degree of hyponatraemia.

When serum sodium concentration falls rapidly, over a period of 24-48 hours, this compensatory mechanism is overwhelmed and severe cerebral oedema may ensue, resulting in brainstem herniation and death. Hyponatraemia is clinically important entity because acute, severe hyponatraemia has substantially high morbidity and mortality. Rapid correction of chronic hyponatraemia can lead to neurological deficit and even death. And lastly the aetiology and treatment is not as simple as the other electrolyte deficit. A common understanding is that the deficit should be treated with supplementation but in case of hyponatraemia the treatment may be contrary to the common understanding. Serum sodium reflects the relative proportion of sodium and water. So basically hyponatraemia can be dilutional (water excretion lesser than the water intake and so needs fluid restriction as the most important treatment or due to sodium loss (needs sodium and fluid replacement).

The severity of symptom depends upon the severity of hyponatraemia and the rate at which the plasma sodium concentration is lowered. So acute and severe hyponatraemia is symptomatic, but chronic and mild hyponatraemia is well tolerated. The symptoms of mild hyponatraemia are anorexia, headache, nausea, vomiting and lethargy. In case of moderate hyponatraemia it varies from personality changes, muscle cramps, confusion and ataxia where as in severe hyponatraemia the symptoms are devastating ranging from drowsiness to convulsion, coma and death.

The diagnosis of the condition is solely based on history, physical examination and laboratory investigation. History and physical examination is often helpful in identifying hypovolaemic hyponatraemia (diarrhoea, vomiting, burns, diuretics, etc.). The degree of hyponatraemia often correlates with the severity of the underlying condition and is an important prognostic factor. The treatment of hyponatraemia can be divided into two steps. First, the surgeon must decide whether immediate treatment is required. This decision is based on the presence of symptoms, the degree of hyponatraemia, whether the condition is acute (arbitrarily defined as a duration of less than 48 hours) or chronic, and the presence of any degree of hypotension. The second step is to determine the most appropriate method of correcting the hyponatraemia.

Acute severe hyponatraemia usually is associated with neurological symptoms such as seizures and should be treated urgently because of the high risk of cerebral oedema and hyponatraemic encephalopathy.

In patients with chronic hyponatraemia, overzealous and rapid correction should be avoided because it can lead to central pontine myelinolysis. In most cases of chronic asymptomatic hyponatraemia, removing the underlying cause of the hyponatraemia suffices. Otherwise, fluid restriction is the mainstay of treatment and the preferred mode of treatment for mild-to-moderate SIADH.

Treatment of hyponatraemia must be individualised considering the aetiology, rate of development, severity and clinical signs and symptoms. The dictum is that hyponatraemia, which develops quickly, should be treated fast; whereas hyponatraemia, which develops slowly, should be corrected slowly. The goal of the therapy is to raise the plasma sodium concentration at a safe rate to replace the sodium deficit and correcting the underlying cause. In general, hyponatraemia is corrected acutely by giving sodium to patients who are volume depleted and by restricting water intake in patients who are normovolaemic or oedematous.

This study has been done to know the incidence of hyponatraemia in emergency surgical patients and the proper management of the condition according to the aetiology by correcting the sodium and volume status of the body.

MATERIALS AND METHODS: The present study was carried out in 72 emergency surgical patients admitted to Department of General surgery of M.K.C.G Medical College and Hospital Berhampur, during the period from September 2013 to August 2015.

In this prospective study, 72 cases were selected randomly, both from preoperative and postoperative settings. Patients who presented with features of dehydration, altered mental status, nausea and vomiting, headache and convulsion are taken into study group. In all these patients, serum sodium concentration was measured. Only emergency cases were taken into consideration.

In all patients, general information regarding age, sex, weight, history, physical examination and laboratory investigation, surgical procedure or treatment modalities are noted.

By definition, those patients present with serum sodium concentration less than 136 mmol were considered hyponatraemic. The serum sodium level was measured by automated ion electrode method in all patients. Other routine blood tests and serum potassium level also recorded during the study. The symptoms of hyponatraemia such as nausea, vomiting, seizures, and changes in sensorium were noted. According to the level of serum sodium concentration the patients having symptomatic hyponatraemia were divided into mild, moderate and severe hyponatraemia. During the hospital stay the types of intravenous fluid therapy also noted.

First the patients are divided into hypotonic or hypertonic hyponatraemia by using the formula: Effective Serum Osmolality=(2XNa)+(glucose÷18) measuring the serum sodium level, and blood sugar level.

Patients with hypotonic hyponatraemia divided into hypervolemia, hypovolemic and euvolaemic according to their volume status by clinical observation. Measurement of urine sodium in mmol/L was done for differential diagnosis in all hyponatraemic patients.

The patients presented with features of acute hyponatraemia (according to definition less than 48 hours of hospital stay). The patients were managed according to aetiology. Hyponatraemic patients were managed with infusion of normal saline 0.9% and 3% according to the need. The sodium requirement was calculated using the formula:

Change in serum Na^+= [Infusate sodium–Serum sodium]/ [Total body water +1].

Total body water in litres is calculated as a fraction of body weight and the fraction is 0.6 in children; 0.6 and 0.5 in adult men and women respectively and 0.5 and 0.45 in elderly men and women respectively.

During management serum sodium concentration was not raised more than 8 mmol/day. Patients with convulsion managed with anticonvulsant in addition to the replacement of sodium containing fluid. Serum sodium level was measured 12 hourly in severe hyponatraemic patients and 24 hourly in mild and moderate hyponatraemic patients. The time period required for normalisation of serum sodium was also noted during the study. The complication during the therapy also noted.

The results obtained from study is analysed and compared with previous observation by other workers.

The summary and conclusion were drawn about the correlation between the incidence, clinical finding, laboratory findings, aetiology and modality of management in the hyponatraemic patients.

Exclusion Criteria:

1. Paediatric patients.
2. Routine surgical cases.
3. Patient refusing to take part in study.

STATISTICS AND RESULTS: 72 cases of emergency surgical patients admitted to the Department of General Surgery, M.K.C.G Medical College in the period of September 2013 to August 2015 were studied for presence of hyponatraemia both in preoperative and postoperative period.

Sex	No. of cases	Cases developed hyponatraemia	Percentage
Male	55	12	21.81
Female	17	4	23.53
Total	**72**	**16**	**22.22**

Table 1: Sex distribution

During the period of study, 16 cases (21.81%) detected hyponatraemia from 72 patients in their hospital stay. All of them developed acute hyponatraemia. As shown in the table -1 and chart 1, 4 female (23.53%) patients developed hyponatraemia and 12 male (22.22%) patients developed hyponatraemia during their hospital stay.

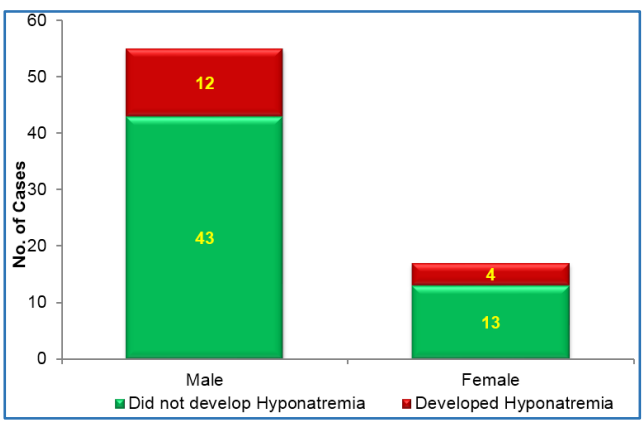

Chart 1

Developed hyponatraemia	No. of cases	Percentage
Preoperative period	10	13.89
Postoperative period	6	8.33
Total	**16**	**22.22**

Table 2: Pre and post-operative distribution

(n=16)

As shown in the table 2, out of 72 cases 10 cases (13.89%) developed hyponatraemia in preoperative period. 6 cases (8.33%) developed hyponatraemia in postoperative period.

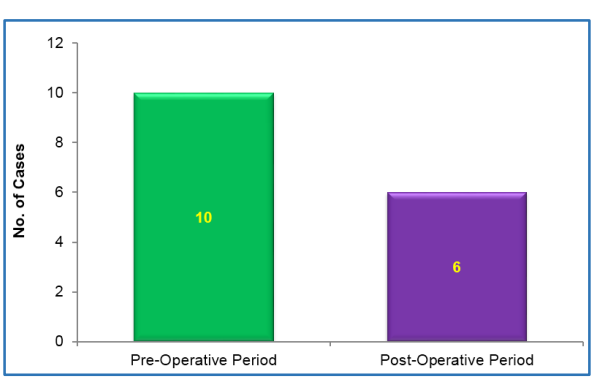

Chart 2: Distribution of pre & post-operative

The 16 hyponatraemic patients presented with different clinical features as shown in the table 3.

Clinical feature	No. of cases	Percentage
Feature of altered mental status	14	87.5
Headache	10	62.5
Nausea and vomiting	7	43.7
Convulsion	3	18.7

Table 3: Clinical features

As shown in the table 3, 14 cases (87.5%) manifested with symptoms of altered mental status and it was the most common clinical manifestation in hyponatraemic patients. 7 cases (43.7%) manifested with nausea and vomiting. 10 cases (62.5%) presented with headache. Only 3 cases (18.7%) had convulsion due to hyponatraemia.

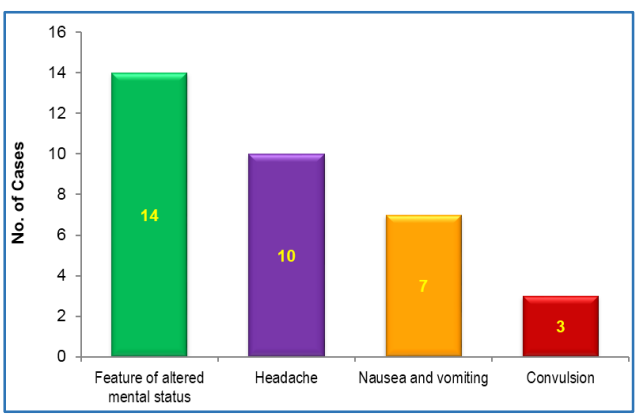

Chart 3: Clinical features

As shown in table 4 from the 14 patients presented with altered mental status 10 cases presented with altered sensorium, 1 case with feature of agitation, 1 case with lethargy and 2 cases developed frank psychosis.

Altered mental status	No. of cases	Percentage
Disorientation	10	71.42
Agitation	1	7.14
Lethargy	1	7.14
Psychosis	2	14.28
Total	**14**	**100.00**

Table 4: Distribution of altered mental status

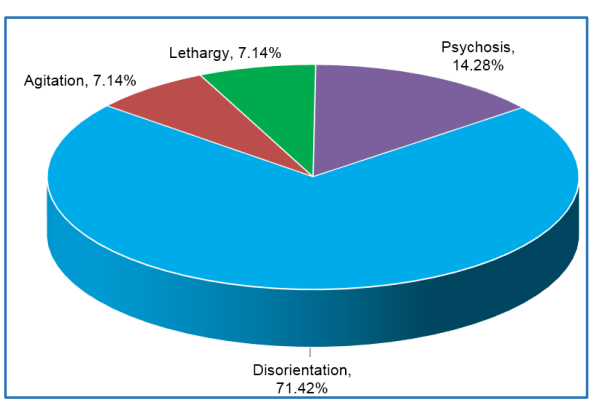

Chart 4: Distribution of altered mental status

According to our study the relation of age to hyponatraemia is shown in table 5.

Age group	Total no. of cases	No. of cases developed hyponatraemia	Percentage
0-20	4	0	0.00
20-40	30	4	13.33
40-60	28	7	25.00
60-80	10	5	50.00

Table 5: Distribution of age group

In the age group 0-20, no cases of hyponatraemia was observed while in the older age group 60-80 we observed 5 cases (50%)out of 10. So the maximum number of cases of hyponatraemia found in the older age group. In the age group 40-60, (25.00%) and in the age group 20-40, (13.33%) of cases developed hyponatraemia in their hospital stay.

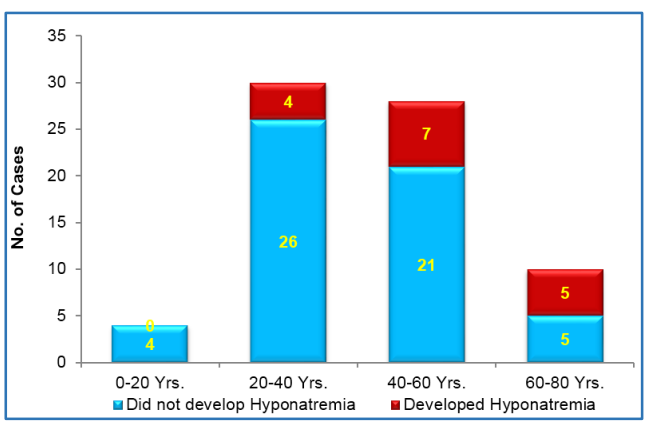

Chart 5: Distribution of age group

In the study, according to the level of serum sodium concentration cases are divided into mild, moderate and severe as shown in table 6.

	Male	Female	Total	Percentage
Mild hyponatraemia (128-135) mmol/L	4	2	6	37.50
Mod hyponatraemia (120-127) mmol/L	3	1	4	25.00
Severe hyponatraemia (<120) mmol/L	5	1	6	37.50

Table 6: Sex wise distribution of hyponatraemia according to severity

As shown in table 6 and Chart 6, 6 cases (2 females and 4 males presented with mild hyponatraemia (37.50%). 4 cases (1 female and 3 males) presented with moderate hyponatraemia (25.00%) and 6 cases (1 female and 5 males) found to have severe hyponatraemia out of total 16 cases (37.50%).

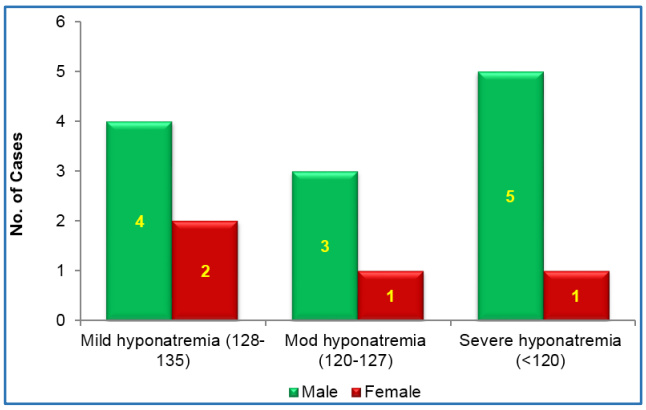

Chart 6: Sex wise distribution of hyponatraemia according to severity

We found low serum osmolality (below 280 mOsmol/L) in all cases. We did not encounter with a case of either hypertonic hyponatraemia or normotonic hyponatraemia. All of our studied cases were found to be hypotonic hyponatraemia. Out of 16 cases, 11 cases were grouped into hypovolaemic hyponatraemia and 5 cases into euvolaemic hyponatraemia according to their extracellular volume status as shown in chart 7 and table 7.

Type of hypotonic Hyponatraemia	No. of cases	Percentage
Euvolaemic Hyponatraemia	5	31.25
Hypovolaemic Hyponatraemia	11	68.75
Hypervolaemic Hyponatraemia	0	0.00
Total	**16**	**100.00**

Table 7: Type of hypotonic hyponatraemia

During the period of study, we observed patients with following diseases developed hyponatraemia during the hospital stay in the preoperative period and all detected having hypovolaemic hypotonic hyponatraemia.

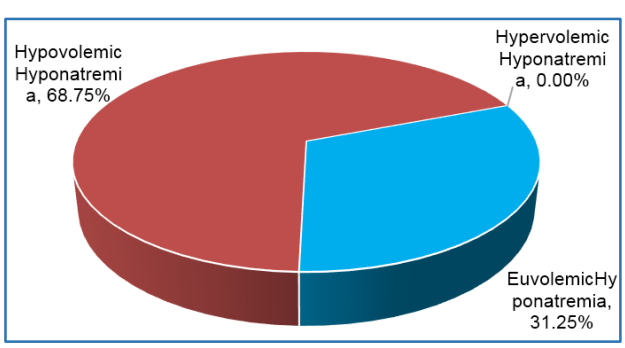

Chart 7: Types of hypotonic hyponatraemia

Disease	No. of cases developed hyponatraemia	Cause
Intestinal obstruction small intestinal obstruction-3, Sub acute intestinal obstruction-1 Sigmoid volvulus-1	5	Third space fluid loss and vomiting
Diseases with third space fluid loss peptic perforation-2 Small intestinal perforation-1	3	Third space fluid loss
Disease with severe vomiting Gastric outlet obstruction-1, obstructive jaundice-1	2	Severe Vomiting
Total	**10**	

Table 8: Preoperative case distribution

As shown in the table 8, from 10 cases we found 5 cases hypovolaemic hyponatraemia due to intestinal obstruction. 3 cases due to gut perforation and another two cases due to severe vomiting. Out of 10 cases, 10 cases developed hyponatraemia due to gastro intestinal fluid loss into third space or externally had urine Na+ less than 20 mmol/L.

Disease	No. of cases	Cause
Brain injury (SDH)	2	Hypotonic fluid administration
Postop CDU (after GJ)	1	Vomiting and Nasogastric aspiration.
Postop obstructed Hernia	1	Hypotonic fluid administration.
Postop appendicectomy	2	Hypotonic fluid administration
Total	**6**	

Table 9: Post-operative distribution

As shown in the table 8, we found two cases of brain injury, one case of postoperative obstructed indirect inguinal hernia, two cases of postop appendicectomy developed hyponatraemia due to hypotonic fluid administration. 1 cases CDU (truncal vagotomy and gastrojejunostomy was done) in the postoperative period developed hyponatraemia due to vomiting and nasogastric aspiration.

Serum sodium	Serum osmolality	Type of hyponatraemia	Days of recovery	Replacement done by
128	261	Mild	3	NS
130	266	Mild	1	NS
130	266	Mild	1	NS
128	261	Mild	2	NS
129	252	Mild	2	NS
132	266	Mild	1	NS
123	256	Moderate	3	NS

122	249	Moderate	2	NS
126	257	Moderate	2	NS
124	253	Moderate	2	NS
118	240	Severe	5	3%S, NS
118	240	Severe	3	3%S, NS
116	237	Severe	4	3%S, NS
119	244	Severe.	3	3%S, NS
112	243	Severe	4	3%S, NS
112	230	Severe	3	3%S, NS

Table 10: Relationship with serum sodium and serum osmolality

In table 10 the relationship between serum sodium with serum osmolality, type of hyponatraemia, days of recovery and the replacement therapy has been correlated. We found that the severity of the condition depends upon the level of serum sodium and serum osmolality. Patients having lower serum sodium concentration, and lower serum osmolality developed severe hyponatraemia which leads to increase in the days of recovery as shown in the chart 7.

The mean duration of recovery from hyponatraemia was 2.6 days in all type of patients. In 6 patients the replacement of sodium was done initially with 3% saline by increasing the serum sodium concentration not more than 8 mmol/day then they were infused with NS after Na+ reached above 120 mmol/L. In 10 patients, replacement was done with 0.9% normal saline. During the period of study, we found one case of death in patient of brain injury with hyponatraemia. But the cause of mortality (6.66%) was not attributed to hyponatraemia but to the underlying illness.

DISCUSSION: The present discussion is based on clinical, evaluation of 72 emergency surgical patients admitted to Department of General Surgery, M.K.C.G. medical College Hospital during the period of study in relation to the incidence of hyponatraemia and its modality of management in them.

Table 1 shows the incidence of hyponatraemia is 22.22 % (16 cases) of total 72 cases during their hospital stay in the surgical wards. During the preoperative period the incidence of hyponatraemia was 13.89% (10 cases) out of total 72 cases. During the postoperative period the incidence was 8.33% (6 cases) out of 72 cases, we studied. Eric E Simon, MD, reported a hospital incidence of 15-20%. Upadhyay A, Jaber BL, Madias NE in 2006 reported incidence of 30% in the intensive care unit and 4.4% in postoperative patient within 1 week of surgery. Hawkins RC in 2003 reported 42.6% of patients in a large acute care hospital in Singapore.[4] Hoorn E, Lindemans J, ZietseR. in 2004 found 30% patients developed hyponatraemia in an acute care setting in Rotterdam.[5] Zada et al reported the incidence of hyponatraemia about 5% in postoperative patients after transsphenoidal pituitary surgery.[6] In a prospective study by Chung H-M et al reported the incidence of hyponatraemia in postoperative patients was approximately 4 in the year 1989.[7] In a prospective study by Olson BR concluded that 25% of the patients developed spontaneous isolated hyponatraemia in their postoperative period after pituitary surgery in the year 1998.

We found slightly high female predominance of 23.53% (4 cases) out of 16 cases as shown in table 1. In the males, the incidence found as 21.82% (12 cases) out of 16 cases as shown in table 1. But the male to female ratio was almost 1:1 EricESimonMD 2006 had reported, hyponatraemia affects all races and no sexual predilection exists. Sandy Craig, MD in 2006 and Trung Q Pham, MD in 2007 concluded that the incidence of hyponatraemia was equal in males and females.[8] Ayus and colleagues in 1987 had reported higher incidence of postoperative hyponatraemia in menstruating woman.[9] Areif had reported higher incidence of postoperative hyponatraemia in female who had undergone routine surgery without significant blood loss in 1986.[10] Zada G et al concluded that, female patients were more likely to develop hyponatraemia than male patients (33% compared with 22%, $p < 0.03$) in the year 2007.

In the study, we observed maximum incidence of hyponatraemia (50%) in the older age group (60-80) as shown in table 4. No case of hyponatraemia found in young age group (0-20). In the age group of 40- 60 yrs., 25% and in the age group of 20-40 yrs., 13.33% of cases developed hyponatraemia in their hospital stay. During the study, we also observed that the incidence of hyponatraemia increases with age. Eric E Simon MD noted, higher incidence of hyponatraemia in elderly population because of the increased comorbid conditions (e.g. cardiac, hepatic, or renal failure) in 2006. But Trung Q Pham, MD in 2007 reported the group commonly affected by hyponatraemia was elderly and young population. People typically affected are aged 59-83 years. Tambe AA et al in 2003 found significant risk of hyponatraemia following orthopaedic surgery, especially in the elderly.[11]

Table 3 shows the clinical manifestation of the hyponatraemic patients. In this study out of 16 hyponatraemic patients 87.5% patient manifested with altered mental status, the most common mode of presentation we observed. Out of 14 cases with altered mental status, 10 cases presented with disorientation, 1 case with agitation, 1 case with lethargy and 2 cases developed frank psychosis. 7 cases (43.7%) manifested with nausea and vomiting. 10 cases (62.5%) presented with headache. Only 3 cases (18.7%) had convulsion due to hyponatraemia. We also observed severity of symptoms directly proportional to the degree of hyponatraemia as shown in table 9. We observed severe neurological symptoms in patient when serum sodium level falls below 120 mmol/L. Adrogue H in 2000 described that the symptoms are related largely to dysfunction of the central nervous system and are more evident when the decrease in the serum sodium concentration is large or fast Janicic N, Verbalis JG in 2003 reported that most patients with a serum sodium concentration greater than 125 mEq/L or with chronic hyponatraemia did not have neurological symptoms, owing to volume adaptation by the brain.[12] SchrierRW, in 2000 reported that, gastrointestinal symptoms, such as nausea and vomiting, were more common in patients with serum

sodium levels between 125 and 130 mEq/L. He noted predominance of neurological symptoms when the serum sodium level went below 125 mEq/L.[13] The symptoms like headache, muscle cramps, reversible ataxia, psychosis, lethargy, restlessness, disorientation, apathy, anorexia, and agitation were seen in patients with serum sodium levels below 125 mEq/L. Nzerue CM et al in a study reported altered sensorium (51%), seizure (22%), nausea and vomiting (4.8%) and comatose state (2.3%) in severely hyponatraemic patients in year 2003.[14]

Table 5 shows distribution of 16 patients of hyponatraemia into mild (Na+ 128-130 mEq/L), moderate (Na+ 120-127 mEq/L) and severe (Na+ below 120 mEq/L). In the study cases (37.50%) presented with mild hyponatraemia. 4 cases (25%) presented with moderate hyponatraemia and 6 cases (37.50%) found to have severe hyponatraemia out of total 16 cases. Madiba et al in the year 1996 in a prospective study found (hyponatraemia was defined as a serum sodium level of <130 mmol/L.) the incidence of hyponatraemia was 2.2%, the most common type being normovolaemic hypotonic hyponatraemia. Hyponatraemia was either mild (sodium level 120-130 mmol/l) or moderate (111-120 mmol/L). No patient had severe hyponatraemia (<110 mmol/L).

In our study, the serum osmolality in all hyponatraemic patients was below 280 mOsmol/L. We did not observe any case of hypertonic hyponatraemia or pseudohyponatraemia. During the study, the serum sodium level was measured using automated ion electrode method, so no case of pseudohyponatraemia was observed. The cases of hyponatraemia we observed, all were hypotonic hyponatraemia. Out of 16 cases, 11(68.75%) cases were hypovolaemic hyponatraemia and 5(31.25%) cases were euvolaemic hyponatraemia as shown in table 6. 9 cases of observed hypovolaemic hyponatraemics were detected during the preoperative period and 2 cases in the postoperative period. 5 cases of euvolaemic hyponatraemia found in the postoperative cases. Callewart CC et al in 1994 in a prospective study in spine surgery patients reported that, hyponatraemia developed in 45 (44.6%) patients.[15] The aetiology of hyponatraemia was the syndrome of inappropriate antidiuretic hormone secretion in seven patients (6.9%), hypovolaemia in 19 patients (18%), and other causes in six patients. Chung HM et al in the year 1986 reported that, in patients with postoperative hyponatraemia (plasma sodium concentration less than 130 mEq/L) most patients (42%) were normovolaemic, oedematous states (21%), hyperglycaemia (21%), volume depletion (8%), and renal failure (8%) of cases.

So the most common type we observed was hypovolaemic hyponatraemia. Intestinal obstruction was the most common cause of hypovolaemic hyponatraemia. We observed (5 Cases) 31.25% of total 16 cases of hyponatraemia were due to intestinal obstruction, which was also the most common cause of preoperative hyponatraemia. In postoperative period, the most common cause of hyponatraemia was postoperative hypotonic fluid administration. Which was also the most common cause of hyponatraemia (31.25%) of 16 hyponatraemic cases overall. According to Bailey and Love's short practice of surgery (24th ed), the most frequent cause of hyponatraemia seen in surgical practice is obstruction of small intestine, with rapid loss of gastric, biliary, pancreatic and intestinal secretions by antiperistalsis and ejection, whether by vomiting or aspiration.[16] But Chung HM et al in the year 1986 concluded that hyponatraemia commonly occurs following all types of surgical procedures and was due to hypotonic fluid administration in the presence of nonosmotic secretion of arginine.

During the treatment of hyponatraemia, the mean days of recovery from hyponatraemia were 2.6 days in all type of patients. In 6 patients, the replacement of sodium was done initially with 3% saline by increasing the serum sodium concentration not more than 8 mmol/day, then they were infused with NS after Na+ reached above 120 mmol/L. In 10 patients, replacement was done with 0.9 normal saline. In both type of replacement therapy, mortality was found to be 6.25%. This was a patient of brain injury and the mortality was due to the underlying illness not due to hyponatraemia. Madiba et al in the year 1996 reported that hyponatraemia was corrected within 1-6 days using normal saline; in 73% of patients it was corrected within 24 hours. A mortality rate of 28% was attributed to underlying illness. Vachharajani in 2003 concluded that in acutely symptomatic hyponatraemia, the serum sodium should be raised by approximately 1-2 mEq/L/h for 3-4 hours until the neurological symptoms subsided or until plasma Na+was over 120 mEq/L.[17] Decaux et al in 2003 treated acute symptomatic hyponatraemia in the postoperative period with hypertonic saline (3%) to prevent seizures and respiratory arrest. They increased serum sodium concentration below 10 mmol/L per day to avoid permanent neurological damage.[18]

CONCLUSION: The study comprising of 72 cases of emergency surgical patients admitted into Department of General Surgery, M.K.C.G Medical college and Hospital, during the period September 2013 to August 2015 due to various causes and belonging to either sex were evaluated for the presence of hyponatraemia by using clinical parameters and various laboratory investigation, their modality of management were correlated with disease status.

The history, clinical finding and investigation report, modality of management and the final outcome were recorded in individual case as shown in the Appendix.

Following important information were obtained from this study. During the period of study out of 72 surgical patients we studied, 16 patients found to be hyponatraemic either in the preoperative or postoperative period. The incidence is 22.22% overall. There is slightly high incidence in female patients than in male patients. But the ratio between male and female hyponatraemic patients is almost 1. So it is well distributed in both sexes.

Age is an important factor for development of hyponatraemia. The incidence and the severity of hyponatraemia increase with the age. It is more prevalent in

older surgical patients than in younger patients. In this setup, hyponatraemia is more common in the preoperative setting than postoperative setting.

A significant correlation is found between clinical manifestations of hyponatraemic patients and the level of serum sodium. Most of the patients manifest severe symptom at lower serum sodium, concentration. Also the severity of the symptoms depends on the rate of drop in serum sodium concentration and other associated comorbid condition. Although majority of the patients detected having hyponatraemia by neuropsychiatry symptoms but gastrointestinal manifestation like nausea, vomiting occur early. Most of the time early symptoms are missed by surgeons. Majority of patients manifest with altered mental, status like disorientation, agitation, lethargy and psychosis. The neuropsychiatry manifestation usually occurs when serum sodium concentration falls below 125 mmol/L. Convulsion in hyponatraemic patient develops when the serum sodium concentration falls below 120 mmol/L. In mild hyponatraemia, headache, nausea and vomiting predominate but neuropsychiatry symptoms may manifest. Moderate hyponatraemia usually manifests with altered mental status but in severe hyponatraemia half of the patient manifest with convulsion.

Although hypertonic hyponatraemia and isotonic hyponatraemia is very common in surgical setup, we have not encountered any case. In our study, all of the hyponatraemic patients belong to hypotonic hyponatraemia as TURP is not done in this setup and diabetic patients with very high blood sugar levels are managed initially in the medicinal wards. According to the volume status of the patients, majority of the patients belong to hypovolaemic group in the preoperative period. Euvolaemic hyponatraemia is only seen in postoperative setting due to infusion of hypotonic fluid as blood AVP level remains high in early postoperative period. Gastrointestinal and third space fluid loss is found to be the most common cause of hyponatraemia. From that it is seen that intestinal obstruction is the most common cause of hyponatraemia in surgical patients in overall.

For diagnosis although clinical signs and symptoms are very helpful but laboratory investigations, like serum [Na+], blood urea, blood glucose, serum osmolality and urine [Na+] are most important for classifying hyponatraemia and making their protocol of management. Blood AVP level measurement has an important role in finding the aetiology, but in our setup as there is no facility of measurement so we couldn't correlate its level and role in pathogenesis of hyponatraemia. During replacement of sodium, it is important to measure serum sodium concentration at 12 hourly when initial [Na+] is below 120 mmol/L. Once it rises above 120 mmol/L then 24 hourly measurement is required. It is seen that patients with mild hyponatraemia are managed with administration of normal saline, but patients with severe hyponatraemia, euvolaemia and in patients with brain injury require urgent correction by 3% hypertonic saline in a calculated manner and not increasing the serum sodium level above 8 mmol/day. In this way of management, we have not found any complication regarding overcorrection of serum sodium. There is a mortality rate of 6.6% of hyponatraemic patients and is not due to hyponatraemia but due to the underlying illness: In majority of hypovolaemic patients, replacement of both fluid and sodium is done by normal saline but it has been seen that patients with severe symptoms require hypertonic saline infusion.

In concluding the study, it can be mentioned that incidence of hyponatraemia is 22.22% in our setup, female has little high predominance, and severe nature of the condition is more prevalent in older age group. Most of the patients manifest with neuropsychiatry symptoms. The diagnosis is purely based on identification of early symptoms, signs and laboratory investigation. Intestinal obstruction or condition with third space fluid loss found to be the most common aetiology in preoperative state while in postoperative state hypotonic fluid administration is the most common cause of hyponatraemia. Urgent attention and watchful correction is required to avoid the serious complication during the treatment.

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