

Original research article

## Serum Electrolyte Estimation as an early Predictor for Grading Severity of Hepatic Encephalopathy

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### Abstract

**Background and objectives:** Alteration in the level of Serum Sodium, Potassium and Chloride in hepatic encephalopathy. Correlation of these levels with severity of hepatic encephalopathy due to various causes. The role of Serum Sodium, Potassium and Chloride as a predictor for grading of severity of hepatic encephalopathy.

**Methods:** Prospective observation study, All children with hepatic encephalopathy, they were admitted in paediatrics ward of Patna Medical College and Hospital, Patna, during the period of Jan 2018 to Dec-2019.

**Conclusion:** The present work is the study to evaluate changes in the Sodium electrolyte level in children suffering from hypokalaemia. The of HE consisting of 70 male children and 50 female children were included in the study group.

**Keywords:** Hypokalaemia, hepatic encephalopathy.

### Introduction

Hepatic encephalopathy is a complex neuropsychiatric syndrome characterised by disturbances in consciousness and behaviour<sup>1</sup>, personality changes, fluctuating neurological signs, asterixis or “Flapping tremor” and distractible EEG changes in a patient with liver dysfunction. Liver is the principal organ that performs a wide variety of functions<sup>2</sup>. It maintains Carbohydrate, Protein and fat metabolism and has a dominant role in synthesis of coagulation factors, Protein and Enzymes<sup>3</sup>. Liver also attributed with a significant role in the maintenance of electrolyte metabolism. It is evident that there is hyponatraemia. It is evident that there is hyponatraemia in patient of hepatic encephalopathy<sup>4</sup>, the relation of these changes in serum sodium, Potassium and Chloride should be known and established. My study provides information about the changes in serum electrolytes could serve as a poor prognostic index but there was no

correlation between serum electrolytes changes in patients of hepatic encephalopathy during different stages of hepatic encephalopathy<sup>5</sup>.

### **Objectives**

\*Correlation of these levels with severity of hepatic encephalopathy due to various causes.

\*The role of Serum Sodium, Potassium and Chloride as a predictor for grading of severity of hepatic encephalopathy.

### **Materials and methods**

This is Prospective observational study, All children with hepatic encephalopathy they were admitted in Paediatric ward of PMCH, Patna during the period of Jan 2018- Dec 2019. Since, PMCH is the best Post Graduate College in eastern side of India; it caters the whole of Bihar and adjacent areas. So, my study will reflect the current status of hepatic encephalopathy in our state.

### **Inclusion criteria**

All children with clinical and biochemical evidence of liver dysfunction having neuropsychiatric changes were included.

### **Exclusion criteria**

Cases with meningioencephalitis and other causes of encephalopathy will be excluded.

Materials and method included\*General information about patient

\*Chief complaints

\*History of presenting illness

\*History of precipitating factors

\*Past history, family history, immunisation history

\*Anthropometry

\*General Physical examination

\*Signs of liver failure, Systemic examination include gastro intestinal system, central nervous system, cardio vascular system, Respiratory system

### **Lab Parameters included:-**

\*Haemoglobin percentage, TC, DC, ESR, Platelets and PBS

\*Blood urea, serum creatinine, RBS

\*TSP, albumin, bilirubin, AST, ALT and alkaline phosphate

\*PT, Serum ceruloplasmin

\*Viral marker. HBSAg, Anti HCV, IgM HAV, IgM HEV

\*Lapto spira serology, if needed

\*Blood culture, Routine Urine examination, Ascitic fluid study, Chest x-ray, USG whole abdomen

\*ABG

### **Statistical Formula employed:-**

\*Standard deviation

\*Standard error of mean

## Results

Observation on Serum Sodium, Potassium and chloride out in 50 healthysubject (control group) and in 120 patient suffering from hepatic encephalopathy due to various etiology. The level of Serum Sodium, Potassium and Chloride were estimated in all individuals of control group. In the study group, the level of Serum Na<sup>+</sup>, K<sup>+</sup> & Cl<sup>-</sup> were estimated on the day of admission, 3<sup>rd</sup> day and 7<sup>th</sup> day of admission. In control group, mean Serum Sodium level was 140.286 ± 38.64 mmol/lit. The mean serum potassium level was 4±0.29mmol/lit. The mean Serum Chloride level was 100.20 ± 2.65 mmol/lit. In study group, mean Serum Sodium level on day 1 was 126±6.95 mmol/lit and on day 3 was 128±6.95mmol/lit and on day 7 was S.Na<sup>+</sup> level was 134.13 ± 7.14 mmol/lit. S.Na<sup>+</sup> level highly signifies full p value < 0.001 on day 1, 3 and 7. In study group Potassium level on day 1 was 3±0.79 mmol/lit, on day 3 was 3.27±0.81 mmol/lit and on day 7 S.potassium level was 3.58 ± 0.87 mmol/lit. P value on day 1 & 3 (0.001) and on day 7 (P value 0.01). In study group mean serum chloride level on day 1 was 98.74 ± 5.99 mmol/lit, on day 3 was 100.25±4.86mmol/lit and on day 7 serum chloride level was 101.11±5.29 mmol/lit. Non significant fall of serum chloride on day 1, 3 & 7 (P value > 0.05)

**Table 1: Showing statistical evaluation of changes in the level of serum sodium in study group with that of control group**

Serum Na <sup>+</sup> in m mol/L in control group			Study Group	Days	Serum Sodium in m mol/L in study group			Z Value	P Value	Remark
Mean	S.D	S.E.M			Mean	SD	S.E.M			
140.02	±2.86	±0.404		1 <sup>st</sup> day	126.10	±6.95	±0.634	13.66	<0.001	Highly Significant
				3 <sup>rd</sup> day	128.70	±6.95	±0.685	11.09	<0.001	Highly Significant
				7 <sup>th</sup> day	134.13	±7.14	±0.744	5.64	<0.001	Highly Significant

In control group, the mean serum sodium level is 140.02mmol/L. In study group the mean serum sodium level on day 1 is 126.10mmol/L, on day 3 is 128.70mmol/L and on day 7 is 134.13mmol/L.

**Table 2: Showing statistical evaluation of changes in the level of Serum Potassium in study group with that of control group**

Serum k <sup>+</sup> in m mol/L in control group			Study Group	Days	Serum potassium in m mol/L in study group			Z Value	P Value	Remark
Mean	S.D	S.E.M			Mean	SD	S.E.M			
4.0	±0.292	±0.084		1 <sup>st</sup> day	3.00	±0.794	±0.115	8.70	<0.001	Highly Significant
				3 <sup>rd</sup> day	3.27	±0.816	±0.104	6.10	<0.001	Highly Significant
				7 <sup>th</sup> day	3.58	±0.876	±263	3.27	<0.01	Significant

In control group, the mean serum potassium level is 4.00mmol/L. In study group the mean serum potassium level on day 1 is 3.00mmol/L, on day 3 is 3.27mmol/L and on day 7 is 3.58mmol/L.

**Table 3: Showing statistical evaluation of changes in the level of Serum Chloride in study group with that of control group**

Serum Cl <sup>-</sup> Z,n in m mol/L in control group			Study Group	Days	Serum Cl <sup>-</sup> in m mol/L in study group			Z Z Z Z- Value	P P- P- Value	Remark
Mean	S.D	S.E.M			Mean	SD	S.E.M			
100.20	±2.65	±0.375		1 <sup>st</sup> day	98.74	±5.99	±0.547	1.65	>0.05	Not Significant
				3 <sup>rd</sup> day	100.25	±4.87	±0.480	0.06	>0.05	Not Significant
				7 <sup>th</sup> day	101.11	±5.29	±0.551	1.16	>0.05	Not Significant

In control group, the mean serum chloride level is 100.20mmol/L. In study group the mean serum chloride level on day 1 is 98.74mmol/L, on day 3 is 100.25mmol/L and on day 7 is 101.11mmol/L.

#### Survivor v/s non survivor

From the above observation, it is clear that the serum sodium and the Serum Potassium and Serum Chloride level has decreased on patient of hepatic-encephalopathy. The 67 patient who survived and recovered from hepatic encephalopathy, the level of serum Na<sup>+</sup> and K<sup>+</sup> were decreased at 1<sup>st</sup> day showed insignificant changes but patients who not survived and not recovered from hepatic encephalopathy, the level of sodium and potassium were decreased on day 1<sup>st</sup> and does not showed improvement on late phase (on day 7) in them the level of Chloride showed insignificant changes.

**Table 4: Comparison of electrolytes level in survivors and non-survivors of study group**

When Sample taken	S. Sodium level (Mean)		K <sup>+</sup>		Cl <sup>-</sup>	
	Survivor	non Survivor	Survivor	non Survivor	Survivor	non Survivor
1 <sup>st</sup> day	127.05	124.90	3.09	2.88	98.14	99.49
3 <sup>rd</sup> day	130.38	125.58	3.43	2.98	100.04	100.63
7 <sup>th</sup> day	135.85	129.52	3.75	3.11	101.52	100.04

Serum sodium level, in survivor on day 1 is 127.05 mmol/l, on day 3 is 130.38 mmol/l and on day 7 is 135.85 mmol/l. In non survivor, serum sodium level on day 1 is 124.90mmol/l, on day 3 is 125.58mmol/l and on day 7 is 129.52mmol/l. Serum potassium level, in survivor on day 1 is 3.09 mmol/l, on day 3 is 3.43 mmol/l and on day 7 is 3.75 mmol/l. In non survivor, serum potassium level on day 1 is 2.88 mmol/l, on day 3 is 2.98 mmol/l and on day 7 is 3.11 mmol/l. Serum chloride level in survivor group on day 1 is 98.14 mmol/l, on day 3 is 100.04 mmol/l and on day 7 is 101.52 mmol/l. In non survivor, serum chloride level on day 1 is 99.49 mmol/l, on day 3 is 100.63 mmol/l and on day 7 is 100.04mmol/l.

#### DISCUSSION

From the observation, it is clear that both the Serum Sodium and Serum Potassium level has decreased in patient of hepatic encephalopathy<sup>6</sup>. The size of hyponatraemia in case of hepatic failure of various etiologies, have been reported from time to time. Donald et al, Swartz et al<sup>7</sup> (1954), Artmem et al<sup>8</sup>, Nancer, Chettriatal, Parbha et al, Pride et al and Vaish Warner et al have all reported of hyponatraemia in case of hepatic failure. Prabha et al had reported hyponatraemia (S.Na<sup>+</sup> < 125 mean/LA) in 52% cases. Vaish Warner et al<sup>9</sup> observed mean serum sodium level

was 130 mean/LA. Wilkinson et al and Sheila Sherlock have reported that hyponatraemia and hypokalaemia is observed in patients of fulminant hepatic failure. Nacker, Sherlock and Ring Larson have commented on the poor prognosis of patients with hyponatraemia in hepatic failure. The mortality in such cases is as high as 82% according to them serum sodium below 130 mean/LA must be regarded as serum and if below 125 mean/LA ominous<sup>10</sup>. This hypo Na<sup>+</sup> is not amenable to treatment and reflects impending cell death rather than body sodium losses. They have also cautioned against the use of RV saline in such cases. In current study mortality is 44%, when mean serum sodium level 124 mean/LA on day 1, 125 mean/LA on day 3 and 129.52 mean/LA on day 7. In current study vomiting and GL bleeding maybe contributing factor is causing hyponatraemia. Vomiting and GL bleeding was noted 76% and 52% of cases of study group. Many hypothesis regarding change of electrolyte concentration of sodium in hepatic cellular failure have been observed by various other and may be caused by determining vomiting and desicite intake. As suggested by Donald et al it may be due to diluternaleffect. Hyponatraemia may be because of primary release of cell K<sup>+</sup> due to metabolic disturbances as result of desire process there by effecting the osmolarim of cellular system or process there by affecting may be due to alteration in of osmotic caution to total body water (Sims et al<sup>11</sup>, Jhonson et al, Narpson et al<sup>12</sup>. Talsco et al have shown that the total exchangeable sodium is increased in some patients with hyponatraemia although total body water was normal or slightly increase. The stat of v hyponatraemia in hepatic encephalopathy of various etilogies has been reported time to time. Altman et al<sup>13</sup> studied 30 patients of cirrhosis in hepatic cellular failure and hepatic coma and found hypokalemia K<sup>+</sup> in 25 patients. I.V. infusion of glucose which is usually gives as the treatment of hepatic coma also causes fall in Serum K<sup>+</sup> level. Glucose loading is well known to lower potassium apparently because potassium is needed in glycogen formation and the withdrawal from extra cellular reserves during accelerated metabolism. The mechanism of hypokalemia K<sup>+</sup> is uncertain. hypokalemia K<sup>+</sup> has been observed in patients with normal total body K<sup>+</sup> (Tofler et al<sup>14</sup>) and in the absence of -ve potassium balance (Neinman). The hypokalaemia may be due to failure of renal conservation of potassium in hepatic failure (Mandel et al<sup>15</sup>) and altered state of cellular metabolism may be cause of loss of potassium balance. However urinary loss of potassium is said to be high fulminate cell failure (Tray and Davidson). Neuro Psychiatric changes following the use of thyroids diuretics has been associated with hypokalaemia (Read et al<sup>16</sup>) correction of potassium efficiency even though diuretics was continues improved the neuro psychiatric state. Potassium deficiency increase ammonia output into renal vain (Baertle et al) and this has related to the encephalopathy, hypokalaemia is associated with alkalosis and this allow more ammonia to penetrate the blood brain barrier. Swartz et al<sup>17</sup>, foulk et al and Gosh and Kanan et al found significant, low level of serum chloride in causes of hepatic coma various etiologies. But Sherlock et al in an observation of the complications of diuretics therapy in In this study, patients with hypokalaemia does not showed significant fall of chloride level, the difference between our study and previous study may be due to small sample size and previously with different types of fluid.<sup>18</sup> In consideration of above discussion it is clear that hypokalaemia is observed in HE. These eleclyte changes are related to the liver function and could serve as poor prognosis index.

### Conclusion

The present work is the study to evaluate changes in the Sodium electrolyte level in children suffering from hypokalaemia. The of HE contesting of 70 male children and 50 female children were included in the study group. They were admitted in paediatric ward of PMCH, Patna from Jan 2018 to Dec 2019. The following points are inferred from the study. As compared to the

control group there was highly significant fall of serum  $\text{Na}^+$  &  $\text{K}^+$  level still. They did not achieve the level of mean of control group by 7<sup>th</sup> day. In patients, who referred from HE there was highly significant fall of serum  $\text{Na}^+$  and  $\text{K}^+$  ( $P \leq 0.001$ ) which gradually improved in 7<sup>th</sup> day and who did not recover from HE there was highly significant fall of mean serum  $\text{Na}^+$  and  $\text{K}^+$  ( $P < 0.001$ ) who did not improve after. In these patients there was no significant change in  $\text{Cl}^-$  level. A significant Hypo  $\text{Na}^+$ , Hypo  $\text{K}^+$  and hypo  $\text{Cl}^-$  were observed in study group.

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