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Original Research Article

Serum Electrolytes as an Early Predictor for Severity of Hepatic Encephalopathy

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Abstract:

Background and Objectives: Hepatic encephalopathy is a complex neuropsychiatric syndrome characterisedby disturbances in consciousness and behaviour, personality changes, (1) Alteration in the level of Serum Sodium, Potassium and Chloride in hepaticencephalopathy. (2) Correlation of these levels with severity of hepatic encephalopathy due tovarious causes. (3) The role of Serum Sodium, Potassium and Chloride as a predictor for gradingof severity of hepatic encephalopathy.

Study Design: Prospective observation study. **Inclusion Criteria:** All children with clinical and biochemical evidence of liver dysfunction having neuropsychiatric changes were included. **Exclusion Criteria:** Cases with meningioencephalitis and other cases of encephalopathy will be excluded. Upgraded Department of Paediatrics, BMIMS, Pawapuri.

Study Group: All children with hepatic encephalopathy, they were admitted in paediatrics ward of BMIMS Pawapuri, Nalanda.

Introduction

Hepatic encephalopathy is a complex neuropsychiatric syndrome characterisedby disturbances in consciousness and behaviour, personality changes, fluctuating neurological signs, asterixis or “Flapping tremor” and distractive EEG changes in a patient with liver dysfunction. Liver is the principal organ that performs a wide variety of functions. It maintains Carbohydrate, Protein and fat metabolism and has dominant role insynthesis of cougulation factors, Protein and Enzymes. [1,3] Liver also attributed with a significant role in the maintenance of electrolyte metabolism. [4,5] It is evident that there is hyponatraemia. It is evident that there is hyponatraemia in patient of hepatic encephalopathy, 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 apoor prognostic index but there was no correlation between serum electrolytes changes in patients of hepatic encephalopathy during different stages of hepatic encephalopathy.[6]

Materials and Methods

All children with hepatic encephalopathy they were admitted in Paediatric ward of BMIMS Pawapuri, Nalanda. in eastern side of India; it caters the whole of Bihar and adjacent areas. So, my study will reflect the current status of hepaticencephalopathy in our state.

Inclusion Criteria

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

Exclusion Criteria

Cases with meningioencephalitis and other causes of encephalopathy will beexcluded.

Materials and Method

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, billirubin, AST, ALT and alkaline phosphate
PT, Serum ceruloplasmin
Viral marker. HBSAg, Anti HCV, IgM HAV, IgM HEV
Laptospira serology, if needed
Blood culture
Routine Urine examination
Ascitic fluid study
Chest x-ray
USG whole abdomen
ABG

Statical Formula Employed:

Standard deviation

Standard error of mean

Observation and Results

Observation on Serum Sodium, Potassium and chloride out in 50 healthy subject (control group) and in 120 patient suffering from hepatic encephalopathy due to various etilogy.

The level of Serum Sodium, Potassium and Chloride were estimated in allindividuals of control group. In the study group, the level of Serum Na^+, K^+ & Cl^- were estimated on the day of admission, 3rd day and 7th 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^+ level was 134.13 ±7.14 mmol/lit. S.Na^+ level highly signifies full p value < 0.001 on day 1, 3 and7. In study group Potassium level on day 1 was 3±0.79 mmol/lit, on day 3was 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 onday 1, 3 & 7(P value >0.05)

Showing statistical evaluation of changes in the level of serumsodium in study group with that of control group

Table 1:

Serum Na^+ 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		1st day	126.10	±6.95	±0.634	13.66	<0.001	Highly Significant
				3rd day	128.70	±6.95	±0.685	11.09	<0.001	Highly Significant
				7th 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.

Showing statistical evaluation of changes in the level of Serum Potasium in study group with that of control group.

Table 2:

Serum k+ 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		1st day	3.00	±0.794	±0.115	8.70	<0.001	Highly Significant
				3rd day	3.27	±0.816	±0.104	6.10	<0.001	Highly Significant
				7th 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.

Showing statistical evaluation of changes in the level of SerumChloride in study group with that of control group

Table 3:

Serum Cl^- Z,n in m mol/L in control group			Study Group	Days	Serum Cl^- in m mol/L in study group			Z-Value	P-Value	Remark
Mean	S.D.	S.E.M			Mean	SD	S.E.M			
100.20	±2.65	±0.375		1st day	98.74	±5.99	±0.547	1.65	>0.05	Not Significant
				3rd day	100.25	±4.87	±0.480	0.06	>0.05	Not Significant
				7th 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. From the above observation, it is clear that the serum sodium and the Serum Potassium and Serum Chloride level has decreased on patient ofhepatic-encephalopathy.

The 67 patient who survived and recovered from hepatic encephalopathy, the level of serum Na^+ and K^+were decreased at 1st dayshowed insignificant changes but patients who not survived and not recovered from hepatic encephalopathy, the level of sodium and potassium were decreased on day 1st and does not showed improvement on late phase (on day 7) in them the level of Chloride showed insignificant changes.

Comparison of electrolytes level in survivors and non-survivors of study group

Table 4:

Days when Sample taken	S. Sodium level (Mean)		K^+		Cl^-	
	Survivor	non Survivor	Survivor	non Survivor	Survivor	non Survivor
1st day	127.05	124.90	3.09	2.88	98.14	99.49
3rd day	130.38	125.58	3.43	2.98	100.04	100.63
7th 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 100.04 mmol/l.

Discussion

From the observation, it is clear that both the Serum Sodium and Serum Potassium level has decreased in patient of hepatic encephalopathy. [7,8] 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 (1954), Artmem et al, Nancer, Chettrietal, Parbha et al, Pride et aland Vaish Warner at al have all reported of hyponatraemia in case of hepatic failure. Prabha et al had reported hyponatraemia (S.Na+ < 125 mean/LA) in 52% cases. [9-12]

Vaish Warner et al observed mean serum sodium level was 130 mean/LA.[13] 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 130mean/LA must be regarded as serum and if below 125mean/LA ominous. This hypo Na+ is not amenable to treatment and reflects impending cell death rather than body sodium losses. [14,15] They have also contained 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, 125mean/LA on day 3 and 129.52mean/LA on day 7. [16,17] 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. [18] As suggested by Donald et al it may be due to diluternal effect. Hyponatraemia may be because of primary release of cell K+ 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, Jhonson et al, Narpson et al. 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. [19]

The stat of v hyponatraemia in hepatic encephalopathy of various etilogies has been reported time to time. Altman et al studied 30 patients of cirrhosis in hepatic cellular failure and hepatic coma and found hypokalemia K+ in 25 patients. [20-24]

I.V. infusion of glucose which is usually gives as the treatment of hepatic coma also causes fall in Serum K+ 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+ is uncertain. hypokalemia K+ has been observed in patients with normal total body K+ (Tofler et al) and in the absence of –ve potassium balance (Neinman). [25,26]

The hypokalaemia may be due to failure of renal conservation of potassium in hepatic failure (Mandel et al) 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). [27,28] Neuro Psychiatric changes following the use of thyroids diuretics has been associated with hypokalaemia (Read et al) 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, foulk et al and Gosh and Kanan et al found significant, low level of serum chloride in causes of hepatic coma various etiologies. But Sh [29] erlock 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 andpreviously with different types of fluid. 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. [30]

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

Reference

1. Pessoa MG. Terrault MA, Detmer J, Et al. Quantitation of hepatitis G and C viruses in the liver: evidence that hepatitis G virus is not hepatottrophic. Hepatology. 1998; 27:877-80.
2. Kuwada SK, Patel VM, Hollinger FB, et al. Non – A, Non- B fulminant hepatitis is also no – E and no – C. Am J Gastroenterol. 1994; 89:57-61.
3. Gimson AES, white YS, eddleston ALWF, et al. Clinical and prognostic differences in fulminant hepatitis type A, B and non- A, non – B, gut. 1983; 24: 1994-8.
4. Khuroo M.S. Acute liver failure in India. Hepatology. 1997; 26:244-6.
5. Srivastava KL, Mittal A, Kumar A, et al. Predictors of outcome in fulminant hepatic failure in children. Indian J Gastroenterol. 1988; 17:43- 5.
6. Diris L, Polson RJ, Richardson A, et al. Primary sepsis Presenting as fulminant hepatic failure. QJ Med. 1989; 271: 1037-43.
7. Bhaduri B, Lau JYN, Heaton N, et al. Acute hepatic failure in childhood etiology, prognostic indicators and role of orthotopic liver transplant. J paediatr Gastroenterol Nutr. 1992; 15: 342. (Abstract).
8. Tibbs C, Williams R, viral causes and management of acute Jiver failure. J Hepatol. 1995; 22(Suppl); 68-73.
9. Schiodt FV, Atillasoy E, Shakil AO et al. Etiology and outcome for 295 patients with acute liver in the united state. Liver Transpl Surg. 1999; 5:29-34.
10. Zimmerman HJ, Maddrey WC. Acetaminophen (Paracetamol). Hepatotoxicity with regular intake of alcohol: analysis of instance of therapeutic misadventure. Hepatology. 1995; 22: 767-73.
11. Lee WM. Drug induced hepatotoxicity. N Engl J Med. 1995; 333: 1118- 27.
12. Arora NK, Jain S. Acute liver failure. In: Singh M editor. Medical emergencies in children. 3rd ed. New Delhi: Sagar publication; 2000; 386-410.
13. Treem WR. Hepatic failure. In: walker WA, Durie PR, Hamilton JR, Walker Smith JA, Watkins JB, editors. Pediatric gastrointestinal diseases. 2nd ed. Philadelphia: BC Decker Inc; 1996; 353 – 4.
14. Podolsky OK, Isselbacher KJ. Derangemonts of hepatic mertabolism. In: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL.
15. Editors. Harrison's principles of internal medicine. 14th ed. Vol-2. Newyork: Mc Graw-Hill; 1998; 1667-72.
16. Mullen KD, Dasarathy S. Hepatic encephalopathy. In: Eugene RS, Michael FS, Willis CM. Editors.

Schiff's diseases of the liver. Sth ed. Philadelphia: Lippincott William Squires R. Liver failure. In: Rudolpt) CD, Rudolph AM, Hostetter MK, Lister G, Siegel MJ. Rudolph's paediatrics. 21st ed. Newyork: Mc Graw – Hill; 2002; 1511-3.
17. Gammal SH, Jones EA. Hepatic encephalopathy. Med Clin N Am. 1989; 73: 793 – 808.
18. Schenker S, Breen KJ, Hoyumpa AM. Hepatic enecephalopathy: Current status. Gastroenterology 1974; 66:121-151.
19. Hoyumpa AM, Desmond PV, Avant GR et al. Encephalopathy. Gastroenterology. 1979; 76:184-96.
20. Bemuau J, Jean-Pierre B, Mc Intyre N, Rizzetto M, Rode's, editors. Oxford textbook of clinical hepatology 2nd ed, vol.2.0 oxford: oxford university press; 1999;1341-72.
21. Komori H, Hirasa M, Takakuwa H et al, Concept of the clinical stages of acute hepatic failure. Am J Gastroenterol. 1986; 544-9.
22. Khuroo MS. Acute liver failure. Ann Saud hi Med. 1998; 18: 318-26.
23. Ede RJ, Williams R. Hepatic encephalopathy and cerebral edema. Semin Liver Dis. 1986; 6: 107-123.
24. Wilkinson SP, Hurst 0, Portmann B et al. Pathologenesis of renal failure in cirrhosis and fulminant hepatic failure. Postgrad. Med J; 51: 503-5.
25. Bihari OJ, Gimson AES, Williams R. Cardiovascular pulmonary and renal complication of fulminant hepatic failure. Semin Liver. Dis. 1986;6: 119- 28.
26. Rakela J, Lange SM, Ludwig J, et al. Fulminant hepatitis. Mayo clinic experience with 34 cases. Mayo Clin Proc. 1985; 60: 289-92.
27. Alper G, Jarjour IT. Outcome of children with cerebral edema caused by fulminant hepatic failure. Pediatr Neurol. 1998; 18:299-30. (Abstract)
28. Karvountzis GG, Redekar AG, Peters RL. Long term follows up studies of patients surviving fulminant viral hepatits. Gastroenterology. 1974; 67:870-77.
29. Diseases of the liver and Biliary System in children – By Deirdre A. Kellyt– 3rd edition. 2008.
30. Dhawan A, Cheeesemen P, Approach to Acute liver failure in children. Nelson textbook of Pediatrics 18th edition. 2008.