Electrolytes Derangement in Portal Systemic Encephalopathy (A Preliminary Study)

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Anjum Shahid, Huma Qureshi, Sarwar J. Zuberi (PMRC Research Centre, Jinnah Postgraduate Medical Centre, Karachi.)

Abstract

Serum and urinary electrolytes were determined in 34 patients with portal systemic encephalopathy. In coma due to hepatitis, 73% had hyponatraemia, 64% hypokalaemia and 75% low urinary sodium and potassium. In coma due to cirrhosis, 78% had hyponatraemia, 69.5% hypokalaemia, 79% low urinary sodium and potassium levels in serum and urine appear sodium and 53% reduced urinary potassium. Low to bad prognostic findings. (JPMA 35 211, 1985).

Introduction

Abnormalities in electrolyte metabolism are almost invariable in advanced liver disease.[1]
Marked renal retention of sodium occurs in fulmiriant hepatic failure and cirrhosis.[2-6] Urinary sodium excretion is usually less than 15 meq/24hrs in hepatorenal syndrome.[7]
Hypokalaemia has also been reported in acute and chronic hepatic failure.[8,9]
Serum and urinary electrolytes were therefore studied in coma due to hepatitis and cirrhosis.

Material and Methods

Sodium, potassium, chloride and bicarbonate were determined in 34 patients with portal systemic encephalopathy and 100 controls. In 11 cases, coma was due to hepatitis (group 1) and in 23 due to cirrhosis (group II).
Sodium and potassium were determined in both blood and 24 hours urine samples by flame photometry. Serum chloride was determined by mercuric nitrate method' 0 and serum bicarbonate by micro carbondioxide system, a simplified version of classical Van Slyke method (Harleco kit).

Results

Table I

Serum and Urinary Electrolytes in Controls.

Electrolytes	Serum (me q/l)		Urine(meq/24 hrs)	
	Mean±S.E.	Range	Mean± S.E.	Range
Sodium	142±0.67	132-164	157.4±5.15	80-280
Potassium	4 ±0.04	3.4–4.8	34.98±1.4	24-79
Chloride	102±0.46	92-112	–	–
Bicarbonate	25±0.12	23-28	–	–

Table I shows serum and urinary electrolytes in 100 control subjects.
Electrolyte abnormalities in hepatic coma are presented in table II-VI.

Table II Serum Sodium in Hepatic Coma.

	Coma Hepatitis	Mortality Rate %	Coma Cirrhosis	Mortality Rate %
No. of cases	11		23	
Severe: ∠120 meq/L	1(9.1%)	100%	7(30.4%)	100%
Moderate: 120-126 meq/L	3(27.3%)	100%	2(8.7%)	100%
Mild: 127-134 meq/L	4(36.4%)	25%	9(39.1%)	89%
Hyponatraemia	73%		78.3%	
Normal 135-145 meq/L	3		5	
> 145 meq/L		–		–

Table III

Serum Potassium in Hepatic Coma.

	Coma Hepatitis	Mortality Rate %	Coma Cirrhosis	Mortality Rate %
No. of cases	11		23	
Severe:				
∠3.1meq/L	6(54.5%)	66.6%	11(47.8%)	81.8%
Moderate:				
3.1-3.29 meq/L	–	–	–	–
Mild: 3.3–3.49 meq/L	1(9.1%)	–	5(21.7%)	100%
Hypo-kalaemia	63.6%	–	69.5%	–
Normal: 3.5–4.5meq/L	3		7	
>4.5 meq/L	1		–	–

Table IV

Serum Chloride and Bicarbonate in Hepatic Coma.

Groups	Number of cases	Chloride (meq/L) Mean ± S.E.	Chloride (meq/L) Range	Bicarbonate (meq/L) Mean ± S.E.	Bicarbonate (meq/L) Range
Controls	100	102 ± 0.46	92 – 112	24.8 ± 0.12	23 – 28
Coma Hepatitis	11	100 ± 2.3	85 – 110	22.6 ± 1.33	15 – 31
Coma Cirrhosis	23	101 ± 1.6	82 – 115	25.56 ± 1.26	15 – 38

Seventy three percent cases of coma due to hepatitis (Group I) had hyponatraemia. Of 8 comatose patients with low serum sodium, 5 died and only 3 recovered, whereas coma due to cirrhosis (Group II) had a higher mortality rate. Of 18 hyponatraemics, 17 died giving a mortality rate as high as 94.4% (Table II)

Seven patients in group I had hypokalaemia. of these, 4 died whereas in group II, out of 16 hypokalaemics, 14 died and the mortality rate was 87.5% (Table III).

Serum chloride had normal mean values in both groups, however low levels were found in 18% and 4% cases of group' and II respectively. Serum bicarbonate also had normal mean values in group II but low levels were found in 35% cases. In group I the mean values were slightly low and the decreased level was found in 64% cases. (Table IV).

Urinary sodium was low in 6 cases of coma due to hepatitis. Only one patient recovered; the remaining five died giving a mortality rate of 83.3% whereas in group II, 15 cirrhotics had low urinary sodium, of these 14 died giving a mortality rate of 93.3% (Table V).

Table V

Urinary Sodium in Hepatic Coma.

	Coma Hepatitis	Mortality Rate%	Coma Cirrhosis	Morta-lity Rate %
No. of cases	8		19	
Severe:				
∠ 25meq/24 hrs	6(75 0)	83.3	11(58%)	91
Moderate:				
25-51meq/ 24 hrs	–	–	2(10.5%)	100
Mild :				
52 – 79				
Meq/ hrs	–	–	2(10.58%)	100
Low urinary sodium	75%	–	79%	
Normal:				
80-290meq/ 24 hrs	2		1	
>290 meq/				
24 hrs	–		3	

Urinary potassium was low in 6 patients of group I and 10 of group II Coma. The mortality rate in each case was 66.6% and 90% (Table VI) respectively.

Table VI

Urinary Potassium in Hepatic Coma.

	Coma Hepatitis	Mortality Rate (%)	Coma Cirrhosis	Mortality Rate (%)
No. of cases	8		19	
Severe: ∠ 8meq/24 hrs.	3(37.5%)	66.6	5 (26.3%)	80.0
Moderate: 8–15 meq/ 24 hrs	1(12.5%)	100	2(10.5%)	100
Mild: 16–24 meq/24 hrs	2 (25%)	50	3 (15.8%)	100
Low urinary potassium	75%		52.6%	
Normal: 25–100 meq/24 hrs.	2		8	
> 100 meq/24hrs	–		1	

Discussion

The involvement of the kidney in advanced liver disease has been known. Significant renal insufficiency was found in patients with acute and chronic liver disease. The occurance of renal failure in hepatic disorders carries grave prognosis.[1]

Abnormal renal retention of sodium is a characteristic finding in both cirrhosis and fulminant hepatic failure.[2] Hyponatraemia[12] was found in 73% cases of coma due to hepatitis and 78% cases of coma due to cirrhosis. The mortality rate was high in both groups. In cirrhosis with ascites and oedema there is a tendency for water retention along with sodium and thus serum sodium falls because of dilutional effect. It should not be corrected with hypertonic sodium chloride unless there is clear evidence of a profound loss of sodium from the body.[13]

Metabolic changes commonly associated with portal systemic encephalopathy include hypokalaemia and extracellular alkalosis.[14-16] Blood in the gut lowers the serum potassium level by producing an exchange of circulating potassium into the intestinal fluid.[17] Serum potassium was low in 64% of groun I and 69.5% cases of group II. Potassium levels were lower in comatose patients in groups I and II than in patients with hepatitis and cirrhosis without coma[18] and a value as low as 1.7 meq/L was observed in coma due to cirrhosis. In group II, coma mortality rate was high in mild type of hypokalemia but as the sample number is too small, the result is not conclusive. As hypokal-aemia carries a grave prognosis, prompt and effective measures should be taken to correct itas soon as possible. Potassium chloride supplements must be given both orally and intravenously. If urinary output is normal, at least 120 meq/day are needed.[13]

Serum sodium when related with serum potassium showed no correlation in coma of both types. Serum chloride had normal mean values in both groups while serum bicarbonate was normal in coma due to cirrhosis but slightly low in coma due to hepatitis.

Increased renal retention of sodium results from either a reduction in glomerular filtration rate or from an increased rate of tubular reabsorp-tion.[19] Patients with hepatic coma are unable to excrete solute free water resulting in excretion of a hypertonic urine devoid of sodium.[7] Seventy five percent and 79% cases of group I and II coma respectively had low urinary sodium excretion which is a bad prognostic sign. In group I the level showed wide variation ranging from 0.24- 203 meq/24hrs and the mortality

rate was 83%. Group II also showed variation in the level (0.92-559 meq/24 hrs) and in this group the mortality rate was even higher i.e. 93%. In general urinary sodium excretion is a more sensitive indicator.

Potassium is thought to be excreted by an ion exchange for sodium in a distal portion of the nephron.[20] The absence of potassium wastage in the presence of increased amounts of aldosterone is explained as the result of nearly complete proximal sodium reabsorption, leaving a sharply reduced amount of sodium in the distal tubule to facilitate potassium excretion.[19] Low urinary potassium was found in 75% and 53% cases of group I and II coma and the mortality rate was 66% and 90% respectively. Thus electrolytes are of significant importance in patients with decompensated liver disease. Hypokalaemia carries grave prognosis and should be corrected immediately. It is presumed that correction might improve the prognosis to a greater extent.

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