Electrolytes in Liver Disease-A Preliminary Study

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Abstract

Serum and urinary electrolytes were estimated in 33 patients with hepatitis and 35 with cirrhosis. Forty two percent patients with hepatitis and 46% with cirrhosis bad hyponatraemia while 30% with hepatitis and 37% with cirrhosis had low serum potassium levels. Low urinary sodium was found in 27.3% cases of hepatitis and 40% cirrhotics and low urinary potassium in 21.2% and 45.7% cases of hepatitis and cirrhosis respectively. Hypokalaemia and low urinary sodium appear to be bad progaostic findings in cirrhosis (JPMA 33: 289, 1983).

Introduction

Impairment in electrolyte metabolism was associated with the severity of liver disease (Baldus et al., 1964). Prominent amongst the renal complications of cirrhosis was progressive impairment of renal sodium handling leading to the formation of ascites and peripheral edema (Epstein, 1979). Hyponatraemia is common in cirrhosis (Shear et al., 1965) and hyperaldosteronism is probably the most important factor which results from an increased release of renin by the kidney (Wilkinson et aL, 1975).

Patients with severely impaired hepatic function had greater degrees of potassium depletion and failed to replenish body potassium stores when potassium supplements were given (Casey et al., 1965). Electrolyte abnormalities occur at all stages of liver disease. Serum and urinary electrolytes were therefore determined in patients withhepatitis and cirrhosis.

Material and Methods

Sodium, potassium, chloride and bicarbonate were estimated in 33 patients with hepatitis, 35 with cirrhosis and 100 control subjects.

Sodium and potassium were determined by flame photometry in blood and twenty four hour urine samples. Serum chloride was estimated by the method of Schales and Schales (1941). Serum bicarbonate was estimated by micro carbon dioxide system, a simplified version of classical Van Slyke method (Harleco Kit).

Results

Table– I

Serum and Urinary Electrolytes in Healthy Adults.

	Serum (meq/L)		Urine (meq/ 24 hrs)	
Electrolytes	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	35 ± 1.4	24 – 79
Chloride	102 ± 0.46	92 – 112	–	–
Bicarbonate	24.8 ± 0.12	23 – 28	–	–

Table I represents serum and urinary electrolytes in apparently healthy adults.
In liver disease the concentration of various electrolytes is presented in tables II-VI.
Hyponatraemia was found in 42.4% cases of hepatitis. Of 14 patients with low serum sodium, 12 survived and only 2 died. In cirrhosis low serum sodium level was observed in 46% cases. Here 10 patients died, 5 went into coma prior to death and only 6 survived (Table II).

Table– I

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Hypokalaemia was found in 10 patients with hepatitis and all these recovered. Thirteen cirrhotics and low serum potassium, six patients died and the remaining ‘recovered. Of the six deaths, patients died in coma (Table III).

Table – III Serum Potassium in Liver Disease.

	Hepatitis	Mortality Rate (%)	Cirrhosis	Mortality Rate(%)
No of cases	33	–	35	–
Severe: <3.1 meq/L	4 (12.1%)	–	10 (28.6%)	50%
Moderate: 3.1 – 3.29 meq/L	–	–	–	–
Mild: 3.3 – 3.4 meq/L	6 (18.2%)	–	3 (8.6%)	33.3%
Hypokalaemia	30.3%	–	37%	–
Normal: 3.5–4.5 meq/L	21	–	22	–
>4.5 meq/L	2	–	–	–

Serum chloride and bicarbonate bad normal mean values in çatients with hepatitis and cirrhosis. However serum chloride showed low values in 3% cirrhotics and serum bicarbonate was low in 9% and 11% cases of hepatitis and cirrhosis respectively (Table IV).

Table–IV Serum Chloride and Bicarbonate in Liver Disease.

	Number of Cases	Chloride (meq/L)		Bicarbonate (meq/L)	
		Mean ± S.E.	Range	Mean ± S.E.	Range
Controls	100	102 ± 0.46	92 – 112	24.8 ± 0.12	23 – 28
Hepatitis	33	105 ± 0.92	93 – 113	26.5 ± 0.42	21 – 33
Cirrhosis	35	104 ± 1.02	91 – 116	25.0 ± 0.47	16 – 32

All 9 cases of hepatitiswith low urinary sodium survived. In cirrhotics 40% showed low urinary sodium. Of 14 cases with low urinary sodium, 3 went into coma and 6 patients died in a short period of time (Table V).

Table V Urinary Sodium in Liver Disease.

	Hepatitis	Mortality Rate(%)	Cirrhosis	Mortality Rate(%)
No of cases	33		35	
Severe: <25 meq/24 hrs	2 (6.1%)	–	3 (8.6 %)	66.6
Moderate: 25–51 meq/24 hrs	2 (6.1%)	–	5 (14.3%)	40
Mild: 52–79 meq/24 hrs	5 (15.1%)	–	6 (17.14%)	33.3
Low urinary sodium	27.3%	–	40%	–
Normal: 80–290 meq/24 hrs	21		19	–
>290 meq/24 hrs	3		2	–

Low urinary potassium was found in 21% cases of hepatitis and 45.7% cirrhotics.
Five cirrhotics with low urinary potassium died, of these 3 went into coma prior to death where. as all patients with hepatitis recovered (Table-VI).

Urinary Potassium in Liver Disease.

	Hepatitis	Mortality Rate (%)	Cirrhosis	Mortality Rate (%)
No of cases	33	–	35	
Severe: <8 meq/24 hrs	–	–	1 (2.9 %)	–
Moderate: 8–15 meq/24 hrs	2 (6.1%)	–	4 (11.4%)	50.0
Mild: 16–24 meq/24 hrs	5 (15.1%)	–	11 (31.4%)	27
Low urinary potassium	21.2%	–	45.7%	–
Normal : 25–100 meq/24 hrs	26	–	19	
>100 meq/24 hrs	–	–	–	

Discussion

Abnormalities in fluid and electrolyte metabolism have not been found in patients with hepatitis (Laragh and Ames, 1963). In the present study hyponatraemia was observed in 42% cases. However, the low value was transient and of the hyponatraemics only 14% died and the remaining recovered. Hypokalaemia was evident in 30% patients and all of them recovered. Serum chloride and bicarbonate showed no abnormalities in patients with hepatitis.
Cirrhosis showed a pattern of physiological disturbances in water and electrolyte metabolism together with the changes in renal and adrenal cortical function (Laragh and Ames, 1963). Abnormal renal retention of sodium is a characteristic finding (Wilkinson et aL, 1975). In the present series low serum sodium was found in 45.7% cases. Follow up of these patients revealed an overall mortality rate of

62.5%. Dilutional type of hyponatraemia is frequent in patients with cirrhosis of the liver (Ring Larsen, 1975). Corrective measures using salt therapy should, therefore, be av s it will further decrease the level due uid retention. Restriction of fluid intake t half or even less than the output will serve the purpose and the false hyponatraemia will be corrected.

The severity of the hepatic disease appeared important in the diminuition of body stores of potassium (Casey et al., 1965). Various factors including diet, gastrointestinal losses and diuretic treatment influence the potassium status of cirrhotics. Studies using exchangeable potassium (42k) have shown that cirrhotics even without edema or ascites may be depleted of total body potassium in the presence of normal serum potassium (Aikawa et al., 1953; Burrows et al., 1953). As reported earlier serum potassium determinations are poor indicators of body potassium stores in patients with cirrhosis (Heinemann and Emirgil, 1960; Casey et al., 1965). Hypokalaemia in the present series was found in 37% cirrhotics. In this case the mortality rate was 46%. Regarding hypokalaemia, effective measures should be taken into consideration and potassium should be given orally or intravenously. Oral administration is preferred. It is presumed that timely correction of potassium could have altered the fate of those patients who went into coma and died.

In the present series, serum chloride and bicarbonate had normal mean values in cirrhosis.

Liver is involved in the regulation of renal sodium excretion. Failure of excretion in liver disease could be related to the metabolic and functional impairment of this organ (Kramer, 1975). Urinary sodium excretion was low in patients with cirrhosis (Baldus et al., 1964). Hepatitis in the present study showed abnormalities in urinary sodium. Wide variation in the level (14-360 meq/24 hrs) was observed. However the abnormality was transient and all the patients recovered. Low urinary sodium was found in 40% cirrhotics and the mortality rate was high (66.6%) in cases of extremely low values. The overall mortality was 43%. Level also showed wide variation (2.0-492 meq/24 has) and a value as low as 2.0 meq/ 24 hrs was found.

Low urinary potassium was found in 21% cases of hepatitis and 46% cirrhotics. All cases of hepatitis with low urinary potassium recovered, whereas in cirrhosis the mortality rate was 31.25%.

Hence electrolytes should be repeatedly done in patients with decompensated liver disease and hypokalaemia should be corrected immediately which may improve the prognosis.

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