

Changes in Electrolyte and Blood Urea in Cirrhotic Patients

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Patients suffering from liver cirrhosis, age between 18 to 60 years both male and female were studied from Biochemistry Deptt, PGMI and Medical I, Services Hospital, Lahore. Out of total 49 patients were found to be positive for hepatitis B virus and 23 percent for hepatitis C virus. Serum bilirubin and alkaline phosphatase was highly significant ($p < 0.001$), while alanine transferase moderately and total protein was non-significantly reduced. Blood urea was normal for majority of patients. Serum electrolytes were normal for potassium but decrease in sodium was non significant. Increase in serum bilirubin and alkaline phosphatase, slight decrease in serum sodium and normal potassium were frequent findings in liver cirrhosis.

Key words: Cirrhotic patients, electrolyte and blood urea

Liver cirrhosis manifests itself in younger adults and is an important cause of premature death¹. It is caused by alcoholic liver disease schistosomiasis and viral hepatitis, inherited and metabolic disorders, autoimmune disease, chronic biliary obstruction, hepatic congestion, nutritional causes, drugs, toxins, inflammatory bowel diseases, cystic fibrosis, diabetes mellitus and sarcoidosis⁸. Hepatitis B virus is considered to be one of the major culprits causing liver cirrhosis in this part of the world⁹⁻¹¹ as well as in other regions especially Africa¹² Hepatitis C viruses are prevalent in Europe, Far East³ and Southern India¹⁴ Hepatitis C is known for causing majority of cases of post transfusion hepatitis infection, hepatocellular carcinoma, chronic hepatitis, liver cirrhosis and other liver diseases. Upto 7 percent of apparently healthy blood donors are usually infected in whole antibodies are produced at the end of incubation period and proliferate through convalescence¹⁸⁻²⁰. Recently it was shown that hepatitis C virus infection play a more important role in the development of hepatocellular carcinoma than chronic hepatitis B virus infection⁹. In liver disease low values of urea^{8,21}, hyponatremia²² and decrease in serum potassium⁸ has been observed.

Materials and methods:

Patients suffering from liver cirrhosis, male and female same age and socioeconomic status were selected from the medical unit-I. Their past history including intake of alcohol, general physical examination, ultrasonography, hospitalization, blood transfusions, injections, jaundice, use of razors, any dental procedures or surgical operations and close contact with persons suffering from jaundice was recorded. In general physical examination patients were examined for jaundice, anemia, hepatosplenomegaly, ascites, palmer erythema, spider telangiectasia and bruises.

Collection of Samples: Venous blood (10ml) was drawn from the patients and controls. About 1.8ml was immediately transferred to sodium citrate tube for determination of fibrinogen, prothrombin time (PT), activated partial plasma thromboplastine (APTT). Remaining blood was centrifuged at 3000 rpm and serum

obtained was analyzed for the determination of liver function tests (LFT) and electrolytes.

Methods: HbsAg- Acon one step device IHB-302 USA for Hepatitis B Surface Antigen, HCVQuick Pac II, one step, Syntron Bioresearch, USA, Bilirubin-Mercostst Germany, ALT (Alanine aminotransferase) and ALP (Alkaline Phosphatase, Wiener Lab Argentina, Total Protein-Biurate and Urea-Diacetyl Monoxime, Boehringer-Mannheim Germany and serum Electrolytes-Flame Photometry, Jenway Clinical UK were used.

Results & Discussion:

Patients (82) suffering from liver cirrhosis age between 18 to 60 years both male and females were investigated. The prevalence of liver cirrhosis was somewhat 2 percent higher in females than males (Table 1). Serological tests performed were positive for hepatitis B in 40 cases and hepatitis C in 19 cases indicating that 59 patients had a viral cause of cirrhosis (Table 2). The remaining 23 patients were found negative for both HbsAg and HCV.

Table 1: Age of controls and patients suffering from liver cirrhosis

Groups	Mean+SD	Male	Female
Control (n=40)	46.0+2.77	20 (50)	20 (50)
Patient (n=82)	48.0+1.27	40 (49)	42 (51)

Percentage given in parenthesis.

Table 2: Prevalence of hepatitis B and C found in liver cirrhosis

	Serological markers	
	HbsAg	Angti-HCV
Total Positive	40 (49)	23 (19)
Male	21 (53)	8 (42)
Female	19 (47)	11 (58)
Total Negative	23 (28)	
Male	11 (48)	
Female	12 (52)	

Percentage given in parenthesis.

Liver function tests performed (Table-3) showed that serum bilirubin was raised in the patients (mean±SD, 4.32±1.38 mg/dl) and was highly significant ($P < 0.001$).

Similarly ALT was found elevated in the patients with a mean value of 49.73 ± 22.9 U/ml and was markedly significant ($P < 0.001$). Serum ALP level was also raised in the patients (98.37 ± 57.89 U/L) showed a highly significant rise ($P < 0.001$). Value of the total protein was slightly lower (5.08 ± 1.14 g/dl) in liver cirrhosis and was non-significant. A mean blood urea of 25.1 ± 13.7 mg/dl recorded in liver cirrhosis revealed a non-significant change and remained normal for majority of patients (Table 4). Serum sodium level of patients was slightly below normal (mean \pm SD, 133.28 ± 5.60 meq/L) showed a non-significant decrease. Unlike serum sodium, the level of serum potassium was normal in majority of patients (mean \pm SD, 3.8 ± 1.91 meq/L) and showed a non-significant difference.

Table 3: Function tests of patients suffering from liver cirrhosis

Parameters	Control (n=40)	Patient (n=82)
Bilirubin (mg/dl)	0.67 ± 0.22	4.32 ± 1.38
ALT (U/ml)	36.22 ± 4.78	49.73 ± 22.9
ALP (U/L)	47.87 ± 7.21	98.37 ± 57.8
Total protein (g/dl)	6.72 ± 0.22	5.08 ± 1.14

Liver cirrhosis is a pathologically irreversible chronic injury of hepatic parenchyma and includes extensive fibrosis of regenerative nodules⁸. Socioeconomic status showed that majority of the patients was poor to middle class. Among these 33 percent were labourer, 27 percent jobless, and only 3 percent belonged to good business class. A careful history of these patients revealed that 68 percent had multiple inoculation and 26 percent received blood transfusions. Among these 37 percent gave history of jaundice, 30 percent of razors at barber shops, 25 percent of some surgery and 12 percent dental procedure as well. There was no gender difference in the development of disease as 49 percent were male and 52 percent female (Table-1). The infection with hepatitis B virus (49 percent) was greater than hepatitis C virus (23 percent) which was the commonest cause and was in good agreement with others^{9,23-24}.

Elevated levels of serum bilirubin, aminotransferases and alkaline phosphatases (ALP) and slightly low level of total protein was observed (Table 3). This may be attributed to liver damage when release of enzymes, aspartate and alanine amino transferase occurs. Moreover, retention of bilirubin and alkaline phosphatase (ALP) at sinusoidal surface increase bilirubin and alkaline phosphatase levels²⁵.

In liver cirrhosis, the failing liver is unable to convert ammonia to urea and urea production is impaired. The reserve power of synthesis are so great that the blood urea concentration in hepatocellular failure is usually normal. Low urea in liver cirrhosis occurring due to impaired hepatic deamination of amino acids, results into amino aciduria and sometime hyperammonaemia. If the reduced formation of urea from amino acids is not balanced by renal retention due to increase in glomerular filtration rate

(GFR), plasma urea concentration becomes low.²⁶ In majority of patients normal blood urea level was recorded (Table 4), similar findings have also been reported earlier²¹⁻²⁷.

Table 4: Results showing blood urea and electrolyte level of cirrhotic patients.

Parameters	Control (n=40)	Patient (n=82)
Bloodurea (mg/dl)	26.3 ± 1.27	25.12 ± 13.7
Serum sodium (meq/L)	38.83 ± 4.24	$33.28 \pm 5.6^{**}$
Serum potassium (meq/L)	$4.3 \pm 0.0s$	3.81 ± 1.91

Non-significant, **Significant.

Hyponatraemia in liver cirrhosis, nephrotic syndrome and cardiac failure has been mentioned=22 Retention of sodium in liver cirrhosis is associated with impaired water excretion resulting into total body sodium. Cirrhotic patients without ascites have a normal urinary sodium excretion, those developing ascites retain sodium avidly, therefore urinary sodium excretion decreases and sodium levels are somewhat lower than normal. This does not reflect sodium deficiency, because due to greatly empowered extra cellular sodium space, the actual body stores of sodium are increased²⁷. The main cause of serum decrease in potassium in liver cirrhosis is secondary hypoaldosterolism leading to sodium release and potassium loss in urine⁸.

Slight hyponatraemia and normal potassium level in the present work (Table-4) are also comparable with the observations of other workers^{22,25}. In conclusion high level of serum bilirubin and alanine aminotrasferase and alkaline phosphatase, slightly low blood sodium and normal potassium are found in liver cirrhosis.

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Erratum

Ref. SZ Haider Y Saeed M Akram S Khawaja. "*Comparison of Quincke and Pencil-point bevels in 25-G needles regarding post dural puncture headache in Caesarean Section*". Annals Vol 11 No. 2 APR-JUN 2005. 81-82. Following lapses were noted in the article cited:

1. The numbering of references 5 & 6 have been interchanged i.e. ref. 5 should be read instead of 6 and vice versa.
2. The source of ref. 5 which should now read 6 is, Reg Anaesth Pain Med 2000;25:393-402 instead of Can J Anaesth.2003 May;50(5):460-9.