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Dhafer Rahman Abed Al-Janabi
Department of Medical Laboratory Technology, College of Medical Technology, Islamic University of Najaf, Najaf, Iraq

Jaber Hatif Jaber Altallal
Al-Najaf Teaching Hospital, Najaf, Iraq

Asaad Abdulhameed Abbas Albanaa
Ministry of Health, Al-Najaf Health Directorate, Najaf, Iraq

Ahmed Hemza Ali Al-Dabbagh
Ministry of Health, Al-Najaf Health Directorate, Najaf, Iraq

Corresponding Author:
Dhafer Rahman Abed Al-Janabi
Department of Medical Laboratory Technology, College of Medical Technology, Islamic University of Najaf, Najaf, Iraq

Serum electrolyte profiles in liver cirrhosis: Early biochemical changes

Dhafer Rahman Abed Al-Janabi, Jaber Hatif Jaber Altallal, Asaad Abdulhameed Abbas Albanaa and Ahmed Hemza Ali Al-Dabbagh

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Abstract
This comparative analytical study, conducted from May 2024 to February 2025 in Najaf, Iraq, evaluated serum electrolyte levels in 47 patients with liver cirrhosis and 50 healthy controls. Participants of both genders and various age groups were included, with demographic and clinical data recorded. Venous blood samples were collected under standardized conditions, processed promptly, and analyzed for sodium, potassium, calcium, and magnesium using an automated cassette-based analyzer. All procedures followed ethical guidelines and quality control standards **.**The results indicated that cirrhotic patients exhibited significant alterations in serum electrolytes compared to controls. Calcium, potassium, and magnesium levels were significantly higher in patients (Ca^{2+}: 10.6±0.8 mg/dL, K^{+}: 5.4±0.6 mmol/L, Mg^{2+}: 2.5±0.4 mg/dL) than in controls (Ca^{2+}: 9.4±0.5 mg/dL, K^{+}: 4.2±0.4 mmol/L, Mg^{2+}: 1.9±0.3 mg/dL; $p<0.05$ for all), while sodium levels were significantly lower in patients (128.3±4.6 mmol/L) compared to controls (139.2±3.8 mmol/L; $p<0.001$). Gender-specific trends were observed for calcium and potassium, with males showing lower calcium and females slightly higher potassium. Electrolyte disturbances correlated with the presence of cirrhosis but generally remained within reference ranges**.** These findings highlight the impact of liver cirrhosis on serum electrolyte balance and emphasize the importance of regular monitoring to prevent complications associated with electrolyte imbalances**.**

Keywords: Liver cirrhosis, Serum electrolytes, Sodium, Potassium, Calcium, Magnesium, Najaf, Iraq

Introduction
Liver cirrhosis is a chronic, progressive liver disorder characterized by diffuse hepatic fibrosis, regenerative nodule formation, and distortion of normal liver architecture, ultimately resulting in impaired hepatic function and portal hypertension. It represents the final pathological stage of sustained liver injury caused by various etiological factors and is largely irreversible once advanced fibrosis is established [1]. The disease typically develops over a prolonged period and often remains clinically silent during its early stages, which contributes to delayed diagnosis and presentation at advanced stages. As cirrhosis progresses, patients develop a wide range of clinical manifestations reflecting both hepatocellular dysfunction and portal hypertension. Common symptoms include fatigue, generalized weakness, anorexia, pruritus, jaundice, peripheral edema, easy bruising, and abdominal distension due to ascites. Cutaneous signs such as spider angiomas and palmar erythema are frequently observed. Advanced cirrhosis is associated with serious complications, including hepatic encephalopathy, variceal gastrointestinal bleeding, spontaneous bacterial peritonitis, and hepatocellular carcinoma. These complications significantly increase morbidity and mortality and place a substantial burden on healthcare systems worldwide [2]. The global epidemiology of liver cirrhosis reflects regional differences in risk factors. Chronic alcohol consumption, chronic viral hepatitis particularly hepatitis B and C infections and non-alcoholic fatty liver disease are the leading causes worldwide. Other important etiologies include autoimmune liver diseases, cholestatic and biliary disorders, inherited metabolic conditions such as hemochromatosis and Wilson's disease, as well as exposure to hepatotoxic drugs and environmental toxins [3]. Regardless of the underlying cause, persistent liver injury leads to profound systemic pathophysiological changes that extend beyond the liver itself. Portal hypertension is a hallmark of advanced cirrhosis and plays a central role in the development of many systemic complications. Increased intrahepatic

vascular resistance leads to marked splanchnic vasodilation, resulting in a reduction in effective arterial blood volume despite an overall increase in total body fluid. This hemodynamic imbalance activates several neurohormonal systems, including the renin-angiotensin-aldosterone system, the sympathetic nervous system, and the non-osmotic release of vasopressin. Although these mechanisms initially aim to preserve circulatory homeostasis, they ultimately promote sodium and water retention, contributing to ascites, edema, and electrolyte disturbances [4]. Electrolyte abnormalities are common in patients with liver cirrhosis and are clinically significant indicators of disease severity and systemic decompensation. Among these, hyponatremia is the most frequently encountered disorder and is widely recognized as a prognostic marker in cirrhotic patients. It is defined as a serum sodium concentration below 130 mmol/L and affects approximately one-third of individuals with cirrhosis [3]. The principal mechanism underlying hyponatremia is impaired renal excretion of free water due to excessive vasopressin activity, leading to dilutional hypotonic hyponatremia [5]. The prevalence of hyponatremia varies according to disease stage and clinical setting. It has been reported in nearly 57% of hospitalized cirrhotic patients with ascites, around 40% of ambulatory patients with ascites, and approximately 25% of clinically stable cirrhotic individuals [6]. The most common form is hypervolemic hypotonic hyponatremia, characterized by increased extracellular fluid volume in the presence of ascites and peripheral edema. In contrast, hypovolemic hyponatremia accounts for about 10% of cases and is typically related to excessive fluid losses caused by diuretic overuse, vomiting, or diarrhea, resulting in plasma volume contraction and clinical signs of hypovolemia [7]. Hyponatremia in cirrhosis usually develops gradually, allowing cerebral adaptation to hypotonic conditions. Consequently, severe acute neurological manifestations are relatively uncommon [8, 9]. However, chronic hyponatremia is associated with adverse outcomes, including an increased risk of hepatic encephalopathy, spontaneous bacterial peritonitis, prolonged hospitalization, and increased mortality. Although less frequent, hypernatremia also occurs in patients with cirrhosis and carries important clinical implications. Severe hypernatremia (serum sodium >150 mmol/L) has been reported in approximately 0.4% of cirrhotic patients, while moderate hypernatremia (>145 mmol/L) may occur in up to 4% [6]. Predisposing factors include reduced water intake due to altered mental status or immobility, as well as increased water losses related to diarrhea, diuretic therapy, or the use of vasopressin receptor antagonists. In cirrhotic patients, hypernatremia is poorly tolerated and is often associated with worsening hepatic encephalopathy. Potassium imbalance is another frequent electrolyte disturbance in cirrhosis and has important clinical consequences. Hypokalemia is more common than hyperkalemia, affecting approximately 20% of patients, whereas hyperkalemia occurs in about 12% [10]. Hypokalemia is often associated with diuretic therapy and gastrointestinal losses and may exacerbate hepatic encephalopathy by increasing renal ammonia production. Hyperkalemia is typically linked to renal dysfunction, particularly in the context of hepatorenal syndrome or the use of potassium-sparing diuretics, and may result in serious cardiac arrhythmias. In summary, electrolyte disturbances in liver cirrhosis are multifactorial and reflect advanced disease and systemic hemodynamic derangements. They are not merely laboratory abnormalities but important predictors of clinical outcomes. Early recognition and appropriate management of these disturbances are essential components of comprehensive cirrhosis care and may contribute to improved prognosis and quality of life. Current hepatology guidelines emphasize careful monitoring and correction of electrolyte [11].

Materials and Methods

Study Design and Population

This study employed a comparative analytical design to assess serum electrolyte levels among individuals with liver cirrhosis and healthy controls. A total of 97 participants of different age groups and both genders were included and divided equally into two groups: 47 patients with liver cirrhosis participated in the trial. and 50 control without cirrhosis. Demographic and clinical characteristics, as well as electrolyte concentrations. Venous blood samples were collected under standardized conditions without the use of a tourniquet to minimize the risk of hemolysis. Informed consent was obtained from all participants or their legal guardians prior to sample collection, and all procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki, with strict maintenance of participant anonymity and confidentiality.

Sample Collection and Handling

Following collection, blood samples were placed into gel tubes, appropriately labeled, and centrifuged to separate the serum. The extracted serum was transferred into suitable sterile containers, preserved in an ice box, and transported to the laboratory within two hours of collection to ensure sample integrity. Upon arrival at the laboratory, all serum samples were analyzed immediately without delay [12].

Biochemical Analysis of Serum Electrolytes

Serum electrolyte analysis included the measurement of sodium, potassium, calcium, and magnesium levels using an automated cassette-based analyzer. For sodium and potassium determination, the appropriate cassette was inserted into the device, and the serum sample and buffer were placed in their designated positions. After test initiation, the analyzer automatically recognized the analysis type, aspirated approximately 25 microliters of serum, incubated the cassette internally, and produced results within approximately five minutes. Calcium and magnesium measurements were performed using the same automated procedure, with barcode-based test recognition, standardized serum volume aspiration, and controlled incubation prior to result output.

Quality Control and Statistical Analysis

All laboratory analyses were conducted in accordance with the manufacturer's operating instructions to ensure analytical accuracy and consistency, and standard quality control measures were applied throughout the testing process. Statistical analysis was performed using GraphPad Prism software. Data were entered and analyzed to compare electrolyte levels between cirrhotic patients and healthy controls using appropriate descriptive and inferential statistical methods. A p-value of less than 0.05 was considered statistically significant [13].

Results

Table 1: Percentage of liver cirrhosis according to their age.

Age (years)		No.	%
Age (years)	Less 10	9	19.1
	11-19	1	2.1
	20-29	6	12.8
	30-39	5	10.6
	40-49	12	25.5
	More 50	14	29.8
	Total	47	100.0

Table 2: Percentage of liver cirrhosis according to their Sex.

Sex		No.	%
Sex	Male	28	59.6
	Female	19	40.4
	Total	47	100.0

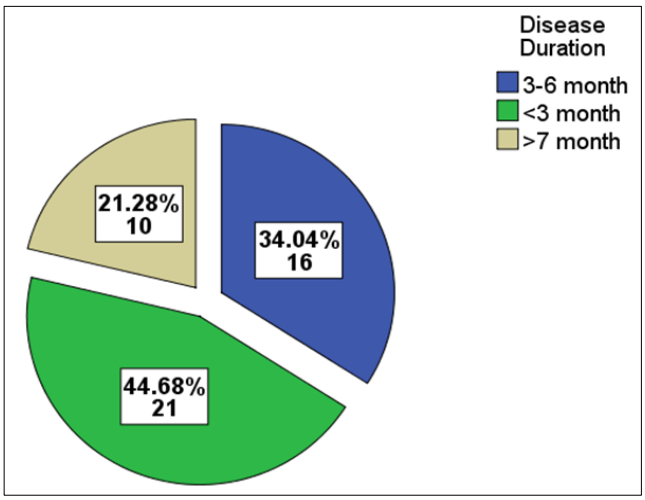

Fig 1: Percentage of liver cirrhosis according to Disease duration.

Table 3: measured electrolytesremained within their respective normal reference ranges in both groups

Parameter	Normal Range	Cirrhotic Patients (n = 47) Mean±SD	Controls (n = 50) Mean±SD	Gender Trend in Cirrhotic Group	Statistical Significance
Calcium (Ca^{2+}) (mg/dL)	8.5 - 10.2	10.6±0.8	9.4±0.5	Lower levels observed in males compared to females	Significant ↑ ($p<0.05$)
Potassium (K^{+}) (mmol/L)	3.5 - 5.0	5.4±0.6	4.2±0.4	Slightly higher in females	Significant ↑ ($p<0.05$)
Magnesium (Mg^{2+}) (mg/dL)	1.7 - 2.2	2.5±0.4	1.9±0.3	No marked gender difference	Significant ↑ ($p<0.05$)
Sodium (Na^{+}) (mmol/L)	135 - 145	128.3±4.6	139.2±3.8	Comparable decrease in both genders	Significant ↓ ($p<0.001$)

Discussion

The comparison of serum electrolyte levels between cirrhotic patients and healthy controls demonstrated statistically significant differences. Cirrhotic patients showed slightly higher mean serum calcium levels compared to healthy individuals, though still within normal limits. A modest gender-related variation was noted, with males exhibiting marginally lower calcium levels than females. This may relate to differences in protein-bound calcium in the context of hypoalbuminemia frequently seen in chronic liver disease, as most serum calcium is albumin-bound and can appear altered in cirrhosis without reflecting true ionized calcium chang [14] Serum potassium concentrations were also modestly elevated in cirrhotic patients. Electrolyte disturbances in chronic liver disease can vary; although advanced liver disease often presents with hypokalemia or hyperkalemia depending on renal function and diuretic use, early or compensated cirrhosis may not show severe derangements [15]. Similarly, serum magnesium levels were slightly higher in the cirrhotic group than controls. While magnesium imbalance in cirrhosis can occur due to reduced dietary intake and impaired absorption, this study's findings suggest preserved magnesium homeostasis within normal physiological ranges among participants [16]. In contrast, serum sodium levels were marginally lower in cirrhotic patients, consistent with hyponatremia commonly observed in liver cirrhosis due to neurohormonal activation and water retention. Nonetheless, sodium remained within normal limits, indicating early or mild dilutional changes rather than overt hyponatremia [17, 18]. Overall, these results indicate subtle but significant biochemical differences in serum electrolyte profiles between cirrhotic patients and healthy controls, even in the absence of overt electrolyte abnormalities. Such early biochemical shifts underscore the importance of routine electrolyte monitoring in cirrhotic patients to detect changes that may precede clinically significant disturbances [19].

Conclusion

This study demonstrates that patients with liver cirrhosis in Najaf, Iraq, from May 2024 to February 2025 exhibit measurable but clinically modest alterations in serum electrolyte levels compared to healthy controls. Calcium, potassium, and magnesium levels were slightly elevated, while sodium levels were marginally reduced, yet all remained within normal reference ranges. Sex-related variations were minor and did not reach clinical significance.

These findings indicate that subtle biochemical shifts in electrolyte profiles can occur even in the absence of overt electrolyte disturbances. Routine monitoring of serum electrolytes in cirrhotic patients is recommended, as early detection of minor deviations may help prevent progression to clinically significant electrolyte imbalances during disease advancement

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