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Electrolytes disturbance in liver cirrhosis

A STUDY OF ADHD & IT'S RELATIONSHIP TO IQ

A THESIS
Submitted to the department of medicine in
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Dedication

To my beloved parents, who were there for me
With their support and encouragement,
I dedicate this work to all their loving tears and beautiful smiles.

To all my respectable teachers,
Who enlightened me with their knowledge and understanding

To all my fellow students, friends, and colleagues
For their unconditional Support and love.

To all patients out there, hoping this little work will do something to help them more in their sufferings.

Keyword

AST = aspartate aminotransferase

ALT = alanine aminotransferase

DM = diabetes mellitus

S.Na = serum sodium

S.K = serum potassium

S.Cl = serum chloride

HBV = hepatitis B virus

HCV = hepatitis C virus

Abstract

Introduction: Cirrhosis is a condition in which the liver does not function properly due to long-term damage, this damage is characterized by the replacement of normal liver tissue by scar tissue. Pathophysiological changes secondary to chronic liver disease and cirrhosis predispose these patients to develop different sorts of electrolytes alterations. Reduced serum sodium concentration is a common finding in patients with cirrhosis.

Objectives: the aim of this study was done to assess the association of the severity of hepatic tissue damage and the serum electrolytes profiles and also focused to observe the correlation among the liver function tests and the serum electrolytes level in sample of Iraqi cirrhotic patients**.**

Patients and methods: This is a descriptive cross-sectional study conducted on cirrhotic patients attending Al- kadhimiya teaching hospital. A total of 20 individuals had been included in the study sample. The patients were included in the study on the basis of diagnosis of cirrhosis.

Results: This study had included 20 individuals with liver cirrhosis. The most prevalent serum electrolytes abnormality was hyponatremia (<136mmol/l) that found in 85% of cases, with a correlation between hyponatremia and low serum albumin in 70% of cases, and between hyponatremia and high total serum bilirubin in 80% of cases. there was a normal value of serum potassium in 65% of patients, and hypokalemia (<3.5mmol/l) in 20% of cases, with no correlation between serum potassium and liver function test. We found normal chloride value in 70% of cases.

Conclusion: Cirrhotic patients are predisposed to different electrolytes disorders**.** Hyponatremia is a common finding in our study (85%). with normal value serum potassium found in (65%) and normal value of serum chloride found in (70%).

Introduction

Cirrhosis is a condition in which the liver does not function properly due to long-term damage.[1] This damage is characterized by the replacement of normal liver tissue by scar tissue.[1] Typically, the disease develops slowly over months or years.[1] Early on, there are often no symptoms.[1] As the disease worsens, a person may become tired, weak, itchy, have swelling in the lower legs, develop yellow skin, bruise easily, have fluid buildup in the abdomen(ascites), or develop spider-like blood vessels on the skin.[1] Other complications include hepatic encephalopathy, bleeding from dilated veins in the esophagus or dilated stomach veins, and liver cancer.[1]

Cirrhosis is most commonly caused by alcohol, hepatitis B, hepatitis C, and non-alcoholic fatty liver disease. [1]

Pathophysiological changes secondary to chronic liver disease and cirrhosis predispose these patients to develop different sorts of electrolytes alterations [1]

The mechanisms underlying these disorders a have significant impact on the prognosis, morbidity and mortality of this group. Cirrhotic portal hypertension leads to systemic vasodilation that reduces renal blood flow and glomerular filtration rate (GFR), reverses normal diurnal rhythm of sodium excretion, and induces sodium–water retention through the activation of renin–angiotensin–aldosterone axis and vasopressin hormone, respectively. Vasopressin increases solute-free water retention by acting on the V2 receptors of the kidney collecting tubules. However, an impaired water excretion is also present in this population due to a combination of renal hypoperfusion and low urinary solute excretion [1].

Reduced serum sodium concentration is a common finding in patients with cirrhosis [2]. being the most common electrolyte disorder in this setting. Indeed, about 20% of patients have values lower than 130 mmol/L, which is the current definition of hyponatremia in cirrhosis [3]. The hyponatremia is thought to be due to a higher rate of renal retention of water in relation to sodium due to a reduction in solute-free water clearance. The consequent inability to adjust the amount of water excreted in the urine to the amount of water ingested leads to dilutional hyponatremia [3]. The prevalence of hyponatremia is around 57 and 40% in hospitalized and ambulatory cirrhotic with ascites, and 25% in stable patients with cirrhosis, respectively [4]. hypotonic hyponatremia associated with increased extracellular volume is the most frequent sort of hyponatremia observed in this population [5].

Patients with cirrhosis can develop two types of hyponatremia which differ markedly with respect to volume status: hypovolemic and hypervolemic hyponatremia. Hypovolemic hyponatremia, which represents 10% of all hyponatremias in patients with cirrhosis [7] ,results from a substantial loss of extracellular fluid in excess of sodium, either from kidneys, as a result of high doses of diuretics, or the gastrointestinal tract due to diarrhea or vomiting. It is characterized by low serum sodium concentration associated with contraction of plasma volume, reduction in the total extracellular fluid volume with clinical signs of hypovolemia, such as tachycardia and reduced renal perfusion. While in patients without cirrhosis hypovolemic hyponatremia is characterized by the absence of edema, ascites and edema can coexist with severely reduced volume in advanced cirrhosis, in most cases, however, hyponatremia develops in the absence of major sodium losses in the context of expanded extracellular fluid volume with ascites and edema that results from renal fluid retention in excess of water with respect of sodium. In fact, although renal sodium retention is a cardinal feature of patients with advanced cirrhosis, solute-free water generation and, therefore, water excretion are also impaired to an extent that leads to a disproportionate increase in total body water relative to total sodium content, ultimately leading to dilutional hyponatremia. [6].

In cirrhosis, hyponatremia generally develops slowly and gradually. Therefore, the brain can adjust to hypo-osmolality and hypotonicity of the extracellular fluid, so that the incidence of neurological manifestations directly attributable to hyponatremia is relatively low [8].

Even though severe hypernatremia (>150 mmol/L) shows low prevalence in cirrhotic (0.4%), moderate hypernatremia (>145 mmol/L) has been reported in up to 4% of these patients. Patients at risk of hypernatremia include those with reduced water intake (altered mental status, immobility, etc.) or increased water loss (diarrhea, diuretics, vaptans, etc.). High serum sodium is much more poorly tolerated (encephalopathy) than hyponatremia in cirrhotic, requiring urgent management [9].

Potassium status is important because it influences general well-being through its physiological effects on muscle contractibility and cardiac function, on hydrogen ion exchange in the tubules,

The serum potassium concentration can vary widely in unstable cirrhotic patients, with a higher prevalence of hypokalemia (20%) than hyperkalemia (12%) in this group [10].

Regarding hypokalemia, there are low body potassium stores in cirrhotic patients which predispose them to present low serum potassium level particularly in the context of malnutrition, secondary hyperaldosteronism, enteric potassium losses (diarrhea), magnesium depletion, renal tubular acidosis, or loop diuretics [11]
However, the increased sympathetic nervous activity is one of the most important factors in the pathogenesis of hypokalemia in cirrhotic patients, due to intracellular potassium shift induced by epinephrine. This sympathetic overactivity can be induced not only by the cirrhosis itself but also by acute hemorrhage or withdrawal syndrome in alcoholic cirrhotic patients [12]. Other causes of hypokalemia secondary to intracellular potassium shift in cirrhotic are: alkalemia, megaloblastic anemia treatment [13].
Hypokalemia can induce stupor and metabolic coma, since it is the extracellular potassium concentration which controls the renal ammonia production in cirrhotic. [14].
Regarding hyperkalemia, it has been demonstrated that there is a reduced tolerance to exogenous potassium loading in cirrhotic patients. During the first three to 6 h after an acute potassium load, the bulk of potassium shifts normally into the intracellular space. Liver and muscles are the main buffering system in the distribution of potassium between compartments partially mediated by insulin. However, a higher increase in serum potassium has been documented after an oral potassium load in cirrhotic patients when compared with healthy people despite similar urine excretion and despite an increase in the insulin secretion response in this population. Skeletal muscles mass reduction and decreased hepatic potassium uptake could be involved in the altered capacity of handling potassium loading in cirrhotic patients. Additionally, liver dysfunction contributes to decreased hepatic cellular potassium uptake despite the presence of insulin hypersecretion. Since insulin stimulates potassium uptake by cells through its action on sodium–potassium–ATPase pump, and also on sodium–hydrogen exchange across the cell membrane, the presence of a sort of insulin resistance has been postulated in order to explain this intolerance to potassium load in this population. [15].
Serum chloride showed no abnormalities in cirrhotic patients with hepatitis [27].

Aim

This study was done to assess the association of the severity of hepatic tissue damage and the serum electrolytes profiles and also focused to observe the correlation among the liver function tests and the serum electrolytes level in sample of Iraqi cirrhotic patients.

Patients and methods

Study setting & design

This is a retrospective & prospective study that was undertaken at Al-Kadhomiya teaching hospital, Baghdad, Iraq. During the period from October 2018 to February 2019.

Materials for this study consisted of 20 patients with liver cirrhosis. The patients were of both sex, 9 Male and 11 female, with age between 24 _ 72, who were admitted at the medical, surgical departments in Al-Kadhimiya teaching hospital.

Selection criteria

Patients were included in the study on the basis of diagnosis of cirrhosis confirmed by clinical, biochemical investigation including liver function test, electrolytes profile (total serum bilirubin, AST, ALT, Serum albumin, serum Na, serum k, serum cl), ultrasonographic findings, and endoscopic findings of esophageal varices.

Exclusion criteria

The patients with hepatocellular carcinoma (HCC), exudative ascites, hypovolemic and hypervolemic causes of hyponatremia and using diuretic within one month were excluded from the study.

Base line assessment

Data was collected through a direct interview with the participants. A verbal consent was taken. Thorough information concerning the patient's condition was obtained, via the questionnaire, from the history, physical examination and biochemical investigations.

Data collection

Venous blood specimen was withdrawn and the tests carried out in the patients were serum electrolytes, LFT, serum albumin were estimated in all patients. Caution had been considered to avoid repetition of the interview with the same patient by looking only for newly registered patients and marking their files during the time of the study.

Study tools

A questionnaire form paper had been developed by direct interview with the patient and can be seen in appendix

Statistical analysis

Data were encoded and filled using Microsoft excel spread sheet (window) then analysis was performed using SPSS.

Results

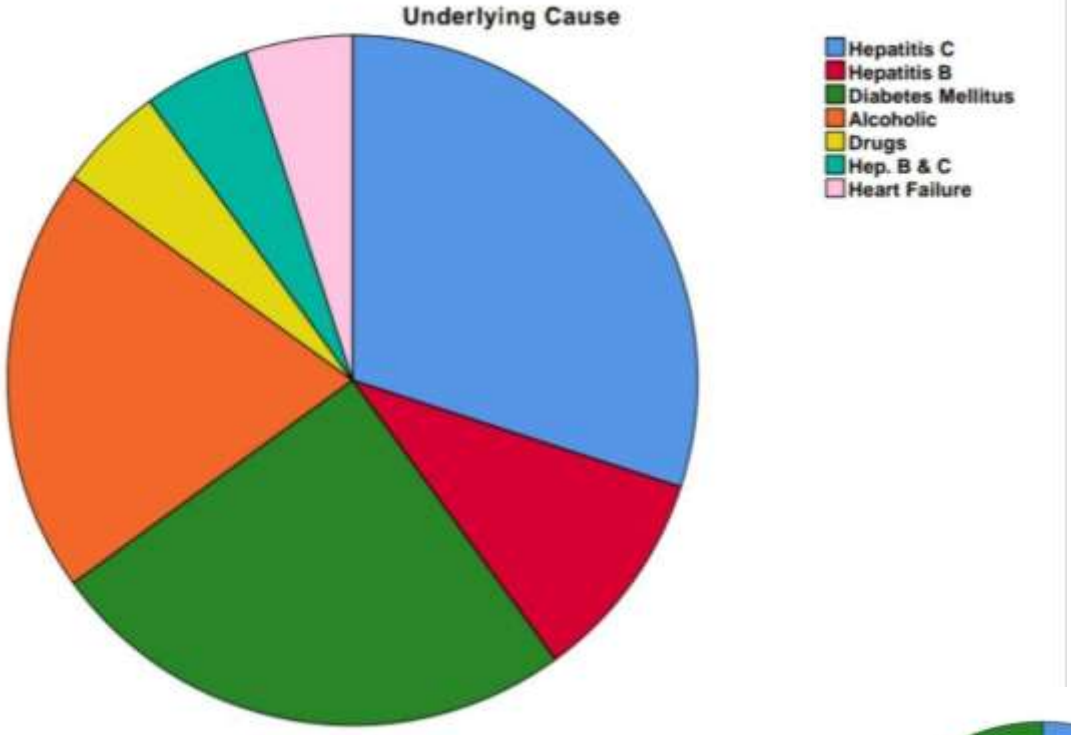
This study had included 20individuals with liver cirrhosis. The mean age of them was (54.45 ± 11.16) years, ranging between 24 and 72 years.

Regarding the sex distribution of the sample, it seemed that the percentage of women was slightly higher than men, 55% vs. 45% respectively. Regarding the duration of cirrhosis, we found 40% of cases with 1-year duration ,30% less than 1 year, 25% of 2 years duration ,5% of 4 years duration.

Regarding the underlying causes of liver cirrhosis, we found that hepatitis C was the cause in 30% of the patient, 10% was hepatitis B, 5% had hepatitis B and C ,25% was non-alcoholic fatty liver disease, 20% were alcoholic liver disease, 5% due to drugs, 5% due to heart failure.

Characters		**Percentage (%)**
sex	males	9 (45%)
	females	11 (55%)
Age group	Range	24_72
	Mean	54.45 ± 11.16
Duration of cirrhosis	<1 year	6 (30%)
	1 year	8 (40%)
	2 years	5 (25%)
	4 years	1 (5%)
Underlying cause	Hepatitis C	6 (30%)
	Hepatitis B	2 (10%)
	Diabetes mellitus	5 (25%)
	Alcoholic	4 (20%)
	Drugs	1 (5%)
	Hepatitis B&C	1 (5%)
	Heart failure	1 (5%)

Table 1: outlines the demographic characteristics of the sample.



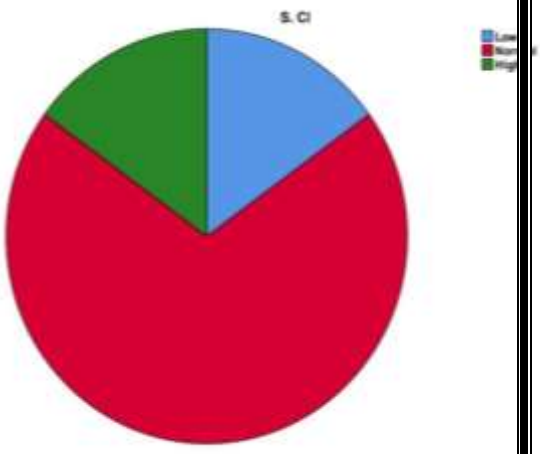
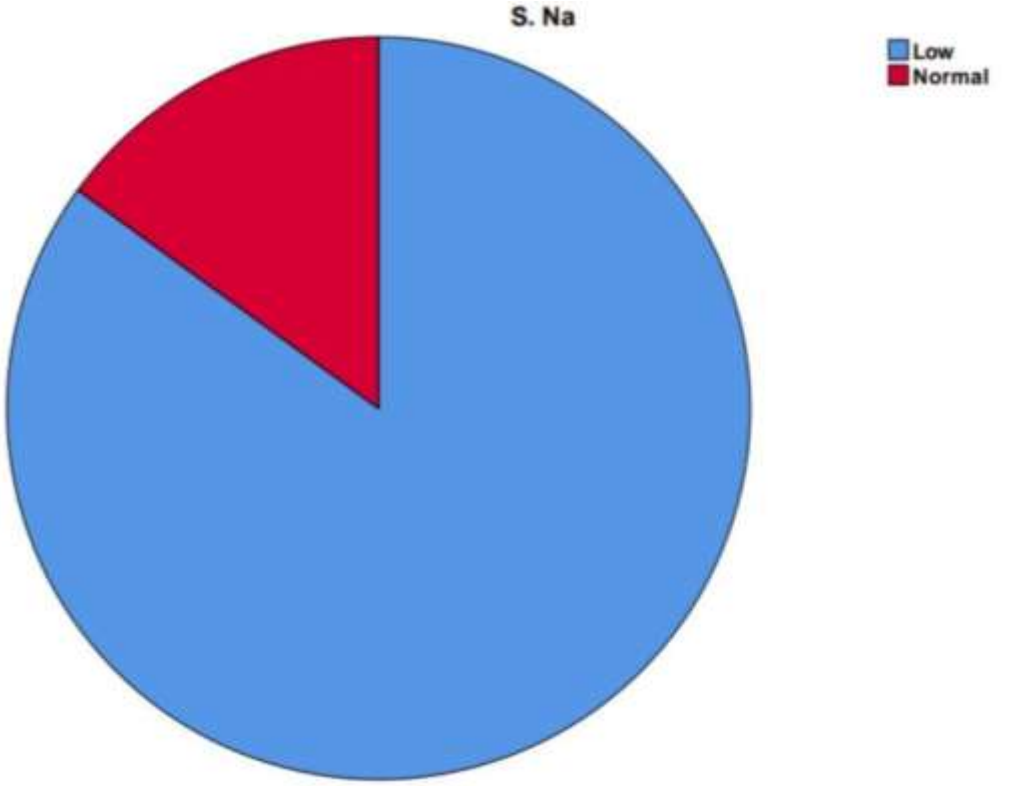


Table 2 has been designed to show the mean and standard of various measures of electrolytes that measured in mmol/l and liver function test, bilirubin and albumin measured in mg/dl, ALT and AST measured in u/l, in patients with liver cirrhosis.

	Mean ± SD
S. Na	132.87 ±3.59
S. K	4.07 ±0.96
S. Cl	103.2±4.9
ALT	43.2 ±39.07
AST	63.2±47.8
S. Bilirubin	3.47 ±2.67
S. Albumin	2.79±0.68

Table 2: mean & standard deviation of electrolytes & liver function tests


S. Na
Low
Normal

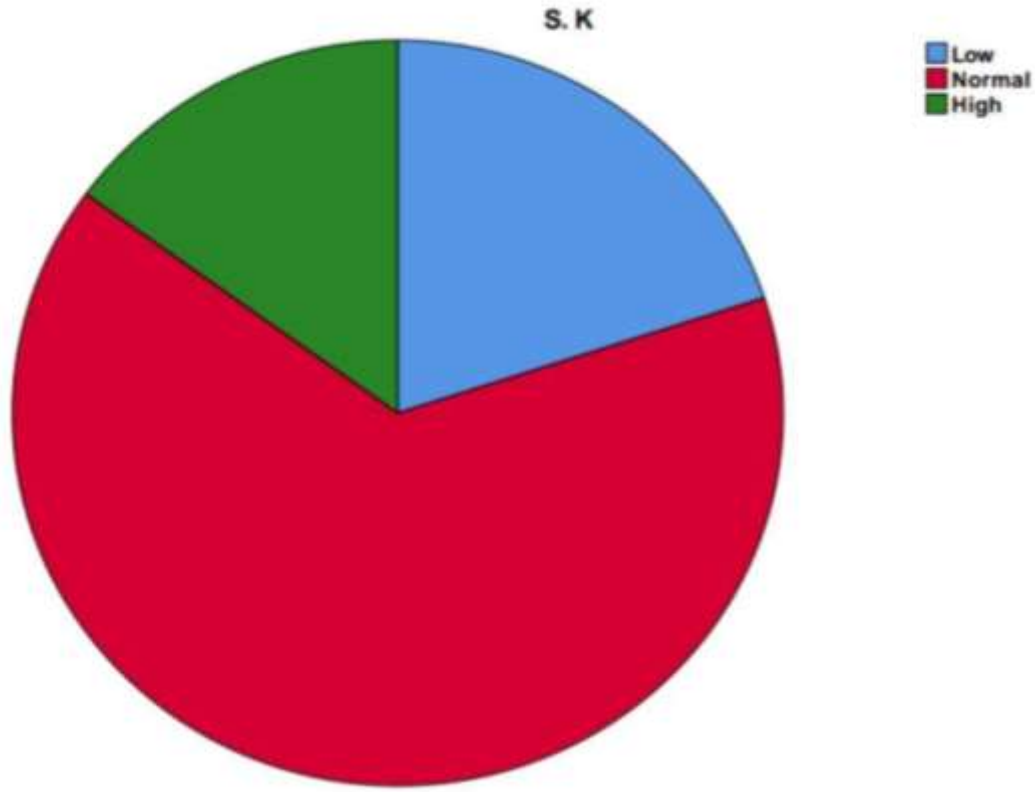

S. K
Low
Normal
High

Table 3 has been designed to show electrolytes profile of patients with liver cirrhosis. The study sought the percentage of Electrolytes disturbance in Cirrhotic patients. The serum sodium below 136 mmol/L was considered as having hyponatremia. In our study, we found, about 85% of the Cirrhotic patient had hyponatremia.
The serum potassium showed a normal value in 65% of cases. the serum potassium below 3.5 mmol/l was considered as having hypokalemia.
in our study we found about 20% of cirrhotic patient had hypokalemia. and above 5.1mmol/l was considered as having hyperkalemia.in our study we found 15% had hyperkalemia.
The serum chloride had a normal value in 70% of cases. However, serum chloride showed low values in 15% of cases and high values in 15% of patients with cirrhosis.

	Range		Percentage (%)
Serum sodium	(136-145mmol/l)	Low	17 (85%)
		Normal	3 (15%)
		High	0 (0%)
Serum potassium	(3.5-5.1 mmol/l)	Low	4 (20%)
		Normal	13 (65%)
		High	3 (15%)
Serum chloride	(98-107mmol/l)	Low	3 (15%)
		Normal	14 (70%)
		High	3 (15%)

Table 3 Electrolytes profile

Table 4 has been designed to show the correlation between electrolytes profile and liver function test. Regarding serum sodium, this study showed correlation between low serum sodium and low serum albumin in 14 (70 %) of cases .and a correlation between low serum sodium and high total serum bilirubin in16(80%)of cases. and correlation between serum sodium and AST in which we found low serum sodium and high AST was in 13 (65%). and no correlation between serum sodium and ALT. regarding serum potassium ,this study showed no correlation between serum

potassium and serum albumin in which normal serum potassium and low serum albumin in 12(60%) of cases .and no correlation between serum potassium and total serum bilirubin in which we found normal serum potassium with high total serum bilirubin in 11(55%),however we found low serum potassium with low serum albumin in 4(20%) of cases and low serum potassium with high total serum bilirubin in4(20%).and no correlation between serum potassium and AST or ALT .Regarding serum chloride ,our study showed no correlation between serum chloride and liver function tests.

Electrolytes		S. albumin		ALT		AST		Bilirubin	
		low	normal	normal	high	normal	high	normal	high
Serum sodium	low	14	3	13	4	4	13	1	16
	normal	3	0	3	0	2	1	1	2
Serum potassium	low	4	0	4	0	2	2	0	4
	normal	12	1	10	3	3	10	2	11
	high	1	2	2	1	1	2	0	3
Serum chloride	low	3	0	2	1	0	3	0	3
	normal	12	2	11	3	4	10	2	12
	high	2	1	3	0	2	1	0	3

Table 4 correlation between electrolytes profile and liver function test:

Table 5 has been designed to show correlation between underlying cause and electrolytes profile. regarding serum sodium in this study showed no correlation between low serum sodium and a specific underlying cause, it was showed low serum sodium in most of the causes. regarding serum potassium, this study showed no correlation between serum potassium and underlying cause, it was showed normal potassium in most of causes. regarding serum chloride, this study showed no correlation between serum chloride and underlying cause, it was showed normal serum chloride in most of causes.

Underlying cause		Hepatitis C	Hepatitis B	Diabetes mellitus	alcoholic	drugs	Hepatitis B & C	Heart failure
S. Na	Low	5	2	5	3	1	1	0
	normal	1	0	0	1	0	0	1
S. K	Low	2	0	1	1	0	0	0
	Normal	3	1	3	3	1	1	1
	High	1	1	1	0	0	0	0
S. CL	Low	0	1	0	1	0	1	0
	Normal	5	1	4	2	1	0	1
	High	1	0	1	1	0	0	0

Table 5: correlation between underlying cause and electrolytes profile

Table 6 has been designed to show past medical history ,80% of patients have diabetes mellitus
50% have hypertension ,20% have heart failure, and 15% have ischemic heart disease.

	frequency	percent
Diabetes mellitus	16	80%
Hypertension	10	50%
Heart failure	4	20%
Ischemic heart disease	3	15%

Table 6 past medical history

Discussion

Twenty subjects were recruited for this study and there was no big difference between the number of males (9) and females (11) subjects. The mean age of the patients was 54.45 ±11.16years.

This study was done to assess the association of the severity of hepatic tissue damage and the serum electrolytes profiles. The study also focused to observe the correlation among the liver function tests and the serum electrolytes level.

The most common causes of cirrhosis in our study is hepatitis, were hepatitis C virus (30%), Hepatitis B (10%), Hepatitis B and C (5 %) and alcoholic is (20%).

this may be because mass injections, traditional practice, occasional exposure, and household contact.

The study done by Eric Levesque1et al shown that alcoholic cirrhosis was 68% while HBV and HCV were the cause of cirrhosis for 4% and 14% of patient. This picture might reflect high consumption of alcohol in western life [16]

In present study nonalcoholic fatty liver disease was found in 25% of cases.

Hsiang et al. reported a retrospective study from a secondary care hospital in South Auckland (New Zealand), The author found that NAFLD cirrhosis was prevalent in 16.4% of cirrhotic patients [17].

Non-alcoholic fatty liver disease has a number of causes, including being overweight, diabetes and high blood pressure [18].

The Percent of patients with diabetes in our study was found in 80%, and patients with hypertension was found in 50%.

In present study, the most prevalent serum electrolytes abnormality was Hyponatremia.

Chronic hyponatremia (defined as a serum sodium concentration below 130 meq/L) occurs in up to 22 % of people with cirrhosis [3]

Dilutional type of hyponatremia is frequent in patients with cirrhosis of the liver [6]

Jong Hoon Kim et al revealed that the prevalence of dilutional hyponatremia, classified as serum sodium concentrations of ≤135 mmol/L, ≤130 mmol/L, and ≤125 mmol/L, were 20.8%, 14.9%, and 12.2%, respectively [19].

In our study we considered chronic hyponatremia those who had serum sodium level of less than 136 meq/L and taking this level as cut-off point we found about 85 %patient with cirrhosis had hyponatremia.

The percentage of hyponatremia sought in this study more conforms to study done by Gines et al. The percent of people with cirrhosis affected by chronic hyponatremia increases to 50 % if a cutoff for serum sodium concentration of 135 mmol/L, the lower limit of normal, is used [3]

The most common reason for chronic hyponatremia in cirrhosis is impairment in renal solute-free water secretion due to increased antidiuretic hormone secretion and decreased effective arterial volume. [19]

In the present study hyponatremia was found with high total bilirubin levels.

Jong Hoon Kim et al sought that the serum sodium level was strongly associated with the severity of liver function impairment [19]

Abnormalities in fluid and electrolyte metabolism have not been found in patients with hepatitis [20].

In the present study hyponatremia was observed in 40% cases with hepatitis

In the present study there is a relation between Hyponatremia and hypoalbuminemia. caused both by decreased synthesis by the hepatocytes and water and sodium retention that dilutes the content of albumin in the extracellular space [21].

The Patients with chronic viral hepatitis (B or C) or nonalcoholic fatty liver disease (NAFLD) have normal or mildly elevated AST and ALT levels [22]

In our study we found AST elevation with normal ALT in 40 % of cases that raise suspicions that the source may be non-hepatic, Non-hepatic causes of AST elevation include injury to skeletal or cardiac muscle [26]. In our study heart failure was in 20% and ischemic heart disease was in 15% of cases. The serum potassium concentration can vary widely in unstable cirrhotic patients, with a higher prevalence of hypokalemia (20%) than hyperkalemia (12%) [10]. The severity of the hepatic disease appeared important in the diminution of body stores of potassium [23]

Studies shown that cirrhotic may be depleted of total body potassium in the presence of normal serum potassium [24]. As reported earlier serum potassium determinations are poor indicators of body potassium stores in patients with cirrhosis [25]. Hypokalemia (serum potassium less than 3.5) in the present series was found in 20% of cases. Various factors including diet, gastrointestinal losses and diuretic treatment influence the potassium status of cirrhotic [24] In the present series, serum chloride had normal mean values in 70 % of cases with cirrhosis. Serum chloride showed no abnormalities in patients with hepatitis. [27] The limitations of our study include: sample size, short time, fallow up, prothrombin time (not available, most patients didn’t do it)

Conclusions

1. Cirrhotic patients are predisposed to different electrolytes disorders since their homeostatic capability is undermined by the combination of their pathophysiological changes and concomitant clinical conditions usually suffered by them.

2. Regarding serum sodium, Hyponatremia is a common finding in our study (85%). we found a correlation between hyponatremia and liver function impairment.

3. Regarding serum potassium, normal value found in (65%), however we found hypokalemia in (20%) of cases. we found no correlation between serum potassium and liver function impairment.

4. Regarding serum chloride, normal value found in (70%) and no correlation between serum chloride and liver function impairment.

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Research Questionnaire:

Name
Gender
Age

- **Diagnosis and duration**

- **Cause of cirrhosis**

- **Past medical history**

- **Drug hx :**

- **Investigations**:

• 1.serum albumin	N (3.2_4.6) mg/dl
• 2.ALT	N (0_55) u/l
• 3.Total bilirubin	N (0.2_1.2) mg/dl
• 4.serum sodium	N (136_145) mmol/l
• 5.serum potassium	N (3.5_5.1) mmol/l
• 6.serum chloride	N (98_107) mmol/l
• 7.AST	N (5_34) u/l