Serum Levels of Zinc and Magnesium in Hepatocellular Carcinoma Patients: A Cross-Sectional Study

Original Article

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ABSTRACT

Background: Hepatocellular carcinoma (HCC) is the sixth most common malignancy worldwide. The liver is the main site of trace element metabolism. Zinc (Zn) is essential for the function of numerous enzymatic molecules. Magnesium (Mg) is fundamental for many physiological and biochemical functions.

The Aim to Work: This work aimed to estimate levels of Mg and Zn in the sera of cirrhotic patients with HCC compared to those in cirrhotic patients without HCC and in healthy subjects. Also, we aimed to evaluate the association of serum levels of Zn and Mg and the Barcelona Clinic Liver Cancer (BCLC) staging of HCC.

Patients and methods: 100 participants were enrolled in our cross-sectional hospital-based study: 60 patients with liver cirrhosis and HCC, 20 cirrhotic patients without HCC, and 20 healthy subjects. Serum levels of Zn and Mg were measured for all subjects. HCC was staged according to BCLC.

Results: This study included 57 males and 43 females; their age was 58.72±14.10. HCC patients had statistically significant lower levels of serum Mg and Zn in comparison to cirrhotic group and healthy controls (p=0.0001 for each). The highest level of serum Mg was seen in patients with BCLC stage C which was significantly higher than the levels seen in stages A and D (p=0.004, p<0.0001 respectively). Serum Zn was not significantly different among BCLC stages.

Conclusions: Serum levels of Mg and Zn are lower in HCC patients in comparison to cirrhotic patients without HCC and healthy subjects. Thus, patients with cirrhotic HCC need Mg and Zn supplementation to compensate for their deficiency.

Key Words: BCLC, HCC, liver cirrhosis, Mg, Zn.

Received: 30 December 2024, **Accepted:** 31 January 2025.

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ISSN: 2735-3540, Vol. 76, No. 1, March 2025.

INTRODUCTION

Hepatocellular carcinoma (HCC) is the sixth most common cancer globally. About 830,000 HCC related deaths and 906,000 new HCC were reported in 2020. Additionally, it ranks third in terms of cancer-related mortality[1,2], with a 15% 5-year survival rate[3].

Trace element metabolism primarily occurs in the liver, and various causes of liver disease have an impact on serum levels of these elements[4]. A vital trace element, zinc (Zn) is necessary for the proper functioning of several enzyme molecules involved in the metabolic processes of human cells. Zn is essential for cell development, differentiation, apoptosis, and metabolism; Zn-binding domains are found in over 300 proteins that control cellular processes[5,6]. Zn also acts as a cofactor for more than 1000 enzymes, which catalyzes superoxide's dismutation to hydrogen peroxide. Zn also increases glutathione biosynthesis, a powerful antioxidant that protects cells from damage by free radicals, toxins, and heavy metals[7].

Increased liver fibrosis[8] and hepatitis C virus (HCV)-related HCC have been linked to zinc deficiency[9]. Additionally, Zn deficiency is linked to hepatic encephalopathy and sarcopenia[10,11].

As a co-factor for up to 600 enzymes, magnesium (Mg) is essential for a wide range of physiological and biochemical processes, such as energy metabolism, cell division, and deoxyribonucleic acid (DNA) repair[12]. According to the current data, Mg plays a different role in

DOI: 10.21608/ASMJ.2025.349140.1358

the field of oncology. A high dietary MG intake has been linked by numerous authors to a decreased risk of breast, colon, and stomach cancers[13,14]. To our knowledge, the role of Mg in liver disease is poorly understood. It has been shown that dietary Mg consumption is negatively correlated with HCC[15]. Moreover, ***Attia et al.***[16] reported hypomagnesemia in patients with HCC. Thus, this work aimed to evaluate the serum levels of Mg and Zn in cirrhotic HCC compared to those in cirrhotic patients without HCC and in healthy subjects. Also, we aimed at investigating the association of serum levels of Zn and Mg and the Barcelona Clinic Liver Cancer (BCLC) stage of HCC.

PATIENT AND METHODS

Study design and patients

This cross-sectional hospital-based study was carried out at the Tropical Medicine and Gastroenterology Department, Sohag University Hospital. From July 2022 to December 2022. A total of 100 participants were enrolled in our study. They were categorized into 3 groups: group 1: including 60 patients with liver cirrhosis and HCC, group 2: including 20 cirrhotic patients without HCC, and group 3: including 20 age and sex matched healthy subjects. Healthy controls were obtained from healthy volunteers who underwent laboratory testing as part of standard medical checkups.

Liver cirrhosis was diagnosed based on the clinical, laboratory data, and ultrasonographic picture of the liver[17]. Liver disease severity was graded according to the modified Child-Pugh score[18]. HCC was diagnosed by triphasic computed tomography (CT). The presence of arterial hyperenhancement with washout on portal venous and/or delayed phases confirms the diagnosis of HCC[19,20]. HCC patients were staged according to the BCLC staging system into stage 0, stage A, stage B, stage C, and stage D[21].

Both male and female adults (≥18 years old) with or without HCC and compensated or decompensated hepatic cirrhosis were included in the current study. Individuals with non-cirrhotic HCC or with other cancers, pregnant and breastfeeding women, and those who took supplements of Zn or Mg during the last three months were excluded.

Methods

All participants underwent a thorough history taking and clinical examination. Ten ml of the peripheral venous blood sample was collected from each participant and then examined for: serum Zn, serum Mg, complete blood count (CBC), liver function tests (Alanine transaminase (ALT), aspartate aminotransferase (AST), total and direct bilirubin, serum albumin, prothrombin time (PT), prothrombin concentration (PC), international normalized ratio (INR)). Serum levels of alpha-fetoprotein (AFP) were measured for cirrhotic patients.

CBC was performed by Sysmex Corporation (XN-1000) automated hematology analyzer. Liver function tests including ALT, AST, bilirubin, and serum albumin were performed by auto-analyzer AU 480 (Bechman Coulter). PT, PC, and INR were done from citrated plasma by Sysmex (CS-1600) coagulation autoanalyzer. AFP assay was performed by ARCHITECTplus (i 1000 SR,Abbott) system using CMIA technology which is a chemiluminescent microparticle immunoassay with reference range for normal adults up to 8.04 ng/ml.

Measurment of serum Zn and Mg

Blood samples for serum Zn and Mg were collected in red tubes without anticoagulant. Serum Mg was measured by colorimetric endpoint method using MG2 kit manufactured by Cobas (Roche) and autoanalyzed by Cobas C311. Normal adult reference range (1.6-2.6 mg/dl). Serum Zn was measured by colorimetric method using kits manufactured by Centronic Gmbh and analyzed by Cobas C311 (Roche). Normal adult reference range (46-150 mcg/dl).

Abdominal ultrasound

Abdominal ultrasound (US) was performed by GE LOGIQ S8 US with convex probe frequency 3.5 megahertz which was used for the assessment of liver size, echo pattern, surface, portal vein (PV) diameter and patency, and the presence or absence of focal lesions (their number and size if any).

Triphasic CT abdomen and pelvis

Triphasic CT of the abdomen and pelvis was done to confirm HCC diagnosis.

ETHICAL CONSIDERATION

All subjects provided written informed consent, and the study was conducted in accordance with the Declaration of Helsinki. Data collecting forms were anonymous,

and data confidentiality was guaranteed. The Scientific Research Ethical Committee of the Faculty of Medicine, Sohag University gave its approval to the study protocol (IRB: Soh-Med-22-07-11). ClinicalTrials.gov Identifier: NCT05488587.

Statistical analysis:

STATA version 17.0 (Stata Statistical Software: Release 17.0 College Station, TX: StataCorp LP) was used to analyze the data. The distribution of different variables was ascertained using the Shapiro-Wilk normality test. The mean, standard deviation, median, and range were used to represent the quantitative data. ANOVA was used to compare the means of three or more groups, and the student t-test was used to compare the means of two groups. When the data were not normally distributed, three or more groups were compared using the Kruskal Wallis test, and two groups were compared using the Mann-Whitney test. Both the Fisher exact test and the Chi-square test were used to compare the qualitative data, which were displayed as numbers and percentages. Serum Mg and Zn levels were correlated with other factors using Pearson's correlation analysis. The STATA or Excel programs were used to create the graphs. *P values* below 0.05 were considered significant.

RESULTS

From July 2022 to December 2022, a total of 100 subjects (57 males, 43 females) were involved in the current study. (Table 1) summarises the demographic and clinical data of cirrhotic patients with and without HCC. HCC group had a statistically significant male predominance (71.67%) compared to liver cirrhosis group (35%) (p=0.003). There was no statistically significant difference between both groups as regard the presence of hepatic encephalopathy, ascites, or gastrointestinal bleeding.

Table 1: Clinical and demographic data of cirrhotic patients with and without HCC.

Variable	Liver cirrhosis N=20	HCC N=60	*P-value*
Age (year)			
Mean ± SD	62.8±9.03	60.81±11.40	0.59
Median (range)	63.5 (40-75)	63 (20-80)	
Gender			
Females	13 (65.00%)	17 (28.33%)	**0.003**
Males	7 (35.00%)	43 (71.67%)	
Hepatic encephalopathy	12 (60.00%)	24 (40.00%)	0.12
Ascites	10 (50.00%)	27 (45.00%)	0.70
GIT bleeding	10 (50.00%)	42 (70.00%)	0.10

Significant *p-values* are in bold. HCC, Hepatocellular carcinoma; GIT, gastrointestinal tract.

(Table 2) summarizes the laboratory parameters of the study groups. Patients of both liver cirrhosis and HCC groups showed statistically significant lower hemoglobin (Hb) levels (p=0.01, 0.001 respectively) and platelet count (p=0.0001, 0.0001) compared to healthy controls. White blood cell (WBC) count showed statistically significant lower values in liver cirrhosis groups compared to healthy controls (p=0.001). However, it was significantly higher in the HCC group compared to the liver cirrhosis group (p=0.01).

Table 2: Laboratory parameters of the studied groups and Child score of liver cirrhosis and HCC groups.

Variable	Healthy controls N=20	Liver cirrhosis N=20	HCC N=60	P1	P2	P3
Hb (g/dl)						
Mean ± SD	12.70±2.34	10.96±2.37	10.83±1.42	**0.01**	**0.001**	1
Median (range)	12.8 (7.2-17.1)	11.5 (4.1-15.8)	10.8 (8.5-14.9)			
WBC ($\times 10^9$/L)						
Mean ± SD	9.01±4.23	5.71±4.04	7.45±3.67	**0.001**	0.12	**0.01**
Median (range)	7.8 (4.25-21)	4.05 (1.4-17.9)	6.55 (2.3-20.1)			
Platelets ($\times 10^9$/L)						
Mean ± SD	300±93.81	133.74±80.33	103.47±52.07	**0.0001**	**0.0001**	0.14
Median (range)	285 (157-516)	131.5 (24-353)	92 (29-227)			
ALT (IU/L)						
Mean ± SD	**13.9±10.14**	**20.5±10.30**	**72.82±79.01**	**0.01**	**0.0001**	**0.0001**
Median (range)	11 (6-53)	18 (7-40)	46 (7-356)			
AST (IU/L)						
Mean ± SD	**18.75±6.60**	**35.55±17.06**	**74.07±80.22**	**0.001**	**0.0001**	**0.01**
Median (range)	18 (10-38)	35 (10-66)	50.5 (15-400)			
Bilirubin (mg/dl)						
Mean ± SD	0.64±0.31	1.27±0.98	2.63±1.71	**0.01**	**0.0001**	**0.0001**
Median (range)	0.65 (0.2-1.3)	0.9 (0.2-4.1)	1.9 (0.7-8.8)			
Albumin (g/dl)						
Mean ± SD	3.78±0.60	2.77±0.68	2.53±0.55	**0.0001**	**0.0001**	0.22
Median (range)	3.9 (1.9-4.6)	2.85 (1.6-4.7)	2.45 (1.7-3.5)			
PT (sec)						
Mean ± SD	11.98±1.72	15.03±5.22	16.27±3.89	**0.004**	**0.0001**	0.07
Median (range)	11.7 (10.2-16.6)	13.65 (10.2-33.3)	15.25 (10.6-24.5)			
INR						
Mean ± SD	1.03±0.15	1.39±0.62	1.42±0.37	**0.001**	**0.0001**	0.18
Median (range)	1 (0.9-1.51)	1.29 (0.9-3.7)	1.39 (0.9-2.23)			
HCV and HBV markers						
-Anti-HCV positive		16 (80.00%)	48 (80.00%)			
-HBsAg positive		1 (5.00%)	6 (10.00%)		0.33	
-Mixed HBsAg and anti-HCV		0	3 (5.00%)			
-Anti-HCV and HBsAg negative		3 (15.00%)	3 (5.00%)			
AFP (ng/ml)						
Mean ± SD		**7.57±4.65**	**451.96±891.08**		**0.0001**	
Median (range)		7 (1.1-17.8)	104.5 (4.5-3500)			
Child classification						
A		5 (25.00%)	12 (20.00%)		**0.02**	
B		11 (55.00%)	15 (25.00)			
C		4 (20.00%)	33 (55.00%)			

Pairwise comparison was done if *p-value* is significant (<0.05). P1 compared healthy control and LC, P2 compared healthy control and HCC, and P3 compared LC & HCC. Significant p-values are in bold. HCC, hepatocellular carcinoma; Hb, hemoglobin; WBC, white blood cells; ALT, alanine aminotransferase; AST, aspartate aminotransferase; PT, prothrombin time; INR, International normalized ratio; AFP, alpha-fetoprotein; HCV, hepatitis C virus; HBV, hepatitis B virus; HBsAg, hepatitis B surface antigen; Sec, second.

Patients of both liver cirrhosis and HCC groups showed statistically significant higher ALT levels (p=0.01, 0.0001 respectively) and AST levels (p=0.001, 0.0001 respectively) compared to healthy controls. Moreover, levels of both ALT and AST were significantly higher in the HCC group compared to the liver cirrhosis group (p=0.0001, 0.01 respectively). Total bilirubin levels showed statistically significant higher levels in liver cirrhosis and HCC groups (p= 0.01, 0.0001 respectively) compared to the healthy group. Moreover, it was significantly higher in the HCC group compared to the liver cirrhosis group (p=0.0001).

Patients of both liver cirrhosis and HCC groups showed statistically significant lower levels of albumin compared to the healthy control group (p=0.0001, 0.0001 respectively).

Patients of liver cirrhosis and HCC groups showed statistically significant prolonged PT (p=0.004,0.0001) and higher INR (p=0.001, 0.0001) compared to the healthy control group. Patients of the HCC group showed significantly higher levels of AFP compared to the liver cirrhosis group (p=0.0001).

Patients of the liver cirrhosis group showed statistically significant lower levels of serum Mg and Zn compared to healthy controls (p=0.0001 for each). Patients of the HCC group showed statistically significant lower levels of serum Mg compared to both liver cirrhosis and healthy control groups (p=0.0001 for each). They also had statistically significant lower levels of serum Zn compared to the other two groups (p=0.0001 for each) (Figures 1,2).

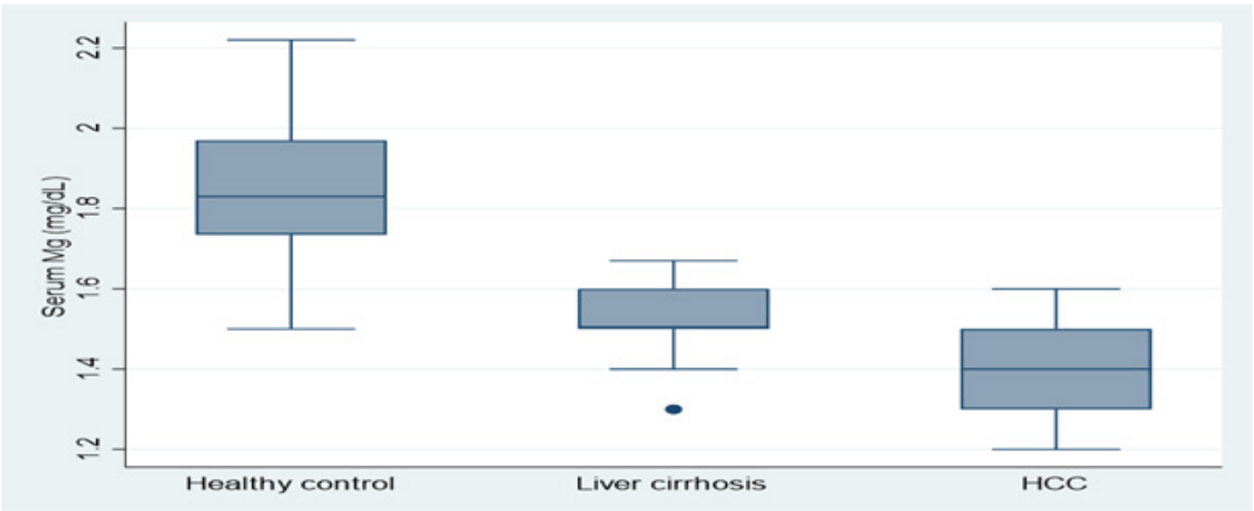

Fig. 1: Serum Mg (mg/dl) of studied groups: Patients of the HCC group showed statistically significant lower levels of serum Mg compared to both liver cirrhosis and healthy control groups (P1=P2=P3= 0.0001).
HCC, hepatocellular carcinoma; Mg, magnesium.

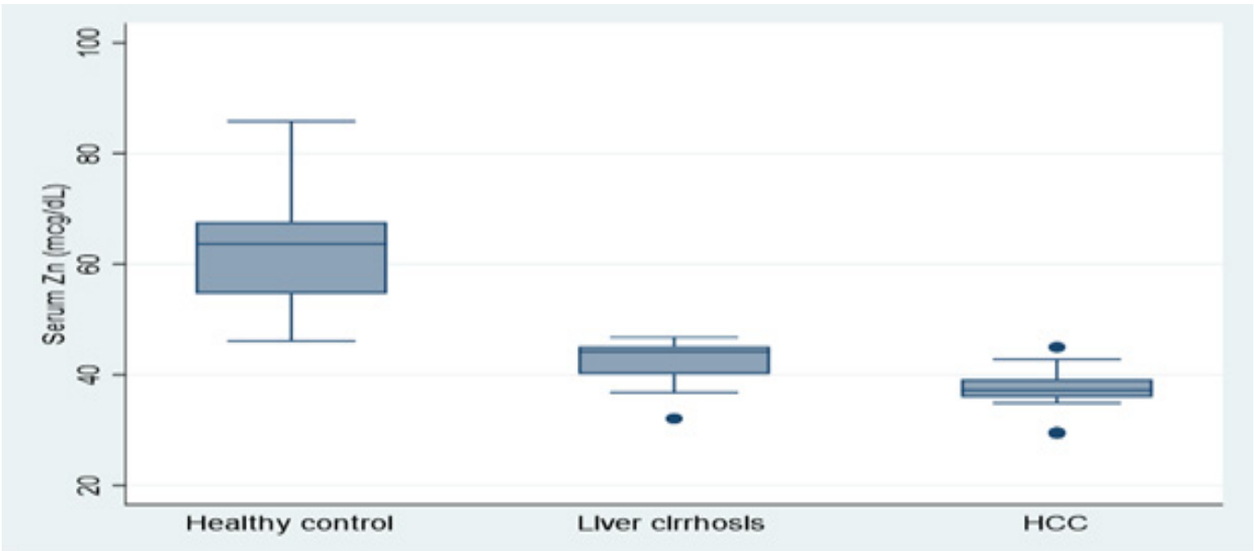

Fig. 2: Serum Zn (mcg/dl) of studied groups: Patients of the HCC group showed statistically significantly lower levels of serum Zn compared to both liver cirrhosis and healthy control groups (P1=P2=P3= 0.0001).
HCC, hepatocellular carcinoma; Zn, zinc

HCC characteristics and BCLC stage of the HCC group are summarized in (Table 3).

Table 3: HCC characteristics and BCLC stage of the HCC group.

Variable	Statistic
No of HFL	
Single	28 (46.67%)
Multiple	32 (53.33%)
Maximum diameter of HFL (cm)	
Mean ± SD	4.69±2.54
Median (range)	4.5 (0.4:9)
Site of HFL	
Left lobe	10 (16.67%)
Right lobe	22 (36.67%)
Both	28 (46.67%)
PV	
Patent	33 (55.00%)
Thrombosed	27 (45.00%)
Performance status	
0	9 (15.00%)
1	6 (10.00%)
2	24 (40.00%)
3	12 (20.00%)
4	9 (15.00%)
5	0
BCLC	
-Stage A (early)	3 (5.00%)
-Stage B (intermediate)	6 (10.00%)
-Stage C (advanced)	12 (20.00%)
-Stage D (terminal)	39 (65.00%)

HCC, hepatocellular carcinoma; HFL, hepatic focal lesion; PV, portal vein; BCLC, Barcelona Clinic Liver Cancer.

Patients of Child C score in the liver cirrhosis group had statistically significant lower levels of serum Mg compared to patients of Child score A and B (p=0.02, 0.0001 respectively) (Figure 3). Moreover, patients with Child C score of the HCC group had statistically significant lower levels of serum Mg compared to patients of Child score A and B (p=0.001, 0.0001 respectively) (Figure 4). Patients with Child score C in the HCC group had statistically significant lower levels of Zn compared to patients with Child score A (p=0.02) (Table 4).

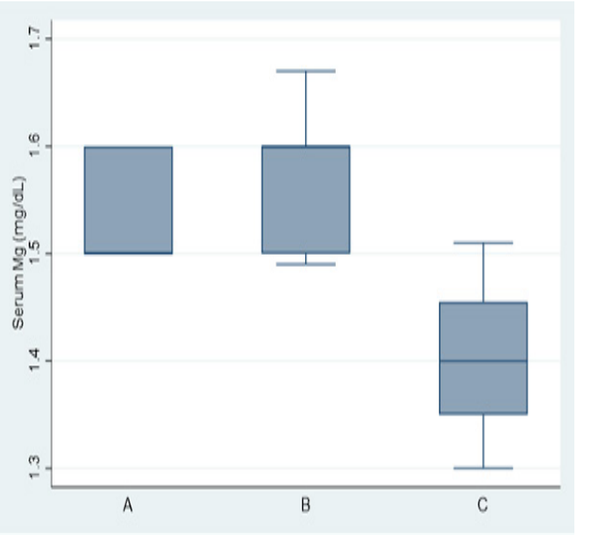

Fig. 3: Relation between serum Mg and Child classification in liver cirrhosis group: Patients of Child C score in the liver cirrhosis group had statistically significant lower levels of serum Mg compared to patients of Child score A and B (*P-value* A&B=1, *P-value* A&C= 0.02, *P-value* B&C= 0.0001).
Mg, magnesium.

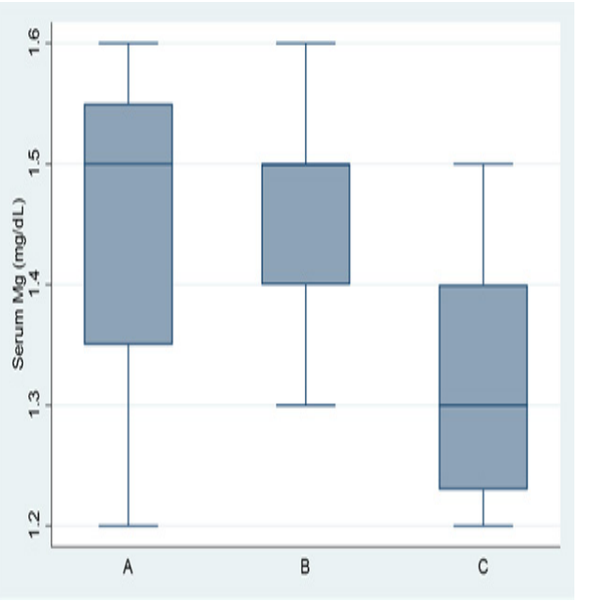

Fig. 4: Relation between Serum Mg and Child classification in the HCC group: Patients of Child C score in the HCC group had statistically significant lower levels of serum Mg compared to patients of Child A and B (*P-value* A&B=1, *P-value* A&C= 0.001, *P-value* B&C= 0.0001).
HCC, hepatocellular carcinoma; Mg, magnesium.

Table 4: Relation between serum Zn and Child score in both liver cirrhosis and HCC groups.

Variable	Serum Zn (mcg/dl) Mean ± SD, median (range)	P B&C	P A&C	P A&B
Child score of liver cirrhosis group				
A	42.16±5.63, 44.5 (32.1-45.2)			
B	43.42±2.80, 44.1 (36.8-46.8)		**0.41**	
C	40.45±3.32, 39.7 (37.3-45.1)			
Child score of HCC group				
A	39.38±3.07, 38.5 (36-45)			
B	37.52±4.24, 39.3 (29.4-41.6)	0.23	**0.02**	1.00
C	36.84±1.27, 37 (34.9-39.5)			

Significant *p-values* are in bold. HCC, hepatocellular carcinoma; Zn, zinc.

When comparing serum levels of Mg among different stages of HCC, the highest level was seen in patients with advanced stage (C) which was significantly higher than the levels seen in stages (A) and (D) (p=0.004, p=<0.0001 respectively). (Table 5). Serum Zn did not show significant difference among different BCLC stages.

Table 5: Relation between serum Mg and both PV thrombosis and BCLC stage in the HCC group.

Variable	Serum Mg Mean ± SD, median (range)	*P-value*	Pairwise comparison (*p value*)			
PV						
Patent	1.35±0.11, 1.3 (1.2-1.6)	**0.01**				
Thrombosed	1.42±0.13, 1.4 (1.2-1.6)					
BCLC						
-Stage A (early)	1.27±0.06, 1.3 (1.2:1.3)			A	B	C
-Stage B (intermediate)	1.45±0.11, 1.45 (1.3-1.6)	**<0.0001**	B	0.09		
-Stage C (advanced)	1.51±0.08, 1.5 (1.4-1.6)		C	**0.004**	1.00	
-Stage D (terminal)	1.35±0.11, 1.3 (1.2-1.6)		D	1.00	0.16	**<0.0001**

Significant *p-values* are in bold. Mg, magnesium; BCLC, Barcelona Clinic Liver Cancer; HCC, hepatocellular carcinoma; PV, portal vein.

DISCUSSION

The current cross-sectional study aimed to evaluate the serum levels of Mg and Zn in cirrhotic HCC compared to those in cirrhotic patients without HCC and in healthy subjects, and to investigating the association of serum levels of Zn and Mg and the stage of HCC. The current study revealed an inverse relation between serum Mg and HCC, as patients of the HCC group had lower levels of serum Mg than liver cirrhosis and healthy controls. This finding agrees with ***Attia et al.***, 2018[16] and ***Parisse et al.***, 2021[22]. Moreover, we found that the liver cirrhosis group had lower levels of serum Mg than healthy controls. Comparable results were documented by ***Kar et al.***[23] and ***Nangliya et al.***[24].

According to the Child-Pugh score, we found a steady and highly significant decline in serum Mg that corresponded with the severity of liver disease. This finding agreed with the results of ***Nangliya et al.***[24], and ***Attia et al.***[16]. However, other studies found no significant difference in serum Mg between the Child-Pugh classes[23,25]. In our study, HCC stage progression was not always associated with lower serum Mg levels. Due to low dietary absorption, increased urine secretion, decreased plasma albumin concentrations, and hormone inactivation, patients with liver cirrhosis exhibit Mg deficiency. Due to impaired protein kinase C translocation, inflammatory reactions, oxidative stress, and metabolic abnormalities, Mg deficiency, on the other hand, exacerbates cirrhosis and can lead to the growth of liver cancer[26].

Mg is an essential component of DNA repair pathways and an enzyme cofactor that helps to preserve genomic integrity. It is essential for controlling apoptosis, differentiation, proliferation, and the cell cycle. Hence, Mg deficit may result in these mechanisms malfunctioning and causing cancer and DNA changes[27,28]. Mg deficiency has been linked to liver cancer[29]. Tumor growth factor- beta (TGF-b) is a tumor promoter that causes the invasiveness and metastasis of HCC by inducing the epithelial-mesenchymal transition. Through pleiotropic mechanisms, Mg helps the body by decreasing TGF-b activity, limiting the transcription of genes that promote tumor growth, and boosting the activity of protein phosphatase Mg-dependent 1a[26]. In cancer patients, Mg deficiency raises the likelihood of cancer metastasis to the liver[30]. The current study found significantly higher levels of serum Mg in HCC patients with PV thrombosis compared to those with patent PV. To the best of our knowledge, the relationship between PV thrombosis in HCC patients and serum Mg was not previously investigated and further work is required to illustrate this relation.

Regarding serum Zn, we found that patients with liver cirrhosis had lower serum levels of Zn as compared to healthy controls. The same results were found by ***Nakayama et al.***[31], ***Elzeiny et al.***[32], and ***Nangliya et al.***[24]. Moreover, patients with HCC had lower levels of serum Zn than healthy controls. Many authors found similar results[16,32,33]. Comparing serum Zn levels in patients with liver cirrhosis and HCC, we found that the HCC group had lower levels of serum Zn, and this agrees with ***Saber et al.***[34]. However, ***Elzeiny et al.***[32] found no significant difference between both groups.

We also found a decrease in serum levels of Zn are with progression of liver disease according to the Child-Pugh classification. These findings agreed with the results of ***Nangliya et al.***[24], ***Agarwal et al.***[25], and ***Attia et al.***[16].

A decrease in Zn levels in cirrhotic patients may be due to low Zn levels with low protein intake due to protein reluctance, increased gastrointestinal system losses secondary to diarrhea or intestinal malabsorption, or increased urine losses[35]. In addition, liver cells absorb more Zn when there is cellular damage and inflammation because they need it to produce proteins, nucleic acids, and enzymes. The intake and absorption of Zn decrease as liver damage progresses because of poor appetite, compromised intestine and stomach function, and high portal vein pressure. Additionally, low serum albumin levels cause less combination of Zn with other nutrients, and due to the blood Zn diffusion properties, it is easily lost through sweating and urine[36,37].

Zn promotes the activity of the enzymes that repair DNA, which protects against cancer development. Moreover, it is a part of the enzyme superoxide dismutase, which eliminates free radicals[38].

Normal Zn concentrations cause malignant cells to undergo apoptosis, slow down their rate of division, and lose their ability to migrate and invade. "Tumor-suppressor" actions are the general term for Zn effects on cancerous cells[39].

Malignant cells have developed ways to decrease cellular Zn accumulation to non-cytotoxic levels that promote their creation, development, proliferation, and malignant activities, hence evading the harmful effects of Zn at normal cell concentrations. To lessen Zn buildup, malignant cells alter the expression and quantity of Zn transporters, utilizing this changed defense mechanism. Higher Zn levels present in normal hepatocytes may have

cytotoxic effects on malignant cells[40].

CONCLUSIONS

Serum Mg levels tend to be low in liver cirrhosis and lower in HCC and their levels become lower with the progression of liver disease as assessed by Child score but not always with the progression of HCC stage as assessed by BCLC. HCC with PV thrombosis is associated with higher levels of serum Mg than HCC with patent PV. Serum levels of Zn tend to be low in liver cirrhosis and lower in HCC and their levels become lower with the progression of the liver disease as assessed by Child score. Thus, patients with HCC and liver cirrhosis need Mg and Zn supplementation to compensate for their deficiency.

ABBREVIATIONS

AFP: alphafetopeotein,

ALT: alanine transaminase,

AST: aspartate transaminase,

BCLC: Barcelona Clinic Liver Cancer,

CBC: complete blood count,

CT: computed tomography,

DNA: deoxyribonucleic acid,

HCC: hepatocellular carcinoma,

HCV: hepatitis C virus,

INR: international randomized ratio,

Mg: magnesium,

PC: prothrombin concentration,

PT: prothrombin time.

TGF-b: tumor growth factor- beta

Zn: zinc,

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مستويات الزنك والمغنيسيوم في مصل الدم لدى مرضى سرطان الكبد: دراسة مقطعية

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الخلفية: يُعد سرطان الكبد سادس أكثر انواع السرطانات انتشارا على مستوى العالم، و يعد الزنك عنصرًا ضروريًا لوظيفة العديد من الإنزيمات، كما أن المغنيسيوم أساسي للعديد من الوظائف الفسيولوجية والكيميائية الحيوية. هدفت هذه الدراسة إلى تقييم مستويات الزنك والمغنيسيوم في مصل الدم لدى مرضى تليف الكبد المصابين بسرطان الكبد ومقارنتها بمستوياتها لدى مرضى تليف الكبد غير المصابين بالسرطان والأشخاص الأصحاء. كما تم التحقيق في العلاقة بين مستويات الزنك والمغنيسيوم في الدم ومرحلة سرطان الكبد وفق تصنيف Barcelona Clinic Liver Cancer (BCLC).

المرضى وطرق البحث: شارك ١٠٠ شخص في هذه الدراسة المقطعية: ٦٠ مريضًا يعانون من تليف الكبد وسرطان الكبد، و٢٠ مريضًا يعانون من تليف الكبد بدون سرطان الكبد، و٢٠ شخصًا من الأصحاء. تم قياس مستويات الزنك والمغنيسيوم في مصل الدم لدى جميع المشاركين، وتم تصنيف مراحل سرطان الكبد وفقًا لتصنيف BCLC.

النتائج: شملت الدراسة ٥٧ ذكرًا و٤٣ أنثى، وكان متوسط أعمارهم ٥٨,٧٢ ± ١٤,١٠ عامًا. أظهرت النتائج أن مرضى سرطان الكبد لديهم مستويات أقل بشكل ملحوظ من المغنيسيوم والزنك في الدم مقارنة بمرضى التليف الكبدي غير المصابين بالسرطان والأشخاص الأصحاء ($p=0.0001$ لكل منهما). لوحظ أن أعلى مستوى من المغنيسيوم في الدم كان لدى المرضى في المرحلة C وفقًا لتصنيف BCLC، وكان هذا المستوى أعلى بشكل ملحوظ مقارنة بالمستويات في المرحلتين A و($p<0.0001$، $p=0.004$) D على التوالي. أما بالنسبة لمستويات الزنك، فلم يكن هناك اختلاف كبير بين المراحل المختلفة لتصنيف BCLC.

الاستنتاجات: مستويات المغنيسيوم والزنك في الدم أقل لدى مرضى سرطان الكبد مقارنة بمرضى التليف الكبدي غير المصابين بالسرطان والأشخاص الأصحاء. لذلك، يحتاج مرضى سرطان الكبد وتليف الكبد إلى مكملات من المغنيسيوم والزنك لتعويض هذا النقص.