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Serum Zinc Levels and Associated Factors in Patients with Liver Cirrhosis: A Cross-Sectional Study at a Tertiary-Level Hospital

Afsana Sharmin[1*], Nafisa Sultana Khanam[2], Md. Sabbir Hossain[3], Siddika Sanjida[4], Sharmin Akhter[5], Mousumi Akter[6], A B M Shakil Gani[7], Tasnim Mahmud[8]

[1]Department of Biochemistry, Shahid Monsur Ali Medical College, Dhaka, Bangladesh

[2]Department of Biochemistry, Dhaka Medical College, Dhaka, Bangladesh

[3]Department of Hepatology, Dhaka Medical College, Dhaka, Bangladesh

[4]Department of Biochemistry and Molecular Biology, International Medical College, Gazipur, Bangladesh

[5]Department of Pharmacology and Therapeutics, Shahid Monsur Ali Medical College, Dhaka, Bangladesh

[6]Department of Biochemistry, Fazlur Rahman Medical College, Dhaka, Bangladesh

[7]Department of Hepatology, Dhaka Medical College, Dhaka, Bangladesh

[8]Department of Public Health, North South University, Dhaka, Bangladesh

Corresponding Author: Afsana Sharmin

Department of Biochemistry, Shahid Monsur Ali Medical College, Dhaka, Bangladesh

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KEYWORDS

Liver Cirrhosis, Serum Zinc, Zinc deficiency, Comorbidities, Bangladesh

ABSTRACT:

Background: Liver cirrhosis is a major global health problem, often accompanied by micronutrient deficiencies, particularly zinc, which is essential for hepatic metabolism, immune function, and ammonia detoxification. Data on serum zinc levels and associated factors in Bangladeshi patients are limited. This study aimed to assess zinc status and its clinical associations in cirrhotic patients at a tertiary hospital.

Methods: A cross-sectional analytical study was conducted from July 2024 to June 2025 at Dhaka Medical College Hospital. Fifty-five patients aged 18–70 years with clinically diagnosed liver cirrhosis were enrolled. Data were collected through a structured questionnaire, and serum zinc levels were measured from blood samples using standard laboratory methods. Statistical analysis was performed using SPSS version 26, with $p < 0.05$ considered significant. Ethical approval and informed consent were obtained.

Results: Of the 55 participants, 67% were male, with a mean age of 46 ± 12 years. Compensated and decompensated cirrhosis were nearly equally represented (49.1% vs. 50.9%). HBV was the predominant etiology (75%), with HCV accounting for 25%. Decompensated patients had higher prevalence of diabetes (71% vs. 37%), hypertension (82% vs. 56%), and chronic kidney disease (89% vs. 33%), whereas ischemic heart disease was similar across groups. Serum zinc levels were significantly lower in decompensated patients (0.41 ± 0.14 mg/L) than compensated patients (0.64 ± 0.20 mg/L). Overall, 65% of patients were zinc deficient, and deficiency was significantly associated with hypertension and chronic kidney disease.

Conclusion: Zinc deficiency is highly prevalent in cirrhosis and correlates with disease severity and comorbidities. Monitoring and correcting zinc levels may provide prognostic information and guide therapeutic strategies, particularly in decompensated cirrhosis.

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1. INTRODUCTION:

Cirrhosis, the final stage of chronic liver disease, is a major cause of morbidity and mortality worldwide [1,2]. It is characterized by progressive replacement of healthy liver tissue with scar tissue, leading to impaired hepatic function [3]. Globally, autopsy studies estimate that 4.5–9.5% of the population may have cirrhosis, while the Global Burden of Disease 2020 report estimated that approximately 112 million people have compensated cirrhosis, with a global prevalence of 1.3% [4,5]. In Bangladesh, liver disease represents a significant public health challenge, with cirrhosis accounting for a substantial proportion of cases across tertiary-level medical colleges, showing regional prevalence ranging from 22.8% in Sylhet to 69% in Rajshahi [6].

Micronutrients are essential for normal liver function, and deficiencies are common in cirrhosis due to reduced dietary intake, impaired absorption, altered metabolism, and increased urinary losses [7,8]. Zinc, a cofactor for over 300 enzymes, plays a critical role in protein synthesis, wound healing, ammonia detoxification, immune regulation, and hepatic metabolism [8,9]. In cirrhotic patients, zinc deficiency often arises from poor intake, increased hepatic utilization, urinary losses, and hypoalbuminemia, and has been linked to hepatic encephalopathy, immune dysfunction, and poor prognosis [9].

Several studies have consistently shown that zinc deficiency is highly prevalent among cirrhotic patients and closely associated with disease severity. Multicenter data from Japan reported that nearly half of cirrhotic patients had overt zinc deficiency, while up to 90% had subnormal levels, with serum zinc positively correlating with albumin as a marker of hepatic synthetic function [11]. Indian studies further demonstrated reduced zinc levels in patients with hepatic encephalopathy, particularly in advanced stages, highlighting the role of zinc in ammonia metabolism and neurotoxicity [12]. A case–control study from Mymensingh, Bangladesh found significantly lower serum zinc levels in decompensated cirrhosis compared to healthy controls; however, it did not assess associations with disease severity, complications, or clinical outcomes, leaving a clear research gap [13].

This study aimed to assess serum zinc levels in patients with liver cirrhosis and evaluate their association with disease severity, clinical complications, and relevant biochemical parameters, to provide insights for potential therapeutic interventions in the Bangladeshi population.

2. MATERIALS AND METHODS:

2.1 Study Design and Setting:

This was a cross-sectional analytical study conducted from July 2024 to June 2025 in the Department of Biochemistry, Dhaka Medical College, Dhaka.

2.2 Study Population and Sample Size:

The study population consisted of patients diagnosed with liver cirrhosis who attended the Department of Hepatology, Dhaka Medical College Hospital (DMCH), Dhaka. A total of fifty-five (55) patients were included in the study.

2.3 Sampling and Data Collection:

The study employed purposive sampling to select participants.

2.4 Selection criteria

Inclusion criteria:

- Age 18-70 yrs of both genders.
- Patients clinically diagnosed with cirrhosis of liver due to any cause.

Exclusion criteria:

- Age <18 yrs
- Age > 70 yrs
- Pregnant women
- Subjects already on any Zinc containing preparations.

2.5 Data Collection and Laboratory Analysis:

Data was collected through personal interviews using a semi-structured questionnaire. Blood samples were collected aseptically, serum was separated for immediate analysis or stored at –20 °C, and serum zinc levels were measured using standard laboratory methods at the Biochemistry Department, Dhaka Medical College and Hospital.

2.6 Statistical Analysis:

Data from 55 participants were analyzed using SPSS version 26. Continuous variables were expressed as mean ± SD and compared with the independent t-test, while categorical variables were presented as frequencies and percentages and analyzed using the chi-square or Fisher's exact test. A p-value < 0.05 was considered statistically significant.

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2.7 Ethical Consideration:

The study posed minimal risk, ensured participant anonymity, obtained informed consent, safeguarded participants' rights, and collected only routine blood samples. Ethical clearance was approved by the Ethical Review Committee of Dhaka Medical College.

3. RESULTS:

Table 1: Distribution of Study Participants by Gender and Age Group (N = 55)

Variable	Category	Frequency (n)	Percentage (%)
Gender	Male	37	67
	Female	18	33
Age Group	31–40 years	6	11
	41–50 years	22	40
	Other age groups	27	49

The study included 55 participants, with a predominance of males (67%) compared to females (33%), indicating a male-to-female ratio of approximately 2:1. Regarding age distribution, participants were represented across all age groups, with the largest proportion (40%) falling in the 41–50 years category, suggesting that middle-aged individuals were most commonly involved in the study. The smallest proportion (11%) was observed in the 31–40 years group, indicating relatively fewer younger participants. This distribution highlights a predominance of middle-aged male participants in the study population. (Table 1)

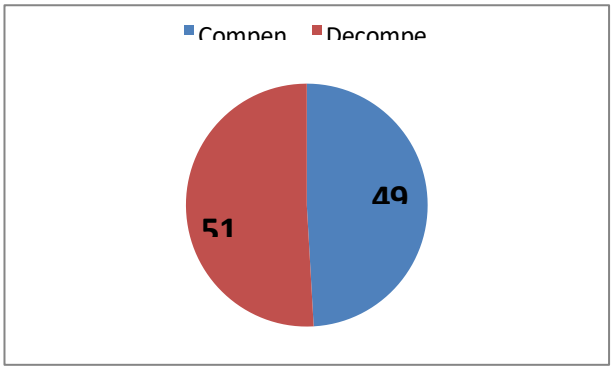

Fig 1: Distribution of Type of Liver Cirrhosis among the Participants (N=55).

Figure 1 illustrates that among the 55 study participants, 27 (49.1%) had compensated liver cirrhosis, while 28 (50.9%) presented with decompensated cirrhosis, indicating an almost equal distribution between the two disease stages.

Table 2: Distribution of Viral Markers and Disease-Related Characteristics Among Study Participants (N = 55)

Variables	Total Number N=55 (%)	Compensated (n=27) n (%)	Decompensated (n=28) n (%)	p-value
Age in years, Mean ± SD	46.20±11.80	46.30±11.40	46.10±12.40	0.95[a]
Range	21-69	24-66	21-69	
Gender				
Male	37 (67)	16 (59)	21 (75)	0.22[b]
Female	18 (33)	11 (41)	7 (25)	
Duration of illness (Years)				
< 1	4 (7)	3 (11)	1 (4)	0.55[b]
1 to <5	47 (86)	22 (81)	25 (89)	
≥ 5	4 (7)	2 (8)	2 (7)	
Viral markers				0.25[b]

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Positive for HBV Virus	41 (75)	22 (81)	19 (68)	
Positive for HCV Virus	14 (25)	5 (19)	9 (32)	

a= Student's unpaired t-test b= Chi square test

The table2 presents the demographic characteristics, disease duration, and viral etiology of the 55 study participants stratified by compensated and decompensated liver cirrhosis. The mean age of the participants was 46.20 ± 11.80 years, with no significant difference between the compensated and decompensated groups (p = 0.95), indicating comparable age distribution. Males constituted the majority of the study population (67%); however, gender distribution did not differ significantly between the two groups (p = 0.22). Most patients had a disease duration of 1 to less than 5 years (86%), and the duration of illness was similar in both groups (p = 0.55). Regarding viral markers, hepatitis B virus infection was more prevalent (75%) than hepatitis C virus infection (25%), with no statistically significant difference in their distribution between compensated and decompensated cirrhosis patients (p = 0.25).

Table 3: Co-morbidities among Study Participants According to Type of Liver Cirrhosis (N=55).

Co-morbidities	Total N=55 %	Compen ated (n=27) n%	Decompensated (n=28) n%	p- Value
DM	30 (54)	10 (37)	20 (71)	0.01 [b]
HTN	38 (68)	15 (56)	23 (82)	0.05 [b]
CKD	34 (61)	9 (33)	25 (89)	<0.001 [b]
IHD	21 (37)	10 (37)	11 (39)	0.87 [b]

b= Chi square test* p-value was calculated by using chi-square test, Fisher's exact test when expected frequencies in any cell of the contingency table are less than 5 for categorical variables

The table 3 shows the distribution of comorbidities among compensated and decompensated cirrhosis patients. Diabetes mellitus was significantly more common in the decompensated group (71%) compared to the compensated group (37%) (p = 0.01). Hypertension was also more frequent among decompensated patients (82%) than compensated patients (56%), reaching borderline statistical significance (p = 0.05). Chronic kidney disease showed a markedly higher prevalence in the decompensated group (89%) compared to the compensated group (33%), with a highly significant difference (p < 0.001). In contrast, ischemic heart disease was similarly distributed between the two groups (39% vs. 37%) and did not show a statistically significant difference (p = 0.87). Overall, diabetes mellitus, hypertension, and chronic kidney disease were significantly associated with decompensated liver cirrhosis.

Table 4: Serum Zinc Levels in Compensated and Decompensated Patients (N = 55)

Variable	Total (N = 55)	Compensated (n = 27)	Decompensated (n = 28)	p-value
Serum Zinc (mg/L)	Mean ± SD	0.64 ± 0.20	0.41 ± 0.14	<0.001[a]
	Range	0.24–0.96	0.14–0.67	
Normal serum zinc level	19 (35%)	10 (37%)	9 (35%)	0.70[b]
Serum zinc deficiency	36 (65%)	17 (63%)	19 (65%)	

a= Student's unpaired t test

b= Chi square test

In this study of 55 liver cirrhosis patients, serum zinc levels were significantly higher in the compensated group (0.64 ± 0.20 mg/L) compared to the decompensated group (0.41 ± 0.14 mg/L; p < 0.001). Overall, 35% of participants had normal zinc levels, while 65% were zinc deficient, with similar proportions in both compensated and decompensated groups (p = 0.70). These findings indicate that zinc deficiency is

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highly prevalent in liver cirrhosis and that lower serum zinc levels are associated with disease decompensation, suggesting its potential role as a marker of disease severity**.** (Table 4)

Table 5: Factors associated with serum zinc level in patients with liver cirrhosis (N=55)

Factors	**Total Number**	**Normal serum zinc level**	**Serum zinc deficiency**	**p-value**
	N=55	(n=19)	(n=36)	
Patients with	(%)	n (%)	n (%)	
Age in years Mean ± SD	47±12	50±12	45±12	0.147[a]
Range	21-69	24-66	21-69	
Gender				
Male	37 (67)	14 (74)	23 (64)	0.462 [b]
Female	18 (33)	5 (26)	13 (36)	
Co-morbidities				
DM	30 (55)	9 (47)	21 (58)	0.571[b]
HTN	38 (69)	6 (32)	32 (90)	<0.001[b]
CKD	34 (62)	7 (37)	27 (75)	0.009[b]
IHD	21 (38)	6 (32)	15 (42)	0.464[b]

a= Student's unpaired t-test, b= Chi square test

The cohort table 5 demonstrates that the mean age of patients with normal serum zinc levels and those with zinc deficiency was comparable (50 ± 12 vs. 45 ± 12 years), with no statistically significant difference (p = 0.147), and a male predominance was observed in both groups without significant gender variation (p = 0.462). Diabetes mellitus and ischemic heart disease were similarly distributed between the two groups (p = 0.571 and p = 0.464, respectively). In contrast, hypertension and chronic kidney disease were significantly more frequent among patients with serum zinc deficiency, with hypertension present in 90% of zinc-deficient patients compared to 32% of those with normal zinc levels (p < 0.001) and chronic kidney disease observed in 75% versus 37%, respectively (p = 0.009), indicating a significant association between zinc deficiency and these comorbid conditions.

4. DISCUSSION:

In our study, males predominated (67%) over females (33%), consistent with Rubin et al. (2020), who reported 39% women among 553,017 hospitalized cirrhosis patients [14]. The mean age of participants was 46 ± 12 years, with the largest proportion (40%) aged 41–50 years, reflecting the chronic progression of cirrhosis and reduced hepatic regenerative capacity with advancing age [1]. Zhou et al. (2019) similarly reported a mean age of 52.8 ± 11.7 years in cirrhotic patients, highlighting that cirrhosis predominantly affects middle-aged adults [15].

Among the 55 participants, 27 (49.1%) had compensated cirrhosis and 28 (50.9%) had decompensated cirrhosis, showing an almost equal distribution between early and advanced stages. Globally, approximately 60–70% of cirrhosis patients remain compensated, with 30–40% progressing to decompensation, which is clinically marked by ascites, variceal bleeding, jaundice, or hepatic encephalopathy [3]. Most patients (86%) had a disease duration of 1–<5 years, aligning with Tsochatzis et al. (2014), who reported that ~70% of cirrhosis cases are within this timeframe, reflecting late presentation or rapid progression, particularly in viral hepatitis [3].

HBV was the predominant etiology in our cohort (75%), while HCV accounted for 25%, consistent with global evidence showing HBV as a more frequent cause of cirrhosis than HCV, with a roughly 2:1 ratio worldwide [16].

Comorbidities were more frequent among decompensated patients: diabetes (71% vs. 37%; p = 0.01), hypertension (82% vs. 56%; p = 0.05), and chronic kidney disease (89% vs. 33%; p < 0.001),

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reflecting the clinical burden of advanced cirrhosis. These findings align with global data reporting high prevalence of diabetes, hypertension, and CKD in advanced liver disease [12,17,18]. Ischemic heart disease prevalence was similar between groups (39% vs. 37%; $p = 0.87$), consistent with previous reports [19].

Serum zinc levels were significantly lower in decompensated patients (0.41 ± 0.14 mg/L) than in compensated patients (0.64 ± 0.20 mg/L; $p < 0.001$), with 65% of participants being zinc deficient. These results are consistent with Kumar et al. (2024), who reported 68% zinc deficiency, particularly among patients with hepatic encephalopathy (0.42 ± 0.13 mg/L vs. 0.61 ± 0.19 mg/L), highlighting its association with disease severity [12].

Although patients with zinc deficiency were slightly younger, this difference was not significant ($p = 0.147$), and gender distribution did not differ between groups. Zinc-deficient patients had significantly higher rates of hypertension (90% vs. 32%; $p < 0.001$) and chronic kidney disease (75% vs. 37%; $p = 0.009$), reflecting zinc's role in vascular health, insulin regulation, and inflammation. These findings align with Betrie et al. (2021), demonstrating zinc's vasorelaxant effects, and Ahmad et al. (2024), who reported that zinc deficiency contributes to impaired glucose metabolism and increased cardiovascular and renal comorbidities [20,21]. Betrie et al. (2021), who demonstrated that zinc promotes vasorelaxation via sensory nerves, endothelium, and smooth muscle, and Ahmad et al. (2024), who reported that zinc deficiency is linked to impaired glucose metabolism and higher diabetes prevalence, highlighting its contribution to cardiovascular and renal comorbidities in cirrhosis [20,21].

5. CONCLUSION:

This study found that liver cirrhosis mainly affects middle-aged men, with similar proportions of compensated and decompensated disease. HBV was the most common cause. Decompensated patients had significantly higher rates of diabetes, hypertension, and chronic kidney disease, while ischemic heart disease showed no stage-related difference. Zinc deficiency was common (65%) and strongly associated with decompensation, hypertension, and chronic kidney disease, suggesting it as a marker of disease severity. Regular zinc monitoring, comorbidity management, and consideration of zinc supplementation may improve outcomes in advanced cirrhosis.

Conflict of Interest: No

Abbreviation:

- CKD – chronic kidney disease
- DM – Diabetes Mellitus
- DMCH – Dhaka Medical College Hospital
- HBV – Hepatitis B Virus
- HCV – Hepatitis C Virus
- HTN – Hypertension
- IHD – ischemic heart disease
- SD – Standard Deviation
- SPSS – Statistical Package for the Social Sciences

REFERENCE:

1. Zhou WC, Zhang QB, Qiao L. Pathogenesis of liver cirrhosis. World journal of gastroenterology: WJG. 2014 Jun 21;20(23):7312.
2. Asrani SK, Devarbhavi H, Eaton J, Kamath PS. Burden of liver diseases in the world. Journal of hepatology. 2019 Jan 1;70(1):151-71.
3. Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. The Lancet. 2014 May 17;383(9930):1749-61.
4. Lim YS, Kim WR. The global impact of hepatic fibrosis and end-stage liver disease. Clinics in liver disease. 2008 Nov 1;12(4):733-46.
5. Hsu YC, Huang DQ, Nguyen MH. Global burden of hepatitis B virus: current status, missed opportunities and a call for action. Nature reviews Gastroenterology & hepatology. 2023 Aug;20(8):524-37.
6. Rahman S, Ahmed MF, Alam MJ, Hoque MI, Hussain MM, Kabir AS, Karim MF, Khondokar FA, Mamun-Al-Mahtab AK, Masud MG, uddin Mollick MK. Distribution of liver disease in Bangladesh: a cross-country study. Euroasian journal of hepato-gastroenterology. 2014 Jan 22;4(1):25.
7. Grüngreiff K, Reinhold D, Wedemeyer H. The role of zinc in liver cirrhosis. Annals of hepatology. 2016 Feb 15;15(1):7-16.
8. Llibre-Nieto G, Lira A, Vergara M, Solé C, Casas M, Puig-Diví V, Solé G, Humanes A, Grau L, Barradas JM, Miquel M. Micronutrient deficiencies in patients with decompensated liver cirrhosis. Nutrients. 2021 Apr 10;13(4):1249.
9. Bloom A, Bloom S, Silva H, Nicoll AJ, Sawhney R. Zinc supplementation and its benefits in the management of chronic liver disease: An in-depth literature review. Annals of hepatology. 2021 Nov 1;25:100549.
10. Rocchi E, Borella P, Borghi A, Paolillo F, Pradelli M, Farina F, Casalgrandi G. Zinc and magnesium in liver cirrhosis. European journal

of clinical investigation. 1994 Mar;24(3):149-55.

11. Ozeki I, Arakawa T, Suii H, Tatsumi R, Yamaguchi M, Nakajima T, Kuwata Y, Toyota J. Zinc deficiency in patients with chronic liver disease in Japan. Hepatology Research. 2020 Mar;50(3):396-401.
12. Kumar D, Prasad MK, Kumar S, Aziz T, Prasad ML, Sinha R, Guria RT, Kumar A, Kumar S, Kumar P. Serum zinc level in liver cirrhosis with hepatic encephalopathy and its correlation with different stages of hepatic encephalopathy. Journal of Family Medicine and Primary Care. 2024 Sep 1;13(9):3979-87.
13. Hossain MS, Rahman MM, Shahidullah AS, Sen B, Hoque MR, Paul AR, Saha KR. A Case Control Study on Serum Level of Micronutrients in Decompensated Liver Cirrhosis Patients in Mymensingh, Bangladesh. Community Based Medical Journal. 2019 Aug 5;8(2):10-6.
14. Rubin JB, Sundaram V, Lai JC. Gender differences among patients hospitalized with cirrhosis in the United States. Journal of clinical gastroenterology. 2020 Jan 1;54(1):83-9.
15. Zhang Y, Luo M, Ming Y. Global burden of cirrhosis and other chronic liver diseases caused by specific etiologies from 1990 to 2021. BMC gastroenterology. 2025 Sep 19;25(1):641.
16. Alberts CJ, Clifford GM, Georges D, Negro F, Lesi OA, Hutin YJ, de Martel C. Worldwide prevalence of hepatitis B virus and hepatitis C virus among patients with cirrhosis at country, region, and global levels: a systematic review. The Lancet Gastroenterology & Hepatology. 2022 Aug 1;7(8):724-35.
17. Lee WG, Wells CI, McCall JL, Murphy R, Plank LD. Prevalence of diabetes in liver cirrhosis: A systematic review and meta-analysis. Diabetes/metabolism research and reviews. 2019 Sep;35(6):e3157.
18. Harinstein ME, Gandolfo C, Gruttadauria S, Accardo C, Crespo G, VanWagner LB, Humar A. Cardiovascular disease assessment and management in liver transplantation. European heart journal. 2024 Nov 1;45(41):4399-413.
19. Girleanu I, Trifan A, Huiban L, Muzica C, Petrea OC, Singeap AM, Cojocariu C, Chiriac S, Cuciureanu T, Costache II, Stanciu C. Ischemic heart disease and liver cirrhosis: Adding insult to injury. Life. 2022 Jul 12;12(7):1036.
20. Betrie AH, Brock JA, Harraz OF, Bush AI, He GW, Nelson MT, Angus JA, Wright CE, Ayton S. Zinc drives vasorelaxation by acting in sensory nerves, endothelium and smooth muscle. Nature communications. 2021 Jun 1;12(1):3296.
21. Ahmad R, Shaju R, Atfi A, Razzaque MS. Zinc and diabetes: a connection between micronutrient and metabolism. Cells. 2024 Aug 15;13(16):1359.