ORIGINAL RESEARCH

Assessment of CRP value in patients with liver cirrhosis

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ABSTRACT

Background: Hepatitis is inflammation of the liver, usually caused by viral infections (hepatitis A, B, C, etc.), excessive alcohol consumption, or autoimmune diseases. Cirrhosis is chronic liver damage leading to the formation of scar tissue, which can impair liver function. The present study was conducted to assess CRP value in patients with liver cirrhosis. **Materials & Methods:**74 patients with known cirrhosisof both genderswere divided into 2 groups. Patients in group I had compensated cirrhosis and group II haddecompensated cirrhosis.Patients were assessed by means of transabdominal ultrasound.Parameters such as etiology, IMC, IVC, serum creatinine, INR, bilirubin, CRP, and MELD was recorded. **Results:** In group I, males were 27 and females were 10 and in group II males were 22 and females were 15. Viral etiology was in 13 in group I and 12 in group II and non- viral etiology in 24 in group I and 25 in group II. The mean IMC was 0.002 in group I and 0.003 in group II. The mean inferior vena cava diameter was 21.4 in group I and 19.8 in group II. The mean serum creatinine level was 0.97 in group I and 1.3 in group II. INR was 1.45 in group I and 1.63 in group II. The bilirubin level was 1.36 in group I and 2.24 in group II. CRP level was 1.74 in group I and 1.57 in group II. MELD was 13.2 in group I and 17.1 in group II. The difference was significant (P< 0.05). Among survivors and dead, viral etiology was in 29% and 32% and non- viral etiology in 71% and 68%. The mean IMC was 0.002 and 0.003. The mean inferior vena cava diameter was 20.6 and 21.5. The mean serum creatinine level was 0.97 and 1.32. INR was 1.5 and 1.7. The bilirubin level was 1.7 and 2.3. CRP level was 1.06 and 1.35. MELD was 14.2 and 17.8 respectively. The difference was significant (P< 0.05). **Conclusion:** C-reactive protein represents a prognostic factor for cirrhosis evolution.The value of CRP influences the decompensation rates.

Keywords: Cirrhosis, CRP, Liver disease

INTRODUCTION

Liver disease is a broad term that encompasses a range of conditions affecting the liver's structure or function.[1] Hepatitis is inflammation of the liver, usually caused by viral infections (hepatitis A, B, C, etc.), excessive alcohol consumption, or autoimmune diseases.Cirrhosis ischronic liver damage leading to the formation of scar tissue, which can impair liver function.[2] It can be caused by long-term alcohol abuse, chronic viral hepatitis, fatty liver disease, or other factors.Fatty liver disease is accumulation of fat in the liver, which can lead to inflammation and liver damage. It can be caused by obesity, diabetes, high cholesterol, or excessive alcohol consumption.[3]

In clinical practice, the C-reactive protein (CRP) is utilized as an inflammatory marker to track a number of disorders, including cancer, autoimmune diseases, and acute infectious diseases. The CRP remains beneficial in cases of cirrhosis as well.[4] Advanced stages of C-reactive protein synthesis are maintained in spite of the decline in liver function. C-reactive protein is thought to be a proxy indicator of bacterial infections and either acute or persistent systemic inflammation.[5] Systemic inflammatory response syndrome (SIRS) is linked to problems from portal hypertension (PHT) and is an independent predictor for survival with or without documented bacterial infection. The pathophysiology of cirrhosis as shown by the vasodilation theory does not fully explain some characteristics of the disease; these are explained by the inflammation theory.[6]The present study was conducted to assess CRP value in patients with liver cirrhosis.

MATERIALS & METHODS

The present study consisted of 74 patients with known cirrhosisof both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Patients were divided into 2 groups. Patients in group I had compensated cirrhosis and group II had decompensated cirrhosis. Patients were assessed by means of transabdominal ultrasound.Parameters such as etiology, IMC, IVC, Serum creatinine, INR, bilirubin, CRP, and MELD was recorded.Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Groups	Group I	Group II
Method	compensated cirrhosis	decompensated cirrhosis
M:F	27:10	22:15

Table I shows that in group I, males were 27 and females were 10 and in group II males were 22 and females were 15.

Table II Comparison of parameters

Parameters	Group I	Group II	P value
Viral etiology	13	12	0.01
Non- viral etiology	24	25	
IMC	0.002	0.003	0.75
IVC	21.4	19.8	0.49
Serum creatinine	0.97	1.3	0.02
INR	1.45	1.63	0.05
bilirubin	1.36	2.24	0.01
CRP	1.74	1.57	0.02
MELD	13.2	17.1	0.05

Table II, graph I shows that viral etiology was in 13 in group I and 12 in group II and non- viral etiology in 24 in group I and 25 in group II. The mean IMC was 0.002 in group I and 0.003 in group II. The mean inferior vena cava diameter was 21.4 in group I and 19.8 in group II. The mean serum creatinine level was 0.97 in group I and 1.3 in group II. INR was 1.45 in group I and 1.63 in group II. The bilirubin level was 1.36 in group I and 2.24 in group II. CRP level was 1.74 in group I and 1.57 in group II. MELD was 13.2 in group I and 17.1 in group II. The difference was significant (P< 0.05).

Graph I Comparison of parameters

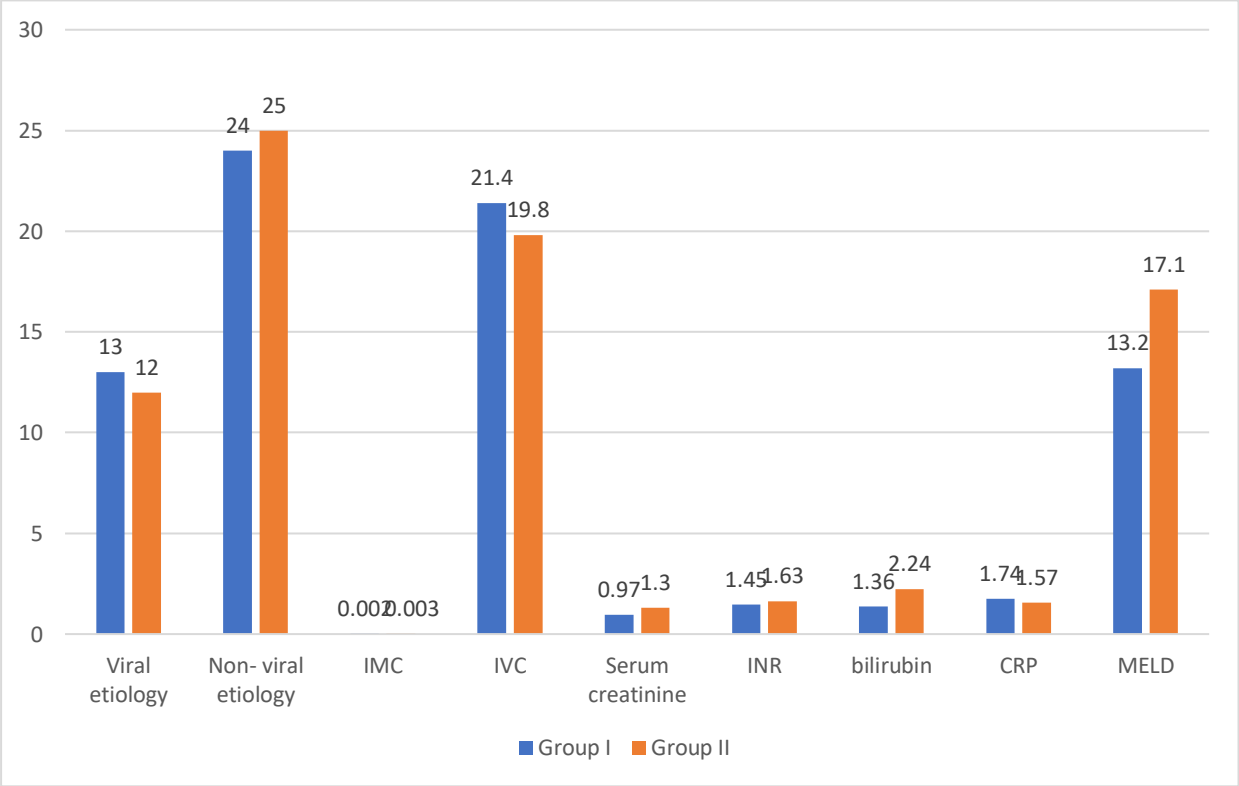

Table III Comparison of parameters among survivors and dead

Parameters	Survivors	Dead	P value
Viral etiology	29%	32%	0.01
Non- viral etiology	71%	68%	
IMC	0.002	0.003	0.35
IVC	20.6	21.5	0.47
Serum creatinine	0.97	1.32	0.02
INR	1.5	1.7	0.62
bilirubin	1.7	2.3	0.61
CRP	1.06	1.35	0.27
MELD	14.2	17.8	0.05

Table III shows that among survivors and dead, viral etiology was in 29% and 32% and non- viral etiology in 71% and 68%. The mean IMC was 0.002 and 0.003. The mean inferior vena cava diameter was 20.6 and 21.5. The mean serum creatinine level was 0.97 and 1.32. INR was 1.5 and 1.7. The bilirubin level was 1.7 and 2.3. CRP level was 1.06 and 1.35. MELD was 14.2 and 17.8 respectively. The difference was significant (P< 0.05).

DISCUSSION

Symptoms of liver disease can vary depending on the specific condition and its severity, but they may include fatigue, jaundice (yellowing of the skin and eyes), abdominal pain or swelling, nausea, vomiting, and changes in appetite or weight.[7,8] Treatment options also depend on the underlying cause and may include medication, lifestyle changes, or in severe cases, liver transplantation. Early diagnosis and management are crucial for preventing complications and improving outcomes.[9,10]The present study was conducted to assess CRP value in patients with liver cirrhosis.

We found that in group I, males were 27 and females were 10 and in group II males were 22 and females were 15. State[11]determined the use of CRP value in the prognosis of 102 patients with cirrhosis.The results showed that the mean CRP value was 0.7+/0.09 mg/dL (CI 0.59-0.90) in patients who did not decompensate throughout the trial and 1.58+/0.4 mg/dL (CI 1.76-2.30) in those who did decompensate, with a statistically significant difference (p=0.045).In rehospitalized patients versus those without any hospitalization, the mean CRP values were 1.35 mg/dL and 0.8 mg/dL, respectively (p=0.032). The increased values of this parameter were positively correlated with the number of hospitalizations (rs=0.35, p=0.05).A CRP value below the threshold of 0.62 mg/dL indicates a smaller probability of future decompensation in liver cirrhosis patients[12].

We found that viral etiology was in 13 in group I and 12 in group II and non- viral etiology in 24 in group I and 25 in group II. The mean IMC was 0.002 in group I and 0.003 in group II. The mean inferior vena cava diameter was 21.4 in group I and 19.8 in group II. The mean serum creatinine level was 0.97 in group I and 1.3 in group II. INR was 1.45 in group I and 1.63 in group II. The bilirubin level was 1.36 in group I and 2.24 in group II. CRP level was 1.74 in group I and 1.57 in group II. MELD was 13.2 in group I and 17.1 in group II. We found that among survivors and dead, viral etiology was in 29% and 32% and non- viral etiology in 71% and 68%. The mean IMC was 0.002 and 0.003. The mean inferior vena cava diameter was 20.6 and 21.5. The mean serum creatinine level was 0.97 and 1.32. INR was 1.5 and 1.7. The bilirubin level was 1.7 and 2.3. CRP level was 1.06 and 1.35. MELD was 14.2 and 17.8 respectively. Di Martino et al[13]assessed the prognostic value of a model combining the variation of C-reactive protein (CRP) levels within 15 days, the Model for End-Stage Liver Disease (MELD) score, and the presence of comorbidities in patients with decompensated cirrhosis with a Child-Pugh score > B7 and to test the relevance of this model in patients with compensated cirrhosis. In these patients with severe cases, the 3-month mortality was independently predicted by the MELD score and a CRP level > 32 mg/L at the baseline and on day 15. This model was better than MELD alone. In the whole population with cirrhosis, the 3-month mortality was also predicted by high MELD scores and a CRP level > 10 mg/L at the baseline and on day 15, but the AUROCs of the 3-variable model and the MELD score alone were no longer significantly different (0.89 versus 0.88, not significant).

The limitation of the study is the small sample size.

CONCLUSION

In summary, our study underscores the potential utility of C-reactive protein (CRP) as a prognostic marker in liver cirrhosis. We observed significant differences in CRP levels between compensated and decompensated cirrhosis, as well as correlations with various clinical parameters and patient outcomes. While acknowledging the study's limitations, including its small sample size, these findings highlight CRP's role in predicting mortality risk and informing patient management strategies. Further research with larger cohorts is needed to validate these findings and elucidate CRP's precise role in liver cirrhosis management, ultimately contributing to more personalized and effective patient care.

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