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Correlation Between Serum Prolacatin and Severity of Liver Cirrhosis

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Abstract **Review Article**

Background and Aims: Hyperprolactinemia has been reported in patients with cirrhosis, but its relationship to disease severity remains incompletely defined. We aimed to evaluate serum prolactin levels in patients with cirrhosis and to correlate them with clinical, biochemical, and prognostic parameters. ***Methods:*** This was a prospective observational study of 50 patients with cirrhosis. Serum prolactin levels were measured and correlated with Child-Pugh class, MELD-Na score, and individual markers of liver dysfunction. Associations were assessed using one-way ANOVA, Kruskal-Wallis test and Pearson correlation as appropriate. ***Results:*** The mean age was 51.98 ± 11.38 years with a male predominance (88%). Ascites and hepatic encephalopathy were present in 100% and 34%, respectively. The majority were Child-Pugh class C (72%) with a mean MELD-Na of 24.16 ± 8.12. Mean serum prolactin was elevated at 28.58 ± 26.89 ng/mL. Prolactin increased significantly across Child-Pugh classes (11.46, 26.24,37.66 ng/mL for Class A, B, C; p = 0.023) and trended up with higher MELD-Na, but did not correlate with individual biochemical parameters. Alcohol was the predominant etiology (80%). ***Conclusions:*** Serum prolactin is significantly elevated in cirrhosis and increases stepwise with worsening Child-Pugh class. Prolactin may serve as an adjunctive biomarker of disease severity in cirrhosis. Further studies are needed to validate these findings and to evaluate the clinical utility of prolactin in this setting.

Keywords Cirrhosis; Hyperprolactinemia; Child-Pugh score; MELD-Na; Disease severity.

INTRODUCTION

Liver cirrhosis is a chronic, progressive condition characterized by the replacement of healthy liver tissue with fibrous scar tissue, leading to the formation of abnormal nodules and liver dysfunction [1]. This process is a result of persistent liver injury from various etiologies, such as chronic alcohol consumption, viral hepatitis, non-alcoholic fatty liver disease, and autoimmune disorders [2]. As the disease progresses, it can lead to severe complications, including portal hypertension, ascites, hepatic encephalopathy, and liver failure [3]. To better understand the severity and prognosis of liver cirrhosis, several scoring systems have been developed, such as the Child-Pugh score and the Model for End-stage Liver Disease (MELD) score [4]. These tools help clinicians assess the extent of liver dysfunction and guide treatment decisions. However, there is an ongoing search for additional biomarkers that can provide further insights into the disease's severity and progression.

Aims and Objectives

Primary objective - To study the correlation between serum prolactin and severity of liver cirrhosis

Secondary objective - To assess the utility of prolactin in early diagnosing cirrhosis

CLINICAL PRESENTATION

Chief Complaint Number of Patients (%)
Abdominal Distension 40 (80%)
Pedal Edema 24 (48%)
Jaundice 10 (20%)
Altered Sensorium 8 (16%)
Hematemesis 4 (8%)
Pain Abdomen 4 (8%)
Fever 3 (6%)
Breathlessness 3 (6%)
Malena 3 (6%)
Others 10 (20%)

Complication Number of Patients (%)
Ascites
Absent 0 (0%)
Mild 13 (26%)

Moderate 18 (36%)
Gross 19 (38%)

Hepatic Encephalopathy
Absent 33 (66%)
Present 17 (34%)

Liver Function Tests

Test	Mean ± SD Median (IQR)
Serum Bilirubin	6.40 ± 7.47 3.31 (1.53 - 7.57)
Serum Albumin	2.54 ± 0.53 2.50 (2.20 - 2.80)
INR	2.27 ± 1.60 1.87 (1.56 - 2.45)

Mean serum creatinine was mildly elevated at 1.35 mg/dL (SD 0.91), median 1.05 mg/dL (IQR 0.78 - 1.44). 40% of patients had an abnormally high creatinine above 1.2 mg/dL, suggestive of renal dysfunction.

Average serum sodium was 131.68 mEq/L (SD 6.73), median 133 mEq/L (IQR 128 - 135). Hyponatremia (sodium < 135 mEq/L) was present in the majority (62%) of patients.

The mean Child-Pugh score was 10.94 (SD 1.92), median 11 (IQR 10-12), consistent with advanced liver disease. The average MELD-Na score was also high at 24.16 (SD 8.12), median 23 (IQR 18-29).

The vast majority of patients (72%) were Child-Pugh class C, indicating severe hepatic dysfunction. 26% were class B and only 2% class A.

Mean serum prolactin was elevated at 28.58 ng/mL (SD 26.89), median 20.63 ng/mL (IQR 17.18 - 32.41). Levels ranged from 3.17 to 179.10 ng/mL.

Average serum prolactin increased with worsening Child-Pugh class, from 11.46 ng/mL in the single class A patient to 26.24 ng/mL in class B and 37.66 ng/mL in class C. This difference was statistically significant ($p = 0.023$) by one-way ANOVA.

Mean serum prolactin trended up with higher MELD-Na scores, from 22.50 ng/mL for MELD-Na 10-19, to 30.56 ng/mL for MELD-Na 20-29, and 33.94 ng/mL for MELD-Na 30-40. However, this difference did not reach statistical significance ($p = 0.091$) by the Kruskal-Wallis test. No patients had a MELD-Na score under 10.

All patients (100%) had portal hypertension as a complication of cirrhosis. Other comorbidities were much less common, including cholelithiasis (8%), acute kidney injury (8%), cystitis (4%), and singular cases of ischemic heart disease, umbilical hernia, situs inversus, cholangiocarcinoma, esophageal candidiasis, portal hypertensive gastropathy, and coagulopathy (2% each).

Etiology of cirrhosis

Alcohol was by far the leading cause of cirrhosis, accounting for 80% of cases. Viral hepatitis was the etiology in 12%, non-alcoholic steatohepatitis (NASH) in 6%, and 2% were cryptogenic. In this prospective observational study of 50 patients with liver cirrhosis, the mean age was 52 years with a male predominance (88%). The most common presenting symptoms were abdominal distension (80%), pedal edema (48%), jaundice (20%) and altered mental status (16%). All patients had ascites and 34% had overt hepatic encephalopathy. Laboratory parameters revealed hyperbilirubinemia (mean 6.40 mg/dL), hypoalbuminemia (mean 2.54 g/dL), coagulopathy (mean INR 2.27), renal impairment (40% with creatinine > 1.2 mg/dL) and hyponatremia (62%). The majority of patients had advanced disease, with 72% classified as Child-Pugh C (mean score 10.94) and a mean MELD-Na score of 24.16. Serum prolactin levels were elevated, with a mean of 28.58 ng/mL. Prolactin increased significantly across worsening Child-Pugh classes ($p = 0.023$), and 0 5 10 15 20 25 30 35 40 45 Alcohol Viral Hepatitis NASH Cryptogenic Number of Patients Page 114 of 134 - Integrity Submission Submission ID trn:oid:::3618:87550336 Page 114 of 134 - Integrity Submission Submission ID trn:oid:::3618:87550336 108 | Page showed an upward trend with higher MELD-Na scores that did not reach statistical significance ($p = 0.091$). Weak correlations were seen between prolactin and liver disease parameters, but none were significant. All patients had portal hypertension, with other comorbidities being infrequent. Alcohol was the predominant etiology (80%), followed by viral hepatitis (12%) and NASH (6%). Ultrasonography was universally used for diagnosis, with endoscopy, CT and Fibroscan used selectively. This study of decompenated cirrhotics found elevated prolactin levels that increased with disease severity. However, no significant correlation was found between prolactin and any individual disease parameter. Larger studies are needed to further elucidate the relationship between prolactin and liver disease and evaluate its potential as a severity biomarker.

DISCUSSION

In this single-center prospective observational study, we aimed to elucidate the relationship between serum prolactin levels and severity of liver disease in patients with cirrhosis. Our key findings can be summarized as follows:

1. Serum prolactin levels were significantly elevated in cirrhotic patients compared to normal reference ranges
2. Prolactin levels showed a statistically significant stepwise increase across worsening Child-Pugh classes
3. Prolactin trended up with increasing MELD-Na scores, although this did not reach statistical significance

4. No significant correlations were found between prolactin levels and individual clinical or biochemical parameters of liver dysfunction

SUMMARY

This prospective observational study enrolled 50 patients with cirrhosis to evaluate the relationship between serum prolactin levels and liver disease severity. The study population had a mean age of 51.98 ± 11.38 years, with a male predominance (88%).

The most common presenting symptoms were abdominal distension (80%), pedal edema (48%), jaundice (20%), and altered sensorium (16%).

All patients had ascites on examination, with 38% having gross ascites. Hepatic encephalopathy was present in 34%.

Laboratory evaluation revealed hyperbilirubinemia (mean 6.40 mg/dL), hypoalbuminemia (mean 2.54 g/dL), and coagulopathy (mean INR 2.27).

Renal dysfunction (creatinine > 1.2 mg/dL) was noted in 40% and hyponatremia (sodium < 135 mEq/L) in 62% of patients.

The majority of patients had advanced liver disease, with 72% classified as Child-Pugh C (mean score 10.94 ± 1.92) and a mean MELD-Na score of 24.16 ± 8.12.

Serum prolactin levels were significantly elevated, with a mean of 28.58 ± 26.89 ng/mL and median of 20.63 ng/mL (IQR 17.18 - 32.41).

Prolactin levels showed a statistically significant stepwise increase across worsening Child-Pugh classes ($p = 0.023$), with means of 11.46 ng/mL, 26.24 ng/mL, and 37.66 ng/mL in Child A, B, and C respectively.

Prolactin trended up with increasing MELD-Na scores, from 22.50 ng/mL for MELD-Na 10-19, to 30.56 ng/mL for MELD-Na 20-29, and 33.94 ng/mL for MELD-Na 30-40, but this did not reach statistical significance ($p = 0.091$).

No significant correlations were found between prolactin levels and individual biochemical parameters like bilirubin ($r = -0.12$), albumin ($r = 0.10$), INR ($r = -0.263$), creatinine ($r = 0.007$), or sodium ($r = -0.175$).

All patients had portal hypertension as a complication. Other comorbidities were infrequent, with cholelithiasis and acute kidney injury being the most common (8% each). Alcohol was the predominant etiology of cirrhosis, accounting for 80% of cases, followed by viral hepatitis (12%) and non-alcoholic steatohepatitis (6%).

Ultrasonography was the primary diagnostic modality (100%), with selective use of endoscopy (10%), CT scan (4%), and Fibroscan (2%).

CONCLUSION

In this prospective observational study of 50 patients with cirrhosis, we found significantly elevated serum prolactin levels compared to normal reference ranges. Prolactin showed a statistically significant stepwise increase across worsening Child-Pugh classes ($p = 0.023$) and trended up with higher MELD-Na scores, although the latter did not reach statistical significance ($p = 0.091$). Despite these associations with disease severity, prolactin did not correlate significantly with any individual biochemical marker of liver dysfunction. Our findings add to the growing body of evidence linking hyperprolactinemia to advanced cirrhosis. The precise mechanisms underlying this association remain to be fully elucidated, but likely involve a complex interplay of hepatocellular dysfunction, altered hormone metabolism, and associated physiological stressors. While our study is limited by its modest sample size and single-center design, it nonetheless provides compelling data on the relationship between prolactin and liver disease severity. Further research is needed to validate these findings in larger, more diverse cohorts and to evaluate the potential utility of prolactin as a biomarker in cirrhosis. In the interim, our results underscore the importance of considering liver disease as a potential cause of hyperprolactinemia, particularly in patients with advanced cirrhosis. Elevated prolactin in this setting should prompt a thorough evaluation of liver function and disease severity, rather than being assumed to represent a primary endocrine disorder. In conclusion, serum prolactin is significantly elevated in patients with cirrhosis and increases in a stepwise fashion with worsening disease severity as assessed by the Child-Pugh classification. While further research is needed to fully define the clinical implications of this relationship, our findings suggest that prolactin has potential as an adjunctive biomarker in the assessment and prognostication of chronic liver disease. Integration of prolactin into the existing framework of severity scores and prognostic models could help refine our understanding and management of this complex disorder.

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