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Research Article

Association Between Serum Prolactin Levels and Severity of Liver Cirrhosis Assessed by Child–Pugh Scoring: A Hospital-Based Cross-Sectional Study

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ABSTRACT

Introduction: Liver cirrhosis represents the terminal stage of chronic liver diseases and is characterized by progressive fibrosis and regenerative nodules, resulting in substantial morbidity and mortality worldwide. Disease prognosis worsens markedly following decompensation. Prolactin, a pituitary hormone primarily regulated by dopamine, is increasingly recognized in this context, as cirrhosis is associated with elevated circulating oestrogen levels that stimulate prolactin secretion through hypothalamic and pituitary mechanisms. Therefore, serum prolactin may have potential as an early marker for disease severity and complications in liver cirrhosis.

Aim: To evaluate the correlation between serum prolactin levels and disease severity, as assessed by the Child–Pugh scoring system, among patients with liver cirrhosis.

Materials and Methods: A hospital-based observational study was conducted among 40 patients with liver cirrhosis at the tertiary care center of Mahatma Gandhi Medical College and Research Institute (MGMCRI), Puducherry. Serum prolactin levels were measured and correlated with Child–Pugh scores. Statistical analysis was performed using SPSS version 22 for Windows.

Results: The median Child–Pugh score was 10.5 (interquartile range: 9.0–12.0), with 10.0%, 35.0%, and 45.0% of patients classified as Child–Pugh classes A, B, and C, respectively. The median serum

prolactin level was 55.2 ng/dl (29.6–79.8), ranging from 4 ng/dl to 99 ng/dl. A strong and statistically significant positive correlation was observed between serum prolactin levels and Child–Pugh scores ($p < 0.001$).

Conclusion: Elevated serum prolactin levels in patients with liver cirrhosis show a strong association with disease severity as assessed by the Child–Pugh score and may serve as a useful indicator of advanced disease. Nevertheless, larger multicentric and longitudinal studies are warranted to validate these findings and to further explore their prognostic significance.

Keywords: L**iver, Cirrhosis, Prolactin, Child–Pugh Score, Disease Severity,** B**iomarkers**

INTRODUCTION

Liver cirrhosis, the end-stage of various chronic liver diseases, is hallmarked by the replacement of liver tissue by fibrotic scar tissue and regenerative nodules, leading to significant morbidity and mortality worldwide. It is a progressive condition characterized by the destruction of the liver architecture and impairment of liver function.[1] Cirrhosis is currently a leading cause of fatalities related to liver conditions. It marks the advanced stage of progressive liver fibrosis, characterized by the distortion of the liver's architecture, and without a liver transplant, it can result in death. In 2017, over 1.32 million people died from cirrhosis worldwide, an increase from approximately 899,000 in 1990. Cirrhosis accounted for 2.4% of all global deaths in 2017, up from 1.9% in 1990. Southeast Asia ranked fifth in terms of the age-standardized death rate from cirrhosis among regions in 2017, with a rate of 29.5 per 100,000.[2] The cause remains unknown in about half of the patients. In India, according to the latest World Health organisation (WHO) data published, liver disease deaths in India reached 268,580 or 3.17% of total deaths, which accounts for one-fifth (18.3%) of all cirrhosis deaths globally.[3] The risk of mortality and health complications associated with cirrhosis significantly escalates when decompensation occurs. The initial decompensatory episode in patients with chronic liver cirrhosis marks a critical juncture in the disease trajectory, indicating a sharp decrease in median survival times from a decade or more to merely one to two years.[4]

Liver cirrhosis disrupts normal liver functions, leading to systemic effects because of the organ's vital role in metabolism, synthesis, detoxification, and hormonal balance. Such disruptions extend

to the endocrine system, largely due to the liver's reduced capacity to metabolize and excrete hormones.[5] The complex pathophysiology associated with hormonal disturbances in cirrhosis involves not just diminished hormone clearance, but also alterations in hormone production and the feedback loops that regulate them.[6]

A hormone that exemplifies this phenomenon is prolactin. Human prolactin is presently regarded as a pituitary hormone, the synthesis and serum concentrations of which are regulated by dopamine. Its physiological effects are confined to lactation and reproductive functions. Extensive debates have revolved around prolactin levels in patients with hepatic dysfunction.[7] The elevation of prolactin primarily results from a reduction in dopamine levels within the tuberoinfundibular tract.[8]

The secretion of prolactin is predominantly under the control of continuous inhibition from the hypothalamus via dopamine, alongside the stimulating effects of hypothalamic releasing factors and the presence of circulating estrogens.[9] In the context of liver cirrhosis, there is an increase in circulating oestrogens due to heightened conversion of testosterone to oestrogen through androstenedione in the periphery, and to a lesser extent, because of reduced elimination by the liver.[10] These oestrogens stimulate the release of prolactin by interfering with the regulation of dopamine secretion from the hypothalamus and by directly affecting the anterior pituitary gland.[11] Moreover, Impaired liver function alters amino acid profiles, increasing aromatic types that promote pseudo-neurotransmitter production, potentially suppressing dopamine release and raising prolactin levels.[12]

The Child-Pugh score remains a widely used clinical tool to assess the prognosis of liver cirrhosis, stratifying patients into classes A through C based on clinical signs and laboratory tests and is used to assess the long-term severity of liver disease and predicts mortality in patients with liver cirrhosis.[13] However, the score does not account for hormonal alterations, an aspect that could provide additional prognostic information. The existing literature indicates a potential link between endocrine dysregulation and liver cirrhosis outcomes, but significant gaps remain, particularly in understanding how serum prolactin levels relate to the severity of liver dysfunction.

The rising prevalence of liver cirrhosis, especially in Asia, challenges the predictive power of traditional measures like the Child-Pugh score in forecasting cirrhosis-related complications. Therefore, integrating a biomarker such as prolactin into clinical practice could provide a more nuanced understanding of disease progression and potential complications,

offering a chance for timely medical intervention. Given the limited research examining the link between serum prolactin levels and the Child-Pugh score, there is a clear need to investigate this potential relationship further. Additionally, this study aims to establish serum prolactin as a preliminary marker for detecting complications in patients with liver cirrhosis.

SUBJECTS AND METHODS

A hospital-based observational study was conducted between 2022 and 2024 among patients diagnosed with cirrhosis of the liver and admitted to the Department of General Medicine at Mahatma Gandhi Medical College and Research Institute (MGMCRI), Puducherry. The study included patients aged 18 years and above who were diagnosed with cirrhosis of the liver and/or its complications and were admitted through the Medicine Outpatient Department, Medical Gastroenterology Outpatient Department, or casualty services to the General Medical Ward. Patients were excluded if they had a history of cranial surgery or irradiation, chest wall trauma, pituitary or hypothalamic disease, chronic renal failure, herpes zoster, were receiving medications known to elevate prolactin levels (such as neuroleptics, metoclopramide, methyldopa, reserpine, cyproterone acetate, morphine, cimetidine, metiamide, or oral contraceptive pills), or had prolactin levels exceeding 100 ng/L. The eligible patients were selected by convenience sampling during the study duration until the sample size was reached.

SAMPLE SIZE:

Since your main outcome is correlation between prolactin and Child–Pugh, this is actually better:

$$n = \frac{(Z_\alpha + Z_\beta)^2}{\left(0.5 \times \ln \frac{1+r}{1-r}\right)^2} + 3$$

Where:

- Expected correlation r = 0.45 (moderate correlation from literature)
- Zα = 1.96 (95% confidence)
- Zβ = 0.84 (80% power)

$n \approx 38$

After obtaining written informed consent from the patient or a legally acceptable relative, a detailed clinical evaluation was carried out, including history taking, physical examination with assessment of vital parameters, height, weight, and body mass index calculated using Asian Indian guidelines, along with evaluation for clinical signs of liver cell failure and a complete gastrointestinal system examination. Following this, all patients underwent routine investigations as part of the standard cirrhosis workup, including complete blood count, blood urea and creatinine with electrolyte panel, liver function tests, coagulation profile, and viral markers (HBsAg, HCV, and HIV antibodies) assessed using the ELISA method. Additional investigations such as stool occult blood testing and ascitic fluid analysis (including glucose, protein, cytology, and microbiological cultures) were performed based on the patient's clinical condition. Ultrasonography of the abdomen was routinely done to assess liver size and echotexture, splenic enlargement, portal vein diameter, and the presence of ascites, and patients with evidence of portal hypertension and splenomegaly were further evaluated with upper gastrointestinal endoscopy to rule out gastroesophageal varices as part of routine clinical care.

For research purposes, an additional 2 ml of blood was collected on the day of admission for estimation of serum prolactin levels. After completion of all routine investigations, disease severity was classified using the modified Child–Turcotte–Pugh scoring system, and the presence of complications such as ascites (graded clinically and radiologically), portal hypertension, esophageal varices, hepatic encephalopathy (graded using the West Haven criteria), hepatorenal syndrome, and spontaneous bacterial peritonitis was documented. The independent variables included age, sex, anthropometric measurements, BMI, liver function parameters, serum prolactin levels, ultrasonographic findings, and hepatic encephalopathy grading, while the dependent variables comprised complications of cirrhosis, Child–Pugh scores, and the correlation between serum prolactin levels and Child–Pugh scores.

STATISTICAL ANALYSIS

Data was analyzed using SPSS V22 for Windows. Categorical variables are expressed as frequency and percentages. Continuous variables are expressed as mean (SD) or median (IQR), depending on the type of distribution. Prevalence of encephalopathy is expressed as percentage with 95% confidence interval (95% CI). Pearson correlation was used to determine

the correlation of age and prolactin levels with Child Pugh Score. Independent samples t test was used to determine the association between gender and Child Pugh Score and Mann Whitney U test was used to determine the association between encephalopathy and prolactin levels. The correlation between the grades of encephalopathy and prolactin levels was determined using Spearman correlation. A p-value of less than 0.05 was considered statistically significant.

Ethical approval

Ethical approval was taken from the Institutional Human Ethics Committee, MGMCRI before the start of the study. Informed consent was obtained from the study participants or caretakers of the study participants before data collection. Confidentiality was maintained by limiting the identifying variables to a minimum. Data was analyzed in aggregate and access to the collected data was limited only to me, my guide and co-guide.

RESULTS

Table 1: Distribution of the participants by age category, Grade of encephalopathy, Grade of ascites and Child Pugh Score class

Age category in years	**Frequency (n)**	**Percentage**
≤45	15	37.5
46-60	17	42.5
>60	8	20.0
Gender		
Male	39	97.5
Female	1	2.5
Grade of encephalopathy*		
Minimal	14	35.0
Grade 1	9	22.5
Grade 2	10	25.0
Grade 3	2	5.0
Grade 4	5	12.5
Grade of ascites		
Mild	12	30.0
Moderate	19	47.5
Large	8	20.0

Tense	1	2.5
Child Pugh Score class		
A	4	10.0
B	14	35.0
C	22	55.0

A total of 40 patients with liver cirrhosis was included in the study. The mean age of the study participants was 51.1 (11.1) years. The median age of the study participants was 50.0 (41.5-57.75) years with a minimum of 34 years and a maximum of 75 years. Table 1 shows that the majority of patients (42.5%) belonged to the 46–60 years age group, followed by those aged ≤45 years (37.5%), and the study population was predominantly male, with 97.5% males. Hepatic encephalopathy was present in 65.0% of patients (95% CI: 48.3%–78.9%); among the 26 affected patients, Grade 1, Grade 2, Grade 3, and Grade 4 encephalopathy were observed in 9, 10, 2, and 5 patients, respectively. Ascites was commonly observed, with mild ascites in 30.0%, moderate in 47.5%, gross in 20.0%, and tense ascites in 2.5% of patients. The mean Child–Pugh score was 10.0 ± 2.4, while the median score was 10.5 (interquartile range: 9.0–12.0), ranging from a minimum of 5 to a maximum of 13. As shown in Table 1, Child–Pugh class A, B, and C were present in 10.0%, 35.0%, and 45.0% of patients, respectively, indicating that nearly half of the study population presented with severe liver disease.

Table 2. Liver function test profile and its correlation with Child–Pugh score in patients with liver cirrhosis

Parameter	**Mean (SD)**	**Median (IQR)**
Serum albumin (g/dl)	2.6 (0.5)	2.4 (2.-3.0)
INR	1.9 (0.9)	1.8 (1.2-2.2)
Total bilirubin (mg/dl)	6.6 (6.6)	3.2 (2.0-11.4)
Variables	**Correlation coefficient (r)**	**p value***
Prolactin (ng/dl) vs Child Pugh Score	0.804	<0.001

Variables	Correlation coefficient (r)	p value*
Age in years vs Child Pugh Score	-0.149	0.359

- Table 2 summarizes the liver function test parameters and their association with disease severity among the study participants. The mean serum albumin level was 2.6 ± 0.5 g/dl (range: 1.8–3.8 g/dl), and the mean INR was 1.9 ± 0.9, with values ranging from 0.9 to 6.5. The median total bilirubin level was 3.2 mg/dl (interquartile range: 2.0–11.4), with a minimum of 0.4 mg/dl and a maximum of 23.7 mg/dl. The mean serum prolactin level was 54.6 ± 27.6 ng/dl, and the median level was 55.2 ng/dl (29.6–79.8), ranging from 4 ng/dl to 99 ng/dl. As shown in Table 2 and Figure 14, there was a very strong and statistically significant positive correlation between serum prolactin levels and Child–Pugh scores (r = 0.804; $p < 0.001$), indicating increasing disease severity with rising prolactin levels. In contrast, age did not show a significant correlation with Child–Pugh scores (r = –0.149; p = 0.359). Additionally, serum prolactin levels were significantly higher in patients with hepatic encephalopathy compared to those without encephalopathy (68.0 ng/dl vs. 29.7 ng/dl; $p < 0.001$), and a strong positive correlation was observed between prolactin levels and the grades of hepatic encephalopathy (r = 0.724; $p < 0.001$).
- Table 3. Association of gender with Child–Pugh score and comparison of serum prolactin levels by hepatic encephalopathy status among study participants

Gender	Child Pugh Score		p value
	Mean	SD	
Males	10.2	2.2	0.029
Females	5.0	-	

Encephalopathy	Prolactin level (ng/dl)		p value*
	Mean	SD	
Yes	68.0	22.1	<0.001
No	29.7	17.6	

***Mann Whitney U test**

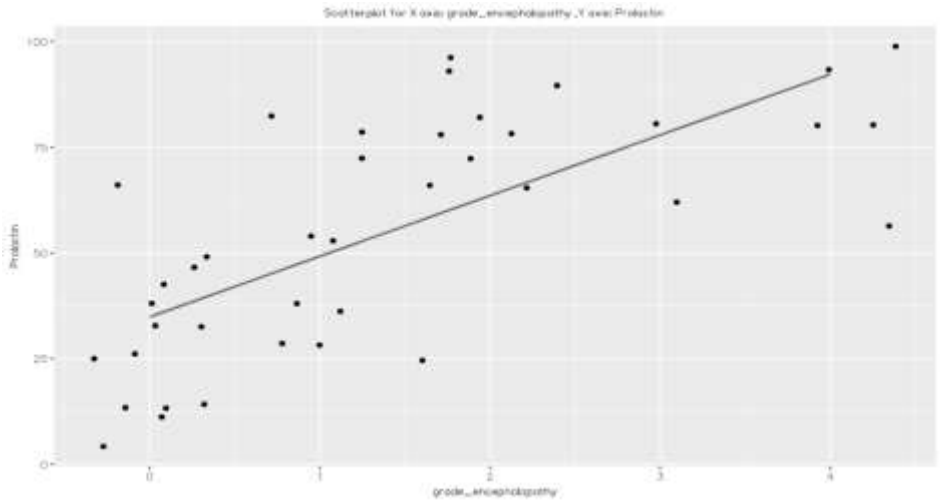

Figure 1: Scatter plot showing the correlation of grades of encephalopathy with prolactin levels (*Spearman correlation)

Table 3 shows that the mean Child–Pugh score was significantly higher among male patients compared to female patients (10.2 vs. 5.0; p = 0.029), indicating more severe liver disease among males in the study population. The table also demonstrates that mean serum prolactin levels were significantly higher in patients with hepatic encephalopathy than in those without encephalopathy (68.0 ± 22.1 ng/dl vs. 29.7 ± 17.6 ng/dl; p < 0.001), as assessed using the Mann–Whitney U test. Figure 1 illustrates a strong positive correlation between the grades of hepatic encephalopathy and serum prolactin levels, with increasing prolactin concentrations observed across higher encephalopathy grades, and this association was statistically significant on Spearman correlation analysis.

DISCUSSION

Cirrhosis, a chronic liver ailment marked by fibrosis and the formation of nodules that hinder hepatic blood flow, is often evaluated using the Child-Pugh score. This score considers factors like bilirubin, albumin levels, prothrombin time, ascites, and hepatic encephalopathy to gauge disease progression. Prolactin, a hormone produced by the pituitary gland, holds immunoregulatory and hepatoprotective functions. Altered prolactin levels have been linked to various liver conditions, hinting at its potential as a marker for hepatic alterations. The existing literature indicates a potential link between endocrine dysregulation and liver cirrhosis outcomes, but significant gaps remain, particularly in understanding how serum prolactin levels relate to the severity of liver dysfunction. Hence, we conducted a study to explore the correlation between serum prolactin levels and the Child-Pugh score which could provide insights into the complex interplay

between hormonal regulation and liver function, ultimately contributing to better patient outcomes in cirrhosis management.

Of 40 patients included in the study, the mean age of the study participants was 51.1 (11.1) years with majority (42.5%) patients in the age group of 46-60 years followed by ≤45 years (37.5%). Almost all except one patient were males. In a study by Balakrishnan CH and Rajeev H [14], the male to female ratio of 5:1 with the 75% of the patients in the age group of 40-50 years of age, which is similar to our study observations. Even in a study by **Metwally RA et al [15,]** the mean age of the study participants was 51.94 years whish also supports our study findings. There are numerous other studies supporting our study findings with respect to age and gender [16-25]. Cirrhosis, a chronic liver disease resulting from long-term liver damage and scarring (fibrosis), tends to be more common among males for several reasons related to both biological and lifestyle factors. Firstly, men are more likely than women to consume alcohol and to drink it in larger quantities and it is well known fact that alcohol abuse is a leading cause of liver cirrhosis [26]. The male body tends to metabolize alcohol differently, and prolonged heavy drinking poses a significant risk for the development of cirrhosis. Secondly, men and women metabolize fats differently, and men are more prone to accumulate fat in the liver, a condition known as non-alcoholic fatty liver disease (NAFLD), which can progress to non-alcoholic steatohepatitis (NASH), a more severe form of liver disease that may lead to cirrhosis [27]. Thirdly, Hepatitis B and C are viral infections that can lead to cirrhosis, where men are at a higher risk of contracting these viruses. The prevalence and progression of these infections to chronic liver disease and cirrhosis can be higher in men. Added to these, certain lifestyle factors like smoking, unhealthy eating habits coupled with genetic, hormonal and delayed health seeking behavior makes males more prone for developing chronic liver disease and fibrosis [28]. With regards to age, Liver diseases, such as hepatitis B and C, fatty liver disease, and alcohol-related liver disease, often progress silently over many years. It takes time for the damage to accumulate to the point where cirrhosis, which is the advanced stage of liver scarring, becomes evident. The timeframe for this progression often spans decades, making middle age a common period for the emergence of cirrhosis symptoms and diagnosis. By the age of 50, many individuals have had prolonged exposure to the risk factors for liver disease, such as chronic alcohol consumption, obesity, and long-standing hepatitis B or C infection.

This prolonged exposure increases the likelihood of developing cirrhosis[29]. This might be the reason for the specific age distribution in our study and other similar studies. Moreover, Child Pugh Score was significantly higher among the males when compared to the females (10.2 vs 5.0; p=0.029). However, there was no significant correlation between age and Child Pugh Score. This observation needs further studies to be proven.

Another observation in our study is that the prevalence of encephalopathy was 65.0% (95% CI: 48.3%-78.9%). Of 26 patients with encephalopathy, Grade 1, Grade 2, Grade 3 and Grade 4 encephalopathy was present 9, 10, 2 and 5 patients respectively. In a study by Animesh D et al[74], hepatic encephalopathy (grades 1-4) was observed among in 71.42% of patients. In a study by Ramani S et al[30], most patients presented with Grade 2 (32%) and Grade 1 (23%) hepatic encephalopathy. A study by Louissaint J et al[31], in their review had reported that the prevalence of encephalopathy among the patients with cirrhosis as 20-3% to 37.0%.[32] The observed higher prevalence of encephalopathy in our study could be due to the higher proportion of severe cases in its late presentation, in our study. The higher proportion of patients with more severity may be due to poor compliance in medication and no change in lifestyle. Moreover, the mean prolactin level was significantly higher among the patients with encephalopathy (68.0 ng/ml vs 29.7 ng/dl; p<0.001) and there was a strong positive correlation between the grades of encephalopathy and prolactin levels and it was found to be statistically significant (r=0.724; p<0.001). The positive correlation between prolactin levels and the severity of hepatic encephalopathy (HE) in cirrhosis patients is attributed to several interrelated factors. Cirrhosis impairs the liver's ability to detoxify blood, leading to the accumulation of substances like ammonia, which adversely affects brain function and contributes to HE.[94] This liver dysfunction also disrupts hormonal balance, affecting the hypothalamic-pituitary axis and leading to elevated prolactin levels.[33] Prolactin release is normally inhibited by dopamine; thus, any imbalance resulting in reduced dopamine activity can cause prolactin levels to rise. Moreover, cirrhosis can cause hormonal metabolism disturbances, including increased estrogen levels, which may further stimulate prolactin release.[59] Physiological stress associated with advanced liver disease may also trigger increased prolactin as part of the body's stress response.16 Interestingly, prolactin might have a neuroprotective role, potentially offering a compensatory mechanism to protect the brain from

damage in HE. Additionally, given its immunomodulatory effects, elevated prolactin levels in cirrhosis patients with HE could reflect an attempt to modulate the inflammation associated with liver disease. This complex interplay highlights the multifaceted relationship between hormonal changes and liver disease progression, emphasizing the need for holistic management approaches in cirrhosis. This hypothesis is being tested and proved by various studies across the globe. For instance, a study by Balakrishnan CH and Rajeev H[34], prolactin levels were increased among all patients suffering from hepatic encephalopathy. In another study by Metwally RA et al[35], patients presenting with hepatic encephalopathy exhibited significantly elevated levels of prolactin compared to those without encephalopathy. Similarly, a study by Vemanamanda S and Srinivasa SV[36], had also concluded that Median prolactin levels were significantly higher in patients with grade 4 hepatic encephalopathy compared to grades 3, 2, and 1 (grade 4: 66.0 [61.5 to 71.5], grade 3: 47.0 [42.0 to 54.0], grade 2: 43.0 [39.25 to 50.5], and grade 1: 40.5 [31.25 to 48.25]), which also supports our study findings. Not surprisingly, the above mentioned hypothesis had also been established by various other studies by Khalil FM et al[37], Sakhnani DR et al[38], Giri R et al[39], Mukherjee S et al, Animesh D et al[24], Ramani S et al[40], Karekar S et al, Sachin S et al[25] and Payer J et al. However, it also recommended to interpret these findings based on clinical significance.

The median Child Pugh Score among the patients was 10.5 (9.0-12.0) with Child Pugh Score class A, B and C among 10.0%, 35.0% and 45.0% patients, respectively. The median prolactin level among the patients was 55.2 (29.6-79.8) ng/dl with a minimum score of 4 ng/dl and a maximum score of 99 ng/dl. There was a very strong positive correlation between prolactin and Child Pugh Score and it was found to be statistically significant ($p<0.001$). For instance, a study by Arafa M et al, had reported that prolactin levels increased as the Child Pugh class increased from A to C, which is in line with our study results where Child Pugh Score increased as Prolactin level increased. The study results of Balakrishnan CH and Rajeev H[34], had revealed that elevated serum prolactin levels were detected in 73% of the patients, with the highest concentrations of serum prolactin (>35 ng/ml) observed in patients classified as Class 'C' according to the Child Pugh scoring system. In a study by Patel HN et al[19], the ROC curve revealed that using a cutoff point of 19.8 ng/ml, prolactin had a sensitivity of 68.94%, specificity of 80.15%, positive predictive value (PPV) of 85.5%, and negative predictive value

(NPV) of 62%. Metwally RA et al[35], in their study also had shown that there was a significant association between prolactin levels and the Child-Pugh score, indicating the dependency of prolactin levels on the severity of liver cirrhosis as assessed by this scoring system. Even in a study by Bhadora A et al[20], elevated prolactin levels were linked to a poorer prognosis in patients with cirrhosis, with those classified as Class-C (based on Pugh scores) exhibiting higher prolactin levels compared to Class-A patients. Vemanamanda S and Srinivasa SV[36] also had shown that the serum prolactin demonstrated a sensitivity of 82.61%, specificity of 73.91%, and diagnostic accuracy of 76.81% in predicting severe Child-Pugh scores. The positive correlation between serum prolactin levels and the Child-Pugh score in cirrhosis patients underscores the complex interplay between liver dysfunction and hormonal imbalances. The Child-Pugh score evaluates the severity of cirrhosis based on several parameters, including liver and kidney function. As cirrhosis progresses, leading to a higher Child-Pugh score, the liver's ability to metabolize and clear hormones, including prolactin, is impaired. This results in elevated serum prolactin levels. Additionally, cirrhosis affects the metabolism of dopamine, a hormone that inhibits prolactin secretion. With reduced dopamine clearance, prolactin levels can further increase.

Cirrhosis also leads to altered metabolism of estrogen, which can stimulate prolactin secretion, contributing to its elevated levels in advanced liver disease. Moreover, the physiological stress associated with severe liver dysfunction can activate the stress response, further increasing prolactin levels as part of the body's attempt to cope with the stress. Impaired renal function, often seen in advanced cirrhosis, exacerbates these effects by reducing the clearance of prolactin from the body. This multifaceted relationship highlights the significance of hormonal changes as cirrhosis progresses, offering insights into the broader systemic impact of liver disease and the importance of comprehensive management strategies.

Strengths and limitations

One of the strengths of the study is that our study is one among the fewer studies establishing the relationship between the prolactin levels and Child Pugh score, assessing the severity of the liver disease. However, our study is not without the limitations. Firstly, the sample size was relatively smaller and hence could lack sufficient power to comment on the statistical significance. However, it could be argued that a representative sample was calculated for our study and hence it could be generalizable to the population of the similar setting. Secondly, the association of

various etiologies on the prolactin level and the Child Pugh score could not be ascertained in our study, which could have added more value and understanding to the mechanisms for the above-mentioned observations. Last but not the least, temporal association could not be ascertained in our study due to the cross-sectional nature of the study design, which is inherent to all cross w4ectional studies. However, it could be safely concluded that the elevated serum prolactin levels in individuals with liver cirrhosis may act as an indicator of the disease's severity, showing a strong association with the Child Pugh score. Nonetheless, more comprehensive studies that are both multicentric and longitudinal, examining patient outcomes and complications, could strengthen the findings of our research.

Conclusion

In this study of 40 patients with cirrhosis of the liver, the mean age was 51.1 ± 11.1 years, with most patients belonging to the 46–60-year age group, and the study population was predominantly male. The mean serum albumin and INR values reflected significant hepatic dysfunction, while median total bilirubin levels were markedly elevated. Hepatic encephalopathy was present in 65% of patients, with varying severity across West Haven grades, and ascites was commonly observed, predominantly of moderate grade. The median Child–Pugh score was 10.5, with nearly half of the patients classified as Child–Pugh class C, indicating advanced liver disease. Serum prolactin levels were substantially elevated, with a median value of 55.2 ng/dl, and showed a very strong, statistically significant positive correlation with the Child–Pugh score. Higher Child–Pugh scores were observed among males, while age showed no significant association with disease severity. Prolactin levels were significantly higher in patients with hepatic encephalopathy and demonstrated a strong positive correlation with the grade of encephalopathy. Overall, elevated serum prolactin levels appear to be a potential indicator of disease severity in cirrhosis, closely associated with Child–Pugh scores, although larger multicentric and longitudinal studies are required to validate these findings and assess their prognostic implications.

There was no external financial aid
Ethical approval – Yes
Informed consent– yes
There is no conflict of interest for any author
The data is with the first author and can be reproduced only if a definite need arises

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