Adrenal Insufficiency in Chronic Liver Disease Patients and its Correlation with Child-Pugh Score

BM Singh Lamba*, Tarun Bansal**, Neera Sharma***, Rajat Kharbanda****

Abstract

Background: Liver is a storehouse for precursors of all adrenal hormones as well as cortisol binding globulin (CBG). Hence, adrenal dysfunction has been reported in patients with liver diseases. Adrenal Insufficiency (AI) may occur in compensated and decompensated cirrhosis without sepsis or in early and late stages after liver transplantation (LT). In cirrhotic patients, there is low HDL, favouring adrenal insufficiency in conditions of stress.

Methodology: This was a cross-sectional, observational study. 60 patients of chronic liver disease admitted to Dr Ram Manohar Lohia Hospital, Delhi and 30, as control were included in study. Patient’s blood was collected in fasting state at 8 AM to measure total baseline serum cortisol. ACTH stimulation was done by injecting 250 mcg ACTH intramuscularly. Another sample was collected after 60 min. Samples were centrifuged to collect serum. Total cortisol levels were measured in both the serum samples using chemiluminescence based method on fully automatic immunoassay analyser.

Results: Mean age in cases was 44.77 ± 7.93 years and mean age in controls was 42.77 ± 5.76 years. Adrenal insufficiency (AI) was present in 24 (40%) patients with chronic liver disease with a statistically significant P value of 0.029. Out of these, 13.33% (2 patients) with Child-Pugh class A, 40% (8 patients) with Child-Pugh class B, and 56% (14 patients) with Child-Pugh class C had adrenal insufficiency. None of the controls showed features of adrenal insufficiency. Patients with AI had a median serum bilirubin level of 2.6 mg/dl with IQR 1.45 - 15.30 compared to median 2.15 mg/dl with IQR 1.4 - 2.95 in those without AI. ROC curve showed that the Child Pugh score may be a good predictor for AI in liver cirrhosis patients.

Conclusion: Adrenal insufficiency (AI) forms an important part of the spectrum of chronic liver disease. Deterioration in functions of liver predicts presence of AI, and these patients should be evaluated for adrenal dysfunction periodically. Adrenal function worsens with progression of liver disease.

Keywords: Chronic liver disease, adrenal insufficiency.

Introduction

Liver diseases are common all over the world (prevalence being 4% - 17.5%) as well as in India and the prevalence of liver diseases is likely to increase in the future1. Liver diseases have been shown to be associated with various endocrine disturbances2. Liver is a storehouse for precursors of all adrenal hormones as well as cortisol binding globulin (CBG). Hence, adrenal dysfunction has been reported in patients with liver diseases3. Relative adrenal insufficiency (RAI) is the term given to inadequate production of cortisol with respect to the severity of adrenal insufficiency in cirrhosis4,5. More recently, another term, namely critical illness related corticosteroid insufficiency (CIRCI) defined as “inadequate cellular corticosteroid activity for the severity of the patient’s illness”6, has been used. It is proposed that adrenal insufficiency is common in cirrhotic patients too7,8. Furthermore, Adrenal Insufficiency (AI) may occur in compensated and decompensated cirrhosis without sepsis9 or post-liver transplantation (LT)10. Mechanisms of adrenal insufficiency in cirrhotic patients are not entirely known, but may include impaired synthesis in total cholesterol, HDL cholesterol and LDL cholesterol, as well as increased levels of proinflammatory cytokines and circulating endotoxins11. Approximately 80% of circulating cortisol is synthesized both at rest and during stress from plasma cholesterol. In cirrhotic patients there is low HDL, favouring adrenal insufficiency in conditions of stress. The effects of corticosteroid therapy in patients with decompensated cirrhosis are controversial12.

Material and methods

This was a cross-sectional, observational study. 60 patients of chronic liver disease admitted to Dr RML Hospital, Delhi and 30, as controls were included in study.

Inclusion criteria: Patients of age > 18 years, of either sex with evidence of chronic liver disease.

Exclusion criteria: Primary adrenal insufficiency,*

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disseminated tuberculosis, sepsis, pregnant females, patients receiving steroid therapy (last dose of steroid received in last 3 months), patients consuming alcohol in last three months.

Methodology
Patients with clinical features suggestive of chronic liver disease were diagnosed on the basis of deranged liver functions of > 6 months duration and/or evidence of chronic liver disease, portal hypertension on radiological imaging and/or evidence of oesophageal varices on upper gastrointestinal endoscopy. All patients underwent a detailed clinical examination at admission. Relevant history and physical examination including symptoms and signs of liver failure, hepatomegaly, splenomegaly and abdominal vein collaterals were recorded. Ascites was graded as none, easily controlled by medications or poorly controlled. Hepatic encephalopathy was graded from 0 to IV. Diagnosis of cirrhosis was based on clinical, biochemical and ultrasonographic findings. Haematological and biochemical workup included measurement of haemoglobin, total leucocyte count, platelet count, prothrombin time, and serum concentration of bilirubin (total and conjugated), protein, albumin, alanine aminotransferase and aspartate aminotransferase. For each patient a Child-Pugh score was calculated\(^1\). All patients underwent ultrasonography after overnight fast and the following details were recorded: Maximum vertical span of liver; nodularity of liver surface; spleen size (length of its longest axis); diameter of portal and splenic veins; presence of portal-systemic collaterals; and presence of ascites. All patients underwent upper gastrointestinal endoscopy for assessment of oesophageal and gastric varices. Patient’s blood was collected in fasting state at 8 AM to measure total baseline serum cortisol. ACTH stimulation was done by injecting 250 mcg ACTH intramuscularly. Another sample was collected after 60 min. Samples were centrifuged to collect serum. Total cortisol levels were measured in both the serum samples using chemiluminiscence based method on fully automatic Immunoassay analyser. Normal range of kit used was 123 - 623 nmol/l. Cut-off level to define adrenal insufficiency was baseline total serum cortisol < 276 nmol/l or peak cortisol levels < 500 nmol/l or delta cortisol (defined as difference between peak and basal cortisol) < 250 nmol/l\(^6\). Readings were compared with that of control group in which only total baseline serum cortisol levels were measured.

Statistical analysis: Categorical variables were presented in number and percentage (%) and continuous variables was presented as mean ± SD and median. Normality of data was tested by Kolmogorov-Smirnov test. If the normality was rejected, then non-parametric test was used.

Statistical tests were applied as follows:-
- Quantitative variables were compared using Independent T-test/Mann Whitney test (for nonparametric data) between two groups.
- Qualitative variables were compared using Chi-Square test /Fisher’s exact test.
- Logistic regression was performed to find out predictor of adrenal insufficiency taking adrenal insufficiency as dependent variable.
- ROC curve was used to find out cut-off value of Billirubin and Child Pugh score.

A p value of < 0.05 was considered statistically significant.
The data was entered in MS EXCEL spreadsheet and analysis was done using Statistical Package for Social Sciences (SPSS) version 21.0.

Results
A total of 60 patients with chronic liver disease were enrolled in this study and 30 age and sex-matched healthy controls were enrolled.

I. Patient characteristics: Cases had a mean age of 44.77 ± 7.93 years and controls had a mean age of 42.77 ± 5.76 years. There was no significant difference between age of cases and controls. Out of 60 patients in case group, 54 (90%) were males and 6 (10%) were females. Out of 30 persons in control group, 27 (90%) were males and 3 (10%) were females.

II. Relation between significant predictors and adrenal insufficiency.

Table I: Adrenal insufficiency: Distribution.

<table>
<thead>
<tr>
<th>Child-Pugh class</th>
<th>Total</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>B</td>
<td>C</td>
</tr>
<tr>
<td>Adrenal Absent</td>
<td>13</td>
<td>12</td>
</tr>
<tr>
<td>(86.67%)</td>
<td>(60%)</td>
<td>(44%)</td>
</tr>
<tr>
<td>Insufficiency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>(13.33%)</td>
<td>(40%)</td>
<td>(56%)</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>(100%)</td>
<td>(100%)</td>
<td>(100%)</td>
</tr>
</tbody>
</table>

Thus, this study shows that adrenal insufficiency was present in 24(40%) patients with chronic liver disease with a statistically significant P value of 0.029. Out of these, 13.33% (2 patients) with Child-Pugh class A, 40% (8 patients) with Child-Pugh class B, and 56% (14 patients) with Child-Pugh class C had adrenal insufficiency. None of the controls showed features of adrenal insufficiency.
Table II: Bilirubin among study groups.

<table>
<thead>
<tr>
<th></th>
<th>Cases</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median</td>
<td>2.25</td>
<td>0.7</td>
</tr>
<tr>
<td>Minimum</td>
<td>0.30 -</td>
<td>0.4 - 1.1</td>
</tr>
<tr>
<td>Quartile</td>
<td>1.400</td>
<td>0.6 -</td>
</tr>
<tr>
<td>Maximum</td>
<td>3.600</td>
<td>&lt; .0005</td>
</tr>
<tr>
<td>Range</td>
<td>27</td>
<td>-</td>
</tr>
</tbody>
</table>

Thus, bilirubin was significantly higher in cases with a median of 2.25 mg/dl as compared to controls with a median of 0.7 mg/dl.

Table III: Bilirubin, urea and, lipid profile and adrenal insufficiency.

<table>
<thead>
<tr>
<th>Adrenal Insufficiency</th>
<th>Absent</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Minimum</td>
<td>2.15</td>
<td>3.05</td>
</tr>
<tr>
<td>Inter- Quartile Range</td>
<td>4.5 -</td>
<td>5.1 -</td>
</tr>
<tr>
<td>Maximum</td>
<td>2.95</td>
<td>3.95</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>0.65</td>
<td>0.4 - 1.4</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.5 - 1</td>
<td>0.6 - 3.7</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>147.5</td>
<td>115.5</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>38</td>
<td>26.5</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>38</td>
<td>15 - 30</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>3.0 - 4.5</td>
<td></td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>2.98</td>
<td>2.5 - 0.58</td>
</tr>
</tbody>
</table>

Thus, on evaluating the serum bilirubin level, this study found that there was a significant correlation relation between AI and bilirubin. Patients with AI had a median serum bilirubin level of 2.6 mg/dl with IQR 1.45 - 15.30 compared to median 2.15 mg/dl with IQR 1.4 - 2.95 in those without AI with statistically significant P value of 0.047. There was a highly statistically significant correlation between serum creatinine level and AI, as the 24 patients diagnosed to have AI, had a median serum creatinine level of 2.70 mg/dl while the other 36 patients who did not have AI, had a median serum creatinine level of 0.65 mg/dl, with a P value of < 0.0005. Thus, this study reveals that patients with adrenal insufficiency had statistically significantly lower values of total cholesterol, HDL and LDL as compared to patients without adrenal insufficiency.

Table IV: AST and ALT among study groups.

<table>
<thead>
<tr>
<th></th>
<th>Case</th>
<th>Control</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT (U/L)</td>
<td>54.2 ± 26.41</td>
<td>24.73 ± 6.72</td>
<td>&lt; .0005</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>53.07 ± 26.42</td>
<td>24.43 ± 6.61</td>
<td>&lt; .0005</td>
</tr>
</tbody>
</table>

This study showed that AST was significantly higher in cases with a mean of 53.07 U/L as compared to controls with a mean of 24.43 U/L. Similarly, ALT was significantly higher in cases with a mean of 54.20 U/L as compared to controls with a mean of 24.73 U/L.

Table V: AST, ALT, albumin, urea, triglyceride and adrenal insufficiency.

<table>
<thead>
<tr>
<th>Adrenal Insufficiency</th>
<th>Absent</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Minimum</td>
<td>35.03</td>
<td>80.12</td>
</tr>
<tr>
<td>Intermediate Range</td>
<td>12.38</td>
<td>16.86</td>
</tr>
<tr>
<td>Maximum</td>
<td>29.0</td>
<td>36.31</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>30.81</td>
<td>46.5</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>9.03</td>
<td>13.27</td>
</tr>
<tr>
<td>Albumin (g/dl)</td>
<td>2.98</td>
<td>2.5</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>118.92</td>
<td>86.88</td>
</tr>
</tbody>
</table>

Thus, it was shown that AST was significantly higher in patients with adrenal insufficiency with a mean value of 80.12 ± 16.86 U/L as compared to patients without adrenal insufficiency with a mean of 35.03 ± 12.38 U/L. Similarly, ALT was significantly higher in patients with adrenal insufficiency with a mean value of 46.5±13.27 mg/dl, while the 36 patients who did not have AI, had serum urea level of 30.81 ± 9.03 mg/dl with a statistically significant P value: 0.005. On evaluating the relationship between serum albumin level and AI, it was found that the patients diagnosed to have AI, had significantly lower mean serum albumin level of 2.5 ± 0.58 g/dl while the patients who did not have AI, had mean serum albumin level of 2.98 ± 0.64 g/dl with a P value:0.005. This study showed that patients with adrenal insufficiency had statistically significantly lower values of serum triglyceride as compared to patients.
without adrenal insufficiency.

III. Logistic regression analysis: In statistics, logistic regression is used for prediction of the probability of occurrence of an event by fitting data to logic function, i.e., logistic curve. This study was done to find out the most significant predictor for AI from the obtained data as Child Pugh classification, serum SGOT/AST, serum bilirubin, serum creatinine and serum albumin.

Table VI: Univariate logistic regression with adrenal insufficiency as dependent variable.

<table>
<thead>
<tr>
<th>Variable</th>
<th>P value</th>
<th>Exp(B)</th>
<th>95% CI for EXP(B)</th>
<th>AIC of Reduced Model</th>
<th>BIC of Reduced Model</th>
<th>-2 Log Likelihood of Reduced Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin</td>
<td>.023</td>
<td>1.331</td>
<td>1.041 1.702</td>
<td>69.474</td>
<td>71.568</td>
<td>67.474</td>
</tr>
<tr>
<td>Albumin</td>
<td>.008</td>
<td>.294</td>
<td>.119 .725</td>
<td>51.710</td>
<td>53.804</td>
<td>49.710</td>
</tr>
<tr>
<td>Child-Pugh score</td>
<td>.010</td>
<td>1.335</td>
<td>1.071 1.664</td>
<td>45.077</td>
<td>47.172</td>
<td>43.077</td>
</tr>
<tr>
<td>ALT</td>
<td>.0005</td>
<td>1.145</td>
<td>1.075 1.221</td>
<td>76.405</td>
<td>78.500</td>
<td>74.405</td>
</tr>
<tr>
<td>Creatinine</td>
<td>.001</td>
<td>38.277</td>
<td>4.797 305.44</td>
<td>68.719</td>
<td>70.814</td>
<td>66.719</td>
</tr>
</tbody>
</table>

All these data were analysed by the logistic regression analysis and all the above variables were found to be significant predictor for AI with a P value < 0.05.

IV. Receiver operating characteristic (ROC) curve for adrenal insufficiency: ROC curve is a graphical plot of sensitivity by plotting the fraction of true positives out of the positives (TPR = true positive rates) versus the fraction of false positives out of the negatives (FNR = false negative rate), in other words sensitivity versus specificity.

Table VII: ROC curve for adrenal insufficiency.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Area under the ROC curve</th>
<th>95% Confidence Interval</th>
<th>Youden Index</th>
<th>Sensitivity Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin</td>
<td>0.652199</td>
<td>0.518227 to 0.770590</td>
<td>0.559</td>
<td>0.4583</td>
</tr>
<tr>
<td>CPS</td>
<td>0.713542</td>
<td>0.582242 to 0.822894</td>
<td>0.0014</td>
<td>&gt; 8</td>
</tr>
</tbody>
</table>

ROC curve showed that the Child-Pugh score may be a good predictor for AI in liver cirrhosis patients. The area under the Child-Pugh score curve was 0.714 and a Child-Pugh cut-off score of 8 had a sensitivity of 87.50% and specificity of 61.11%. Child-Pugh score could be a good predictor with a considerable sensitivity and specificity as, with high score values, the specificity in detecting AI increased.

Discussion

60 patients of chronic liver disease were enrolled in this study with a mean age of presentation 44.77 ± 7.93 years (range 21 - 70 years). 90% patients (54 patients) were male and 10% patients (6 patients) were female. Similarly, 30 controls were enrolled with a mean age of presentation 42.77 ± 5.76 years. 90% controls (27 controls) were male and 10% controls (3 controls) were female. This study revealed, firstly, high percentage of adrenal insufficiency in chronic liver disease patients. Secondly, there was increase in percentage of subjects with adrenal dysfunction with progression of liver disease as assessed by Child-Pugh scoring. And lastly, various parameters related to liver dysfunction were also predictors of AI.
In this study, 40% patients (24 patients) with CLD had adrenal insufficiency. Out of these, 13.33% (2 patients) with Child-Pugh class A, 40% (8 patients) with Child-Pugh class B, and 56% (14 patients) with Child-Pugh class C had adrenal insufficiency. Various authors have reported percentage of AI ranging from 33 - 68% in patients with CLD. This is due to varied aetiology, severity and different methods used for the diagnosis of AI in different studies. Galbois et al15 and Tan et al16, noted AI in 33% and 39% of subjects with stable cirrhosis with the same method and criteria as in the present study. Fernandez J7 prospectively evaluated the effects of steroids on shock resolution and hospital survival in a series of 25 consecutive patients with cirrhosis and septic shock. It was found that adrenal dysfunction was frequent in patients with advanced cirrhosis (Child C: 76% versus Child B: 25%, P = 0.08) and resolution of septic shock (96% versus 58%, P = 0.001), survival in the intensive care unit (68% vs. 38%, P = 0.03), and hospital survival (64% versus 32%, P = 0.003) were significantly higher in those who received corticosteroid. Regarding serum creatinine level, this study found that patients with AI had a median serum creatinine level of 2.7 mg/dl with inter quartile range (IQR) 1.65 - 3.10, compared to median of 0.65 mg/dl with IQR 0.5 - 1.0 in patients without AI, with a statistically significant P value of < 0.0005. These findings are in agreement with Tsai et al6, who found that patients with AI had mean serum creatinine level of 3.2 ± 2.7 mg/dl and in patients without AI the mean was 1.9 ± 1.6 mg/dl which showed statistically significant P value: 0.004. On evaluating the serum bilirubin level, this study found that there was a significant correlation between AI and bilirubin. Patients with AI had a median serum bilirubin level of 2.6 mg/dl with IQR 1.45 - 15.30 compared to median 2.15 mg/dl with IQR 1.4 - 2.95 in those without AI with statistically significant P value of 0.047. This is in agreement with Tsai et al6, who found a significant relation between AI and serum bilirubin P < 0.001. ROC curve analysis was done and showed that serum bilirubin may be a good predictor for AI in liver cirrhosis patients, as the area under the curve of bilirubin was 0.652. With a serum bilirubin cut-off level of 5.10 mg/dl a sensitivity of 45.83% and specificity of 100% were shown.

This is in agreement with Tsai et al6, who reported that serum bilirubin was an independent factor in predicting adrenal insufficiency in critically ill patients with cirrhosis and severe sepsis. The significantly low serum HDL levels (median = 38 mg/dl), serum LDL (median = 72.5 mg/dl), total cholesterol (median = 147.5 mg/dl) and triglyceride (mean = 118.92 mg/dl) in adrenal insufficiency observed in this study further support the notion that liver disease may lead to impaired cortisol synthesis. In a study by Paul E Marik6, they found the HDL level at the time of adrenal testing was the only variable predictive of adrenal insufficiency (p < 0.0001). This can either be explained by direct decrease in substrate supply or indirect effect of cytokines. The adrenal gland does not store cortisol, increased secretion arises due to increased synthesis under the control of adrenocorticotropic. Cholesterol is the principal precursor for steroid biosynthesis in steroidogenic tissue. At rest and during stress, about 80% of circulating cortisol is derived from plasma cholesterol, the remaining 20% being synthesised in situ from acetate and other precursors. Experimental studies suggest that HDL is the preferred lipoprotein source of steroidogenic substrate in the adrenal gland. Cicognani et al18 demonstrated a striking decrease in the level of serum HDL in patients with cirrhosis that was related to the severity of disease (Child class).

Conclusion

Adrenal insufficiency (AI) forms an important part of the spectrum of chronic liver disease. Deterioration of functions of liver disease predicts presence of AI, and these patients should be evaluated for adrenal dysfunction periodically. Adrenal function worsens with progression of liver disease.

Steroid replacement in CLD patients at time of stress and critical illness may be beneficial as supported by studies of Marik et al7 and Fernandez et al7. They showed that steroid replacement in patients of CLD with adrenal insufficiency leads to resolution of septic shock, decreased doses of vasopressor, increased survival in intensive care and increased hospital survival. Also, presence of AI may predict survival of CLD patients.

Larger studies are needed to establish role of steroids in improving outcome of liver disease patients, also standardisation of criteria for diagnosis of AI in setting of liver disease needs to be established.

References


ACKNOWLEDGEMENT

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