

**Research Article****Serum Iron and TIBC Parameters in Chronic Liver Disease****Dr Shivam Khare<sup>1</sup>, Dr Vijay Kumar Garg<sup>1</sup>, Dr Omprakash jatav<sup>2</sup>**<sup>1</sup>Senior resident, Department Of Medicine, Gajra Raja Medical College Gwalior, India<sup>2</sup>Professor and head, Department Of Medicine, Gajra Raja Medical College Gwalior, India**\*Corresponding author**

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**Abstract:** To assess and compare the significance of serum iron and TIBC in patients with chronic liver disease and also comparison between alcoholic and non alcoholic CLD. The study was conducted J.A.Group of Hospital during the period of June 2013 to November 2014 and about 100 patients of chronic liver disease were selected in random from patients coming to department of general medicine OPD and ward patients. The study of 100 patients with chronic liver diseases, which were examined for frequency and correlation between elevated liver enzymes, haematological parameters, ultrasound diagnosed features of chronic liver disease and indices of iron. Mean TIBC in Child Pugh class C was  $276.03 \pm 33.48$  (p value 0.00001). TIBC was higher significantly in non alcoholic CLD patients  $335.47 \pm 35.91$  (p value  $<0.00001$ ). Mean serum iron in Child Pugh class C was  $129.03 \pm 51.53$  which was significantly higher than rest of group. Child Pugh class A has serum iron mean  $82.33 \pm 26.59$ . Serum iron was significantly higher in alcoholic CLD patients  $143.73 \pm 16.21$  (p value  $<0.0001$ ). Serum iron increased and TIBC decreased with increasing disease severity according to Child Pugh score. So there is good correlation of severity of CLD with serum iron and TIBC. In our study these changes are more in alcoholic group.

**Keywords:** child Pugh score, chronic liver disease, iron, TIBC (total iron binding capacity).

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**INTRODUCTION**

Chronic liver disease in the clinical context is a disease process of the liver that involves progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis [1]. Typical presenting symptom of liver disease include fatigue, jaundice, and right upper quadrant pain, itching, nausea, poor appetite, edema, abdominal distension, intestinal bleeding. Chronic liver disease frequently associated with haematological abnormalities. Anaemia of diverse etiology occurs in about 75% of patients of chronic liver disease [2]. The liver is an important organ in iron homeostasis. Besides its involvement in iron storage, the liver also produces transferrin and hepcidin, an iron carrier protein in plasma and a hormone regulating iron metabolism, respectively. Another aspect of the relationship between iron and the liver is that this organ is one of the main targets in hemochromatosis. Serum iron (SI), total iron binding capacity (TIBC) and ferritin levels are the principal tests used in the evaluation of iron burden. Another frequently used parameter, transferrin saturation (TS), is calculated by dividing SI level by TIBC, and it shows the percent saturation of transferrin. Testing and understanding serum iron parameters in liver disorders are important for different reasons: 1) Serum TS and ferritin levels are used for the screening of hereditary hemochromatosis. 2) It has been proposed that iron might be important for progression

of liver fibrosis in viral hepatitis, and serum iron parameters, especially ferritin level, might reflect hepatic iron accumulation. Anemia is very frequent in cirrhotic patients for many different reasons including iron deficiency. Identification of iron deficiency in these patients is especially important, because it is an easily correctable cause of anemia. Total iron binding capacity (TIBC) level (i.e., transferrin activity) may change in hepatic disorders as transferrin is produced in the liver. Ferritin is increased in many patients with acute and chronic liver diseases (CLDs). Therefore, serum iron parameters may not truly reflect iron homeostasis in hepatic disorders. It has been proposed that serum iron parameters were unreliable in CLD, and that systemic iron overload should be confirmed histologically in these patients. However, the effect of the severity of hepatic compromise on the test results has not been clearly defined in any study. Similarly, differences between liver disease- and iron overload-related iron parameter changes have not been clarified. Previous studies related to iron homeostasis in CLDs might be confounded by difficulties in the diagnosis of hereditary hemochromatosis. Currently, this disease can be easily defined by genetic testing.

**Alcohol's Effects on Iron Metabolism [4]**

In addition to interfering with the proper absorption of iron into the haemoglobin molecules of

red blood cells (RBC's), alcohol use can lead to either iron deficiency or excessively high levels of iron in the body. Because iron is essential to RBC functioning, iron deficiency, which is commonly caused by excessive blood loss, can result in anemia. In many alcoholic patients, blood loss and subsequent iron deficiency are caused by gastrointestinal bleeding. Iron deficiency in alcoholics often is difficult to diagnose, however, because it may be masked by symptoms of other nutritional deficiencies (e.g., folic acid deficiency) or by coexisting liver disease and other alcohol-related inflammatory conditions. Alcohol abuse can increase iron levels in the body, iron absorption from the food in the gastrointestinal tract maybe elevated in alcoholics. Iron levels also can rise from excessive ingestion of iron-containing alcoholic beverages, such as red wine.

## MATERIALS AND METHODS

The present study was conducted in J A group of hospital, Gwalior, M.P. (G.R. Medical College, Gwalior, M.P.). Study was included 100 patients of CLD presenting in indoor and outdoor department. The present study is conducted over a period between June 2013 to November 2014 on the patients of chronic liver disease patients. In the present study two groups has been created and studied. Group I comprises of patients of Alcoholic chronic liver disease & Group II comprises of patients of Non-Alcoholic chronic liver disease in the present study.

### Inclusion criteria

- All confirm cases of chronic liver disease by clinical, biochemical and radiological evaluation..
- Study include both sex.
- Age >15 years.

### Exclusion criteria

- Patient on drugs which cause in defect parameters such as glucocorticoid, synthetic estrogens, aspirin, tamoxifen, methotrexate, OCP.
- Malignancy
- Pregnancy
- Previous history of haematological and coagulation disorder other than CLD.

### Statistical Methods

Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups Inter group analysis and chi square test has been used to analyze the data having ordinal variables. Significant figures were analyzed, + Suggestive significance(P:<0.05)

### Statistical software

The Statistical software namely SPSS 22.0, for the analysis of the data and Microsoft word and Excel were used to generate graphs, tables. A p value of <0.05 was considered as significant.

## Data Collection

All the chronic liver disease patients attending JAH Groups of Hospital, Gwalior, MP. were screened for eligibility. Informed consent was taken from the eligible patients and enrolled in the present study. The patients were interviewed and underwent thorough physical examination.

## History and examination

A detailed history was elicited from all patients with emphasis on symptomatology and history of presenting & past illness; personal & family history; drug & addiction history is taken. Detailed clinical evaluation including history including questioning about risk factors for chronic liver disease ,history of hepatitis, alcohol consumption, diabetes mellitus, use of illicit drugs (by injection or inhalation), transfusions, family history of liver disease, travel, and the presence of autoimmune diseases (including inflammatory bowel disease, rheumatoid arthritis and thyroid disease). The review of systems including questioning related to fatigue, easy bruisability, lower extremity edema, fever, weight loss, pruritus, increasing abdominal girth, and confusion or sleep disturbance (possibly indicating encephalopathy). Clinical signs including spider naevi, gynaecomastia, anemia, low grade fever, white opaque nails, clubbing of nails, foetor hepaticus, jaundice, ascitis, encephalopathy, Prominent veins over abdomen, caput medusa, hepatomegaly, splenomegaly. Patients are classified on disease severity on basis of CHILD PUGH SCORE.

## Investigations

All patients were subjected to the following investigation at the time of inclusion into the study. Routine hemogram.(Hb, TLC, DLC, Platelet),peripheral blood smear, Liver function test (Serum Bilirubin, SGOT, SGPT, SAP, S. protein, PT),Serum iron ,TIBC,U pper GI endoscopy, Chest xray, Urine r/m, Ascitic r/m, Fasting and post prandial blood sugar. Electrolyte, Ultrasonography of abdomen, Blood urea and serum creatinine.

## RESULTS

In present study out of 100 cases, 45 cases of alcoholic chronic liver disease, in alcoholic group all were Male, while in 55 cases of Non alcoholic liver disease both male and female were included whose numbers were almost equal (28 females, 27 males). In overall CLD cases, Males are predominant with 72%. In present study mean age of all patients were 47.56±13.77 years. Mean age of males was 48.708±12.36 years and Mean age of female was 44.607±16.74 years. Mean age of alcoholic and non alcoholic were 47.07±10.92 years and 47.96±15.81 years respectively (Table-1).

Majority of patients belonged to Child Pugh score B in both Alcoholic and Non Alcoholic CLD with

51.11% and 51.85% respectively, followed by child pugh score C and least in Child Pugh score A (Table-2).

TIBC was higher significantly in non alcoholic CLD patients 335.47±35.91 (p value <0.00001). Serum iron was significantly higher in alcoholic CLD patients 143.73±16.21 (p value <0.0001) (Table-3).

All low TIBC patients belong to Child Pugh class C. Majority of patients in all class of Child Pugh

score have normal TIBC. As Child Pugh Score increases, TIBC decreases. Class C having mean TIBC 276.03 ± 33.48 (p value 0.00001) (Table-3).

Majority of cases had normal serum iron. Mean serum iron in Child Pugh class C was 129.03 ± 51.53 which was significantly higher than rest of group. Child Pugh class A has serum iron mean 82.33 ± 26.59 which was significantly lower than other groups. As CPS class upgrade serum iron increases (Table-4).

**Table-1:-Distribution of cases according to Child pugh score in Alcoholic CLD and Non-Alcoholic CLD**

CPS	Alcoholic CLD		Non Alcoholic CLD		
	No.	%	No.	%	
A	6	13.33%	12	22.22%	P = 0.27
B	23	51.11%	28	51.85%	P = 1.0
C	16	35.56%	14	25.93%	P = 0.27
Total	45	100.00%	55	100.00%	

**Table-2: Comparison of TIBC and serum iron in alcoholic and non alcoholic CLD**

	Alcoholic CLD		Non Alcoholic CLD		Significance
	Mean	Std dev	Mean	Std dev	
TIBC	269.15	26.27	335.47	35.91	P<0.00001
Serum Iron	143.73	16.21	76.64	16.21	P < 0.0001

**Table-3: Comparison of TIBC according to Child Pugh Score in all cases of CLD**

TIBC (µg/dl)	Child Pugh Score			Total
	A	B	C	
≤250	0	0	10	10
251-350	10	43	20	73
>350	8	9	0	17
Total	18	52	30	100
Mean	339.11	311.11	276.03	305.63
S D	39.46	44.91	33.48	46.05
Significance	P=0.55	P= 0.21	P= 0.00001	---

**Table-4: Distribution of serum iron according to Child Pugh Score in all CLD cases**

Serum Iron (µg/dl)	Child Pugh Score			Total
	A	B	C	
<50	2	1	0	3
51-150	16	38	18	72
>150	0	13	12	25
Total	18	52	30	100
Mean	82.33	102.50	129.03	106.83
Std dev	26.59	40.43	51.53	44.96
Significance	A Vs B&C P=0.007		C Vs A 0.004	C Vs A&B P=0.04

**DISCUSSION**

In present study out of 100 cases, 45 cases of alcoholic chronic liver disease, in alcoholic group all were Male, while in 55 cases of Non alcoholic liver disease both male and female were included whose numbers were almost equal (28 females, 27 males). In

overall CLD cases, Males are predominant with 72%. In present study mean age of all patients were 47.56±13.77 years. Mean age of males was 48.708±12.36 years and Mean age of female was 44.607±16.74 years. Mean age of alcoholic and non alcoholic were 47.07±10.92 years and 47.96±15.81 years respectively. In a study done by

M.Radicheva et al. [5], total 160 cases of chronic liver disease was considered. Out of which 104 males and 56 females was studied with mean age overall was  $49.90 \pm 12.2$  years. This is almost comparable with our study.

Iron might be important for progression of liver fibrosis in CLD. Serum iron reflect hepatic iron

accumulation and disease severity. TIBC level may be change in chronic liver disease. TIBC level depend on transferrin which is produced in liver. Due to decrease synthesis of transferrin in CLD, TIBC progressively decreased with disease severity.

	Our Study		Naciye semnur et.al [3]	
	Serum Iron	TIBC	Serum Iron	TIBC
Child Pugh A	82.33±26.59	339.11±39.46	72±26	291±65
Child Pugh B	102.03±40.43	311.11±44.91	77±43	252±75
Child Pugh C	129.03±51.53	276.03±33.48	89±34	193±75

In present study serum iron and TIBC results clearly indicates the cirrhotic stage of CLD. Cirrhotic patients of any Child-Pugh class had deviations from normal results. Serum iron and TIBC, according to Child-Pugh class were comparable to previous study done by Naciye semnur et al. [3]. It was observed that serum iron value was increased with Child-Pugh class from A to C and TIBC was decreased with Child-Pugh class from A to C. However absolute value lies in normal range in both studies except TIBC of Child pugh score C in Naciye et al. [3] study was lower than normal value with absolute value  $193 \pm 75$ .

	Our study	Krzysztof Jurczyk et al. [6]
Serum Iron	269.15±26.27	268.3
TIBC	143±16.21	159.5

In present study, in alcoholic CLD mean of serum iron and TIBC were  $143 \pm 16.21$  and  $269.15 \pm 26.27$  which were comparable with previous study Krzysztof Jurczyk et al. [6] with mean of serum iron and TIBC 159.5 and 268.3 respectively. In present study, the serum iron was more in alcoholic CLD ( $143 \pm 16.21$ ) then non alcoholic CLD ( $76.64 \pm 16.21$ ) which was comparable with previous study Krzysztof Jurczyk et al. [6]. In present study, the TIBC was more in alcoholic CLD ( $269.15 \pm 26.27$ ) than non alcoholic CLD ( $335.47 \pm 35.91$ ) which was comparable with previous study Krzysztof Jurczyk et al. [6].

**CONCLUSION**

Most of the CLD patients had normal serum iron and TIBC but serum iron increased and TIBC decreased with increasing disease severity according to Child Pugh score, this may be probably because of increased iron store leads to raised serum iron value and decreased synthetic capacity of liver which leads to decrease transferrin which in turns to lower TIBC. So there is good corelation of severity of CLD with serum iron and TIBC. In our study degree of deviation of serum iron (increased) and TIBC (decreased) were more in alcoholic etiology than non-alcoholic chronic liver disease. So alcohol consumption and iron levels play a key role in the progression of liver disease and

pathogenecity. Regular monitoring of iron and TIBC especially in alcoholic patients is necessary for better patient management and to minimize the morbidity and mortality related to liver injury.

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