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Mri Quantification Of Liver Iron Concentration In Chronic Liver Disease

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ABSTRACT
Introduction: *Chronic liver disease leads to decreased production of hepcidin causing increased iron deposition in the liver. This can lead to the progression of the disease. Liver biopsy is considered as the gold standard for diagnosis of liver iron concentration. But invasive nature, sampling error, inter and intra-observer variability, incidence of pain and need for hospitalization are the major drawbacks of this procedure. Hence other non invasive modalities for diagnosis and liver iron concentration are needed. MRI signal intensity reduction technique and T2* relaxation technique are the common non invasive methods studied widely for this purpose.*
Aims and objectives: *1) To assess the feasibility and evaluate the performance of various 1.5 Tesla MR imaging techniques in the detection and quantification of hepatic iron in patients with chronic liver disease. 2) To understand the scope of liver diseases with respect to iron deposition and to define a correlation between liver disease and liver iron concentration.*
Methodology: *It is a hospital based cross sectional study conducted among 30 patients attending Department of Radio-diagnosis for investigation of chronic liver disease. Socio-demographic characteristics were taken, followed by laboratory reports for Hb, TC, Serum ferritin, TIBC and MRI liver iron concentration quantification in liver.*
Results: *Mean age of participants was 55.1 $\pm$ 14.2 yrs. 46.7% of cases had increased serum ferritin with the mean serum ferritin of 393 $\pm$ 431 µg/dL and the median was 329 µg/dL. Similarly, 63% of cases had reduced TIBC with mean and median of 242 $\pm$ 43 µg/dL and 226 µg/dL respectively. The mean MRI LIC was 73.6 $\pm$ 70.1µmol/g. There was positive, very strong and significant correlation between MR Liver iron concentration and serum ferritin ($r = 0.840$, $p=0.000$) and negative, medium and significant correlation between MR Liver iron concentration and Total iron binding capacity ($r = -0.421$, $p=0.021$).*
Conclusion: *The current study concludes that MRI can be used in quantification of liver iron concentration as a non invasive technique in chronic liver diseases wherever feasible. In addition, fat quantification of liver can be used as a predictor for increased serum ferritin according to the current study.*
Keywords: *Chronic liver disease, quantification of liver iron concentration, non invasive technique, MRI, Signal intensity ratio*

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I. INTRODUCTION

Chronic liver diseases are set of diseases caused by chronic inflammation or insult to liver for more than 6 months, leading to reduced hepatic function[1]. It can progress from inflammation to fibrosis and further into Cirrhosis and end stage liver disease. 1.5 billion cases of Chronic disease is estimated worldwide with NAFLD (59%), HBV (29%), HCV (9%) and Alcoholic liver disease (2%) contributing for most of the cases. In addition, increase in obesity and alcohol consumption is expected to increase the burden of NAFLD and ALD in the future. It can lead to significant complications like portal hypertension, encephalopathy, hepatocellular insufficiency and hepatocellular carcinoma and hence is the 11th leading cause of death, accounting for 2.2% of death worldwide with an estimated 1.32 million death in 2017. Hence it is a disease of public health importance.

Iron is one of the essential micronutrients required primarily for oxygenation of blood. It is maintained primarily by liver, intestine, erythroblast and macrophages. Excess iron is usually stored in liver. It also produces ferritin, hepcidin and transferrin which are major proteins in iron metabolism. Hence in 10-30% of cases of chronic liver disease, there is evidence of some degree of iron overload. Excess iron in hepatic cell can lead to hepatic oxidative stress, immune cells activation and ballooning injury to the cells. In addition, studies have shown that iron overload is associated with progression of disease. Hence early detection, quantification and management of cases are of utmost importance in chronic liver disease.

Quantification of iron overload can be done by many techniques. Percutaneous liver biopsy with prussian blue staining followed by semi-quantitative scoring using Rowe et.al. was considered as gold standard for quantifying iron overload. It helps in direct visualization of hepato-pathology and in distinguishing different pathological conditions and in establishing severity. However, invasive nature, cost, sampling error, inter and intra-observer variability, bleeding and increased hospitalization are few of the disadvantages of the method. Hence there is a need for alternative for liver biopsy.

Magnetic resonance imaging (MRI) is the non invasive radiological technique studied and used worldwide for quantification of liver iron. Due to its paramagnetic property, iron affects MRI in multiple ways. Among the majority of techniques used in liver quantification; signal intensity ratio (SIR) techniques based on T_2-weighted or T_2^*-weighted, imaging, quantitative relaxometry (largely R2- and R2*-based), and MR susceptometry are the commonly used techniques. Signal intensity ratio (SIR) is the widely used technique which compares the signal intensity between liver and paraspinal muscles.

Aetiology of chronic liver disease

Above mentioned in epidemiology section are the common causes of chronic liver disease worldwide. Following are few salient features about the same.

1. Non alcoholic fatty liver disease

NAFLD is a disease which is increasing worldwide due to increased fat deposition in the liver due to either increased intake of food/calorie or/and sedentary lifestyle. Increased macrovesicular fat deposition in the hepatocyte (>5% of hepatocytes) leads to lipotoxicity; which leads to cascade of reactions causing hepatic injury. It has a pooled global prevalence of 25.24% with the prevalence of 9-32% in India. It is usually a diagnosis of exclusion. Few cases of NAFLD progress to marked hepatic injury with ballooning, fibrosis and inflammation which is termed as Non alcoholic steatohepatitis (NASH).

2. Alcoholic liver disease

ALD is caused by increased fat deposition in the liver secondary to excessive alcohol consumption. It is also due to excessive fat deposition in liver which can progress from steatosis to fibrosis to cirrhosis. Prolonged abstinence is the main therapy for this condition. However, studies have shown that in 20% cases, continuous drinking leads to eventual cirrhosis of liver and among them, 2/3rd of the patients develop decompensated liver disease and 15% develop hepatocellular carcinoma.

3. Hepatitis B and C

Hepatitis B is one of the common infectious diseases worldwide. WHO estimates that around 296 million population were living with chronic HBV in 2019 with 1.5 million new infections every year and 82,000 deaths. Similarly 71 million population are chronically infected with HCV i.e. 1% of world population with an estimated 1.75 million new cases in 2015.

4. Genetic causes

Alpha 1 antitrypsin deficiency is the most common genetic cause of chronic liver disease in children. It is a autosomal recessive disorder with co-dominant expression leading to disturbance in protein folding. This leads to increased accumulation of toxic insoluble ATZ protein aggregates in the endoplasmic reticulocyte of hepatocytes.

Hereditary haemochromatosis is a genetic disorder with decreased production of hepcidin. This leads to increased intestinal absorption of iron, leading to inherited iron overload. It is classified into 4 types based on the protein affected. Common mutations seen are in the C282Y or HFE gene.

Wilson's disease is a disease of impaired copper metabolism, leading to excess accumulation mainly in liver. It is a genetic disease caused by defective ATP7B protein product and can lead to CLD and fulminant liver disease.

5. Autoimmune causes

Autoimmune hepatitis is one of the common causes of autoimmune disease affecting hepatocytes where as primary biliary cirrhosis and primary sclerosing cholangitis, primarily affect biliary epithelial cells. While high

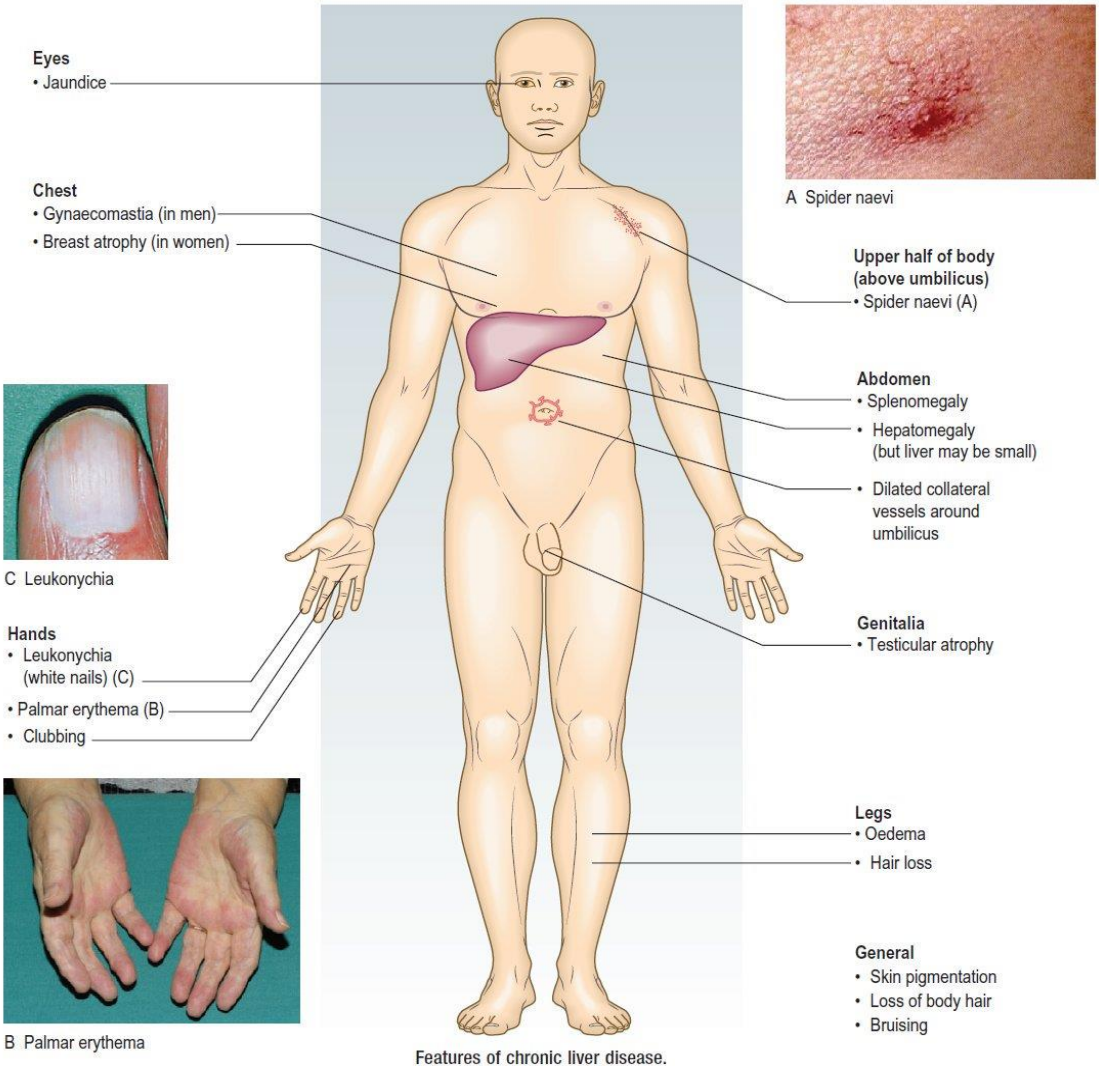
level of serum anti-microbial antibodies (AMA) are seen in PBC, ANA and/or anti-smooth muscle autoantibodies (SMA) is seen in Autoimmune hepatitis.

6. Other causes

Hepatotoxic drugs, vascular disease like Budd Chiari syndrome, idiopathic causes are the other main causes of CLD.

Clinical manifestations in Chronic liver disease

The clinical manifestations of CLD vary based on cause or stage or complications of the disease. At the early stage, the signs and symptoms of CLD may be vague. It may present as fatigue, anorexia and weight loss. Additional symptoms may be seen based on cause of CLD. If the patient develops hepatic insufficiency, he may present with jaundice, pruritis and in advanced stage may show features of hepatic encephalopathy. Obstruction of portal blood flow due to cirrhosis and increased vascular tone in hepatic micro-circulation can lead to portal hypertension. Portal hypertension may lead to formation of collateral vessels and arterial vasodilation, which progresses to increased portal circulation causing an increased hyperdynamic circulation resulting in ascites and/or esophageal varices. Reduced destruction of estrogen causes symptoms related to hyperestrogenism i.e. palmar erythema, spider angioma, gynaecomastia and testicular atrophy. Other major complications of CLD include coagulopathy and hepatorenal syndrome



Features of chronic liver disease.

Severity Indicators in Chronic liver disease

Cirrhosis in chronic liver disease can further be classified as compensated stage and decompensated stage. Decompensated stage increased the mortality of patients and is usually indicated by development of ascites, hepatic encephalopathy, and/or gastroesophageal variceal hemorrhage.

There are other indicators to detect severity of chronic liver disease. Child-Pugh scoring system (also known as the Child-Pugh-Turcotte score) and Model for end stage liver disease (MELD) are the commonly used severity scoring system. Child Pugh scoring system was initially designed by Child and Turcotte in 1964.

Factor	1 point	2 points	3 points
Total bilirubin (μmol/L)	<34	34-50	>50
Serum albumin (g/L)	>35	28-35	<28
PT INR	<1.7	1.71-2.30	>2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

	Class A	Class B	Class C
Total points	5-6	7-9	10-15
1-year survival	100%	80%	45%

Child Child-Pugh-Turcotte score[60]

The score uses 5 parameters namely total Bilirubin, serum albumin, PT INR, Ascites and hepatic encephalopathy and which parameter is given a score from 1-3 based on progression. Following this, based on the cumulative score, the patients are classified as Class A (5-6), B (7-9) and C (10-15). It helps to predict the 1 year survival of the patients. This scoring system has been validated in previous studies.
Similarly MELD score is used to valuate hepatic function reserve in cirrhotic patients.

Model for End-Stage Liver Disease (MELD) Score

MELD = 3.78 x $\log_e$ serum bilirubin (mg/dL) +
11.20 x $\log_e$ INR +
9.57 x $\log_e$ serum creatinine (mg/dL) +
6.43 (constant for liver disease etiology)

NOTES:

- If the patient has been dialyzed twice within the last 7 days, then the value for serum creatinine used should be 4.0
- Any value less than one is given a value of 1 (i.e. if bilirubin is 0.8, a value of 1.0 is used) to prevent the occurrence of scores below 0 (the natural logarithm of 1 is 0, and any value below 1 would yield a negative result)

Role of liver in iron metabolism

Hepcidin is the major systemic iron regulator. Based on the intracellular and extracellular requirements of iron, hepcidin action is regulated by bone morphogenic protein receptors and their ligands and sensors. Hepcidin primarily acts by degradation of ferroportin and hence regulating intestinal iron absorption, plasma iron concentrations, and tissue iron distribution. Haemochromatosis is a condition where there is a genetic mutation of HFE gene leading to reduction in the concentration of the iron regulatory hormone hepcidin, or a reduction in hepcidin-ferroportin binding. Due to reduction of hepcidin, there is iron overload in this condition. The role of ferritin and transferrin has been discussed before.

Chronic liver disease and iron overload

The major cause of iron overload in CLD is due to decreased production of hepcidin. Various additional mechanisms add to this depending on the cause of CLD. Studies have shown that iron overload increases with severity of CLD and vice versa. In addition, studies have shown that among Hepatitis C patients, iron overload resistance to interferon/ribavarin combination therapy. In additions, studies have shown that liver iron is a predictor of death in alcoholic cirrhosis and also a risk factor for HCC. The major effect of iron on hepatocytes is through superoxide radical and reactive hydroxyl radical generated by iron via Fenton or Harber-Weiss reactions. These free radicals can damage DNA, protein, nucleic acids and lipids. Usually this reaction is neutralized by

anti-oxidant reaction but in excess iron accumulation, this action becomes insufficient leading to damage to hepatocytes

II. AIM AND OBJECTIVES

1) To assess the feasibility and evaluate the performance of various 1.5 Tesla MR imaging techniques in the detection and quantification of hepatic iron in patients with chronic liver disease.
2) To understand the scope of liver diseases with respect to iron deposition and to define a correlation between liver disease and liver iron concentration.
3) To propose a classification of iron overload severity with respect to liver disease severity.

III. MATERIALS AND METHODS

After getting permission from Institutional ethical committee and other appropriate authorities, patients with symptoms of chronic liver disease attending the study area for MRI who satisfied the inclusion and exclusion criteria were approached for the study. After informing the participants about the study using a Patient information sheet, informed consent to participate in the study was taken. Using a preformed semi structured questionnaire, socio-demographic characteristic characteristics of the participants were assessed. Patients were examined on a 1.5T MR scanner with a body coil. It was performed by obtaining single gradient echo sequence acquisition with 8-10 TEs in multiples of 1.2 ms, alternating in phase and opposed-phase echoes and 20° flip angle, and the time to repetition (TR) was constant at 120 ms. We measured liver signal intensity in three different locations. The ROIs was drawn with 2-3cm^2 as large as possible, avoiding large vessels or lesions. The first slice was positioned just below the diaphragm through the right lobe of liver, and the next two slices was spaced 8 cm from the first one. To calculate muscle signal intensity, we performed the same procedure by placing two regions of interest on right and left paraspinous muscles, on the same transverse sections as those used to measure liver signal intensity, and avoided inclusion of intermuscular fat. We calculated the L/M ratio (signal intensity ratio/SIR) by dividing mean liver signal intensity by mean muscle signal intensity. The Liver Iron Concentration (LIC) was obtained from SIR using the DICOM Software MRQuantif.

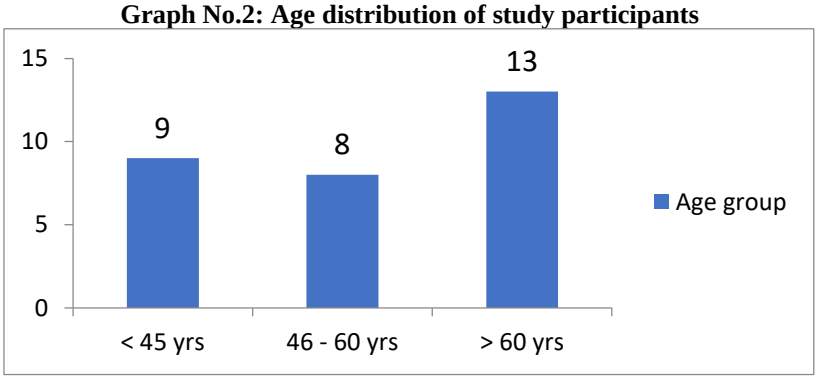
Study analysis: Data was entered using MS-Excel and analysed using IBM-SPSS version 26. Normality of the data was assessed using Shapiro-Wilk test of normality and $p > 0.05$ was classified as normally distributed. Qualitative data was measured by frequency and percentages and quantitative data was measured using measures of central tendency like mean and median and measures of dispersion like standard deviation. Difference between the mean was calculated using student t test or Mann Whitney test. Correlation was assessed using Pearson's correlation test. AUC ROC curve was used to detect differentiating power of MR Liver iron concentration. Graphical representation of data was done using tables and graphs.

IV. RESULTS

Table No.3: Age distribution of study participants

Age characteristic	**Years**
Mean age	55.1 ± 14.2 yrs
Range	28 – 75 yrs

Graph No.2: Age distribution of study participants

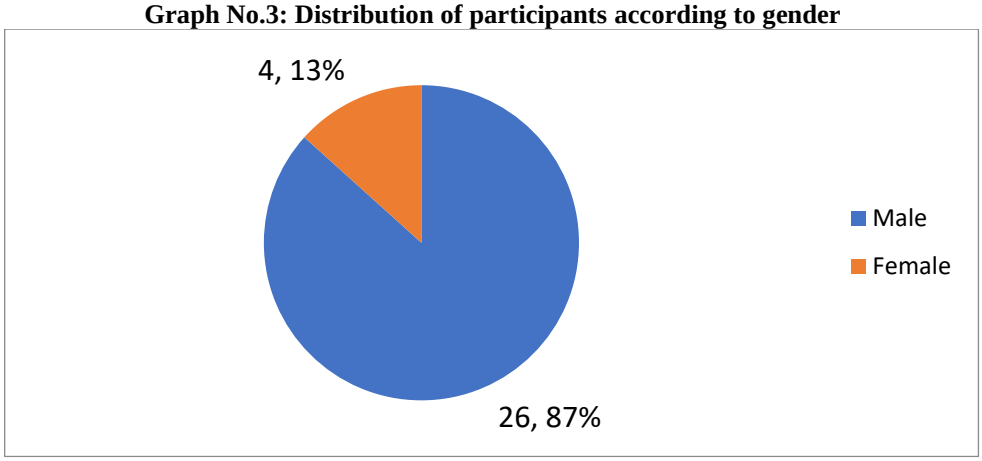


There were total 30 participants in the current study. The participants ranged from 28 – 75 yrs with the mean age of study participants of 55.1 ± 14.2 yrs. Out of them, 30.0% (9), 26.7% (8) and 43.3% (13) participants belonged to the age group of <45 yrs, 46-60 yrs and > 60 yrs respectively.

Table No.4: Distribution of study participants according to gender

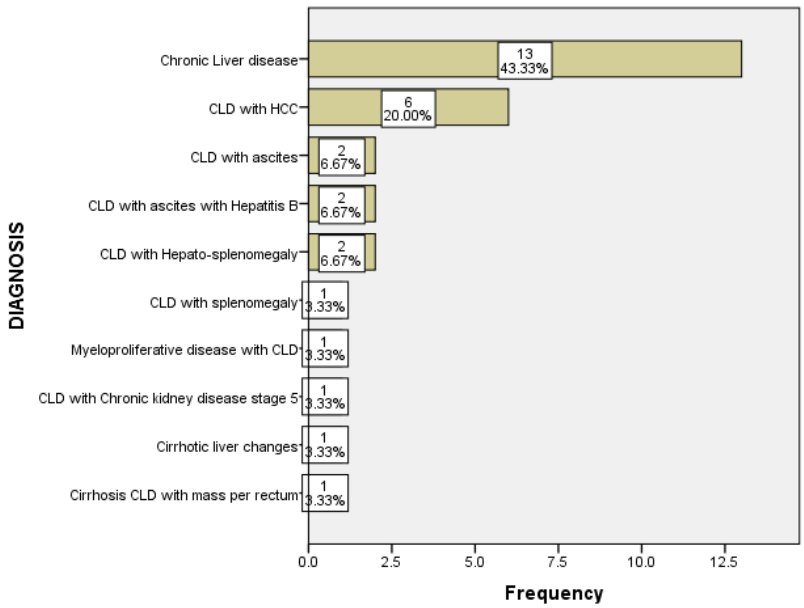
Gender	Frequency	Percentage
Male	26	86.7%
Female	4	13.3%

Graph No.3: Distribution of participants according to gender



86.7% (26) of the participants were male and rest 13.3% (4) were female.

Graph No.4: Distribution of cases based on diagnosis

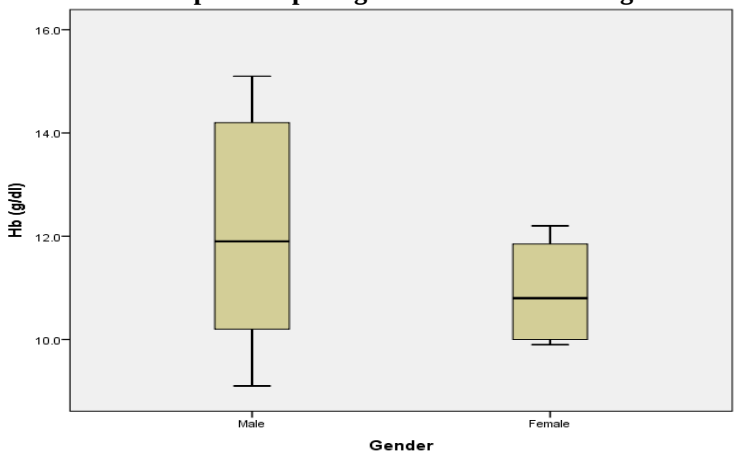


The current study was done on patients having chronic liver disease. While 43.3% (13) had been diagnosed of chronic liver disease alone, others were associated with other diagnosis. They were hepatocellular carcinoma (6, 20%), ascites (2, 6.67%), ascites and hepatitis B (2, 6.67%), with hepatosplenomegaly (2, 6.67%), splenomegaly (1, 3.3%), cirrhotic liver changes (1, 3.3%), cirrhotic liver changes (1, 3.3%), cirrhotic liver changes with mass per rectum (1, 3.3%), myeloproliferative disease (1,3.3%) and with stage V Chronic kidney disease (1, 3.3%).

Table No.5: Distribution of participants based on haemoglobin level

Haemoglobin	Male	Female	Total
Mean	12.1±1.9 g/dl	10.9±1.1 g/dl	12.0±1.9 g/dl
Range	9.1 - 15.1 g/dl	9.9 – 12.2 g/dl	9.1 – 15.1 g/dl

Graph No.5: Box and whisker plot comparing distribution of haemoglobin according to age

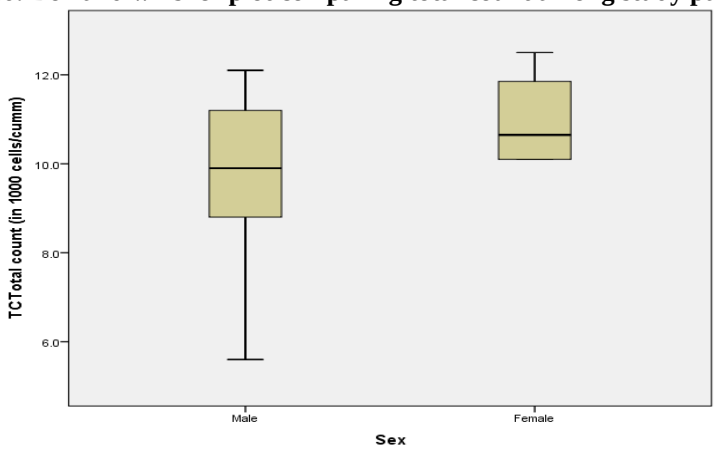


Overall Hb of study participants ranged from 9.1 - 15.1 g/dl with mean Hb of 12.0±1.9 g/dl. The mean Hb among male and female were 12.1±1.9 g/dl and 10.9±1.1 g/dl respectively.

Table No.6: Distribution of participants based on total count

Total count (cells/cumm)	**Male**	**Female**	**Total**
Mean	12,100 ± 9,665	10,975 ± 1,141	9,840 ± 1,706
Range	5,600 – 12,100	10,100 – 12,500 g/dl	5,600 – 12,500

Graph No.6: Box and whisker plot comparing total count among study participants

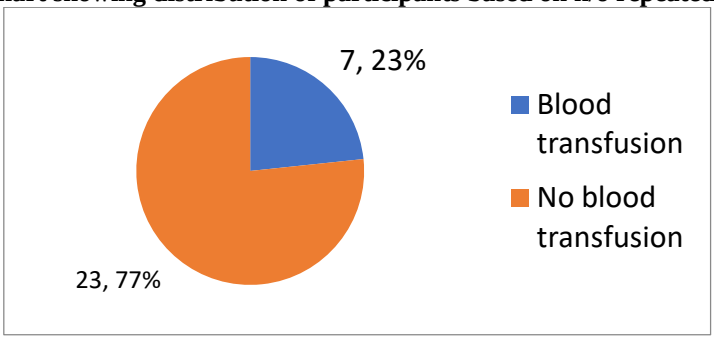


Total count ranged from 5,600 – 12,500 cells/cumm with mean of 9,840 ± 1,706. On further evaluation, mean total count among males and females were 12,100 ± 9,665cells/cumm and 10,975 ± 1,141 cells/cumm respectively.

Table No.7: Distribution of participants based on repeated blood transfusion

Blood transfusion	**Frequency**	**Percentage**
Yes	7	23.3
No	23	76.7
Total	30	100

Graph No.7: Pie chart showing distribution of participants based on h/o repeated blood transfusion



23.3% (7 participants) had history of repeated blood transfusion.

Table No.8: Distribution of participants based on Serum ferritin level

Serum ferritin µg/dL	Male	Female	Total
Mean	397 ± 457	361 ± 236	393 ± 431
Range	15 – 2,000	6.75 – 496	6.75 – 2,000

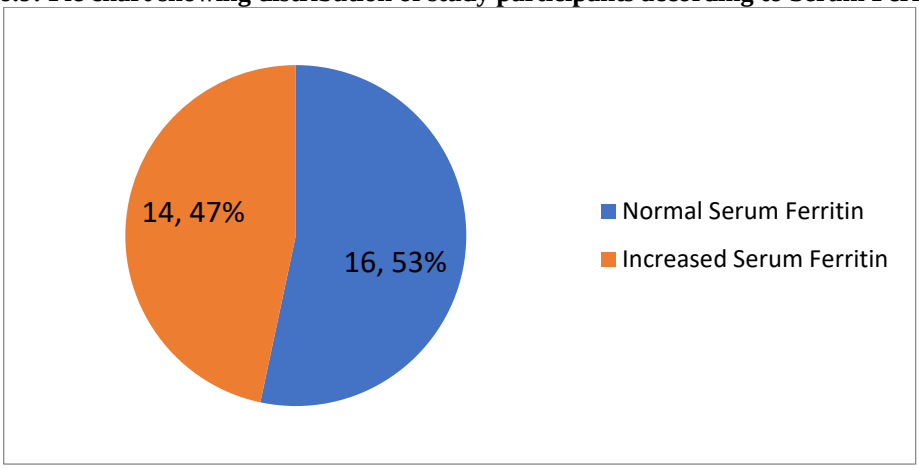
Graph No.8: Box and whisker plot comparing distribution of ferritin among gender

The variable serum ferritin was not normally distributed based on Shapiro Wilk assessment for normality (p=0.000). Serum Ferritin ranged from 6.75 – 2,000 µg/dL with mean of 393 ± 431 µg/dL. On further evaluation, mean total count among males and females were 397 ± 457 µg/dL and 361 ± 236 µg/dL respectively and this difference wasn't statistically significant (p=0.837). The median of serum ferritin was 329 µg/dL.

Table No.9: Distribution of cases based on Serum Ferritin level.

Serum Ferritin	Frequency	Percentage
Normal	16	53.3
Increased	14	46.7

Graph No.9: Pie chart showing distribution of study participants according to Serum Ferritin level

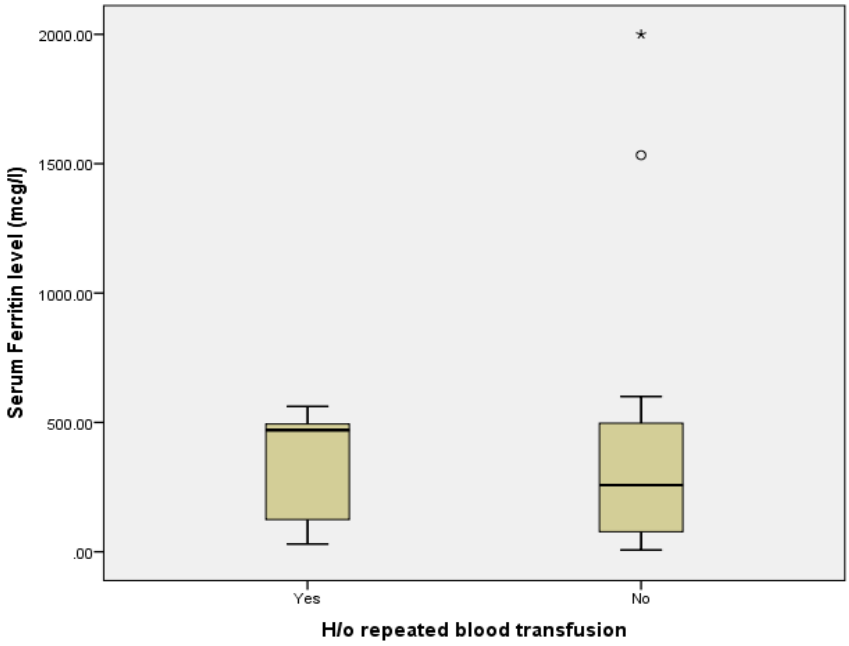


On further evaluation, it was noted that the serum ferritin was increased to > 464 µg/dL in 46.7% participants (14 cases).

Table No.10: Association between serum ferritin level and h/o repeated blood transfusion

Serum ferritin	Blood transfusion	No blood transfusion	P value
Mean	328 ± 228	412 ± 479	0.660

Graph No.10: Box and whisker blot comparing serum ferritin with h/o blood transfusion

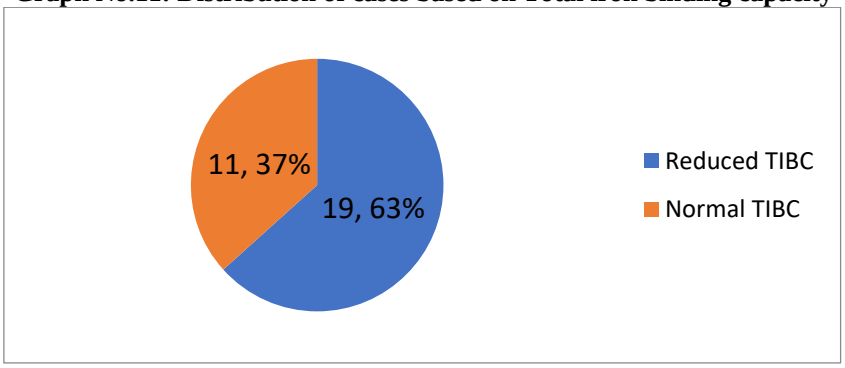


There is no significant difference in serum ferritin level between patients with or without blood transfusion ($p < 0.05$).

Table No.11: Total iron binding capacity among study participants

TIBC µg/dL	Male	Female	Total
Mean	243 ± 45	231 ± 28	242 ± 43
Range	188 – 350	203 – 270	188 – 350

Graph No.11: Distribution of cases based on Total iron binding capacity

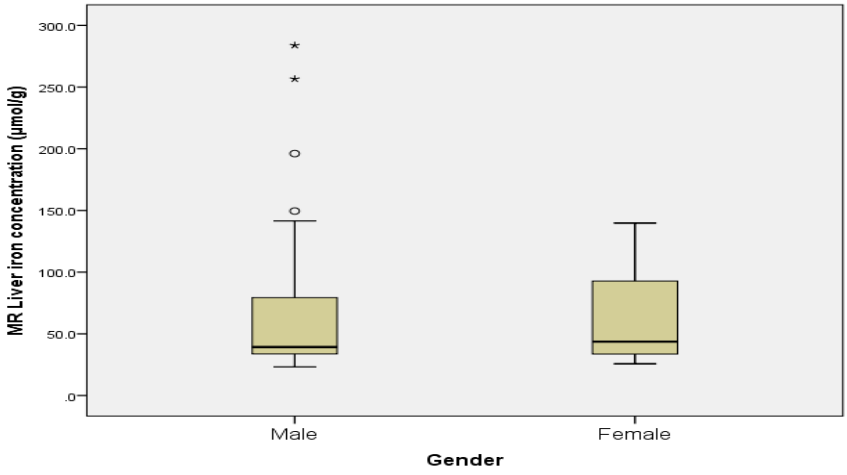


Total iron binding capacity wasn't normally distributed (Shapiro-Wilk test for normality: p = 0.01). The TIBC value ranged from 188 – 350 µg/dL with a mean of 242 ± 43 µg/dL. The mean TIBC among male and female were 243 ± 45 and 231 ± 28 µg/dL respectively and this difference wasn't statistically significant (p=0.584). Furthermore, 63% (19) participants had reduced TIBC value of < 261 µg/dL. The median value was 226 µg/dL.

Table No.12: T2 * MR Liver iron concentration among study participants

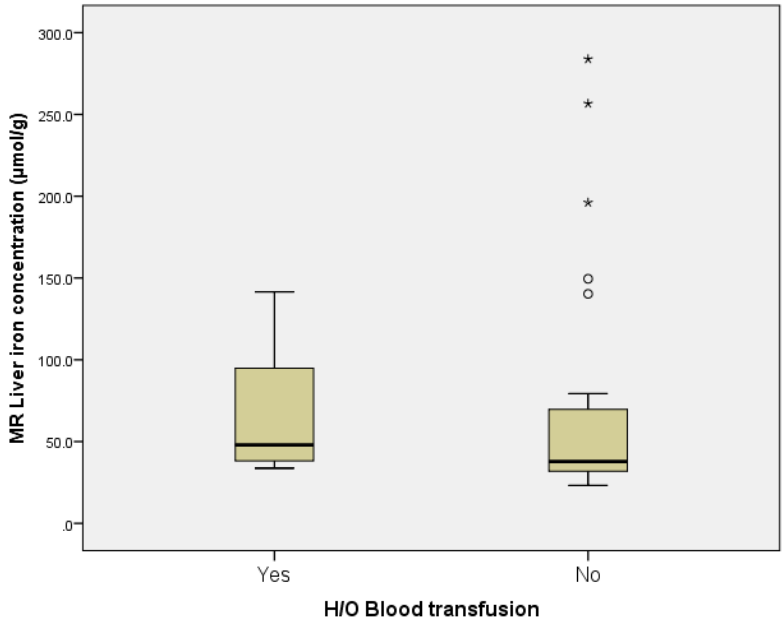
MR Liver iron concentration (µmol/g)	Male	Female	Total
Mean	75.2 ± 73.2	63.2 ± 51.8	73.6 ± 70.1
Range	23.2 – 283.8	25.7 – 139.8	23.2 – 283.8

Graph No.12: Box and Whisker plot comparing MR Liver iron concentration across gender



The data on MR liver iron concentration wasn't normally distributed (Shapiro-Wilk test for normality: p =0.00). The values ranged from 23.2 – 283.8µmol/g with a mean value of 73.6 ± 70.1µmol/g. The median value was 41.15µmol/g. There was no significance difference in mean liver iron concentration among gender (p = 0.756) with mean value among male and female were 75.2 ± 73.2 µmol/g and 63.2 ± 51.8 µmol/g respectively.

Graph No.13: Comparison of MR Liver concentration with h/o blood transfusion

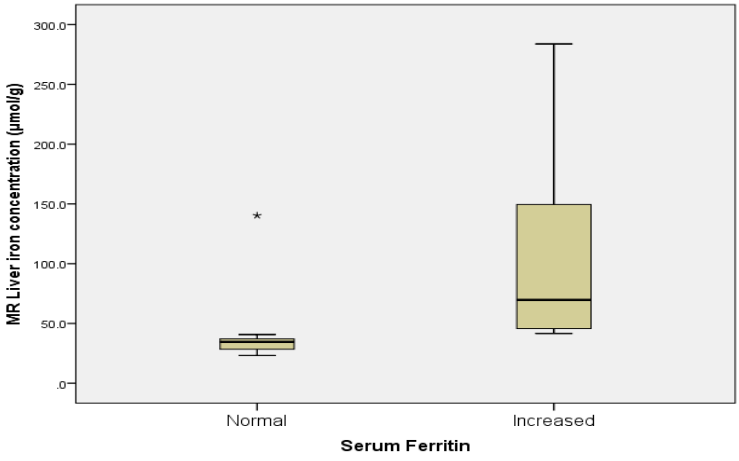


The mean MR Liver iron concentration among groups who had h/o blood transfusion was 69.9± 48.7 µmol/g and among the participants who didn't have h/o blood transfusion was 74.7±76.3 µmol/g and this difference wasn't statistically significant (p=0.877).

Table No.13: Comparison of MR Liver iron concentration based on serum ferritin

MR Liver iron concentration (µmol/g)	Normal Serum ferritin	Increased Serum Ferritin	P value
Mean	39.4±27.4	112.7±83.7	0.007

Graph No.14: Box and whisker plot comparing MR Liver iron concentration based on Serum Ferritin

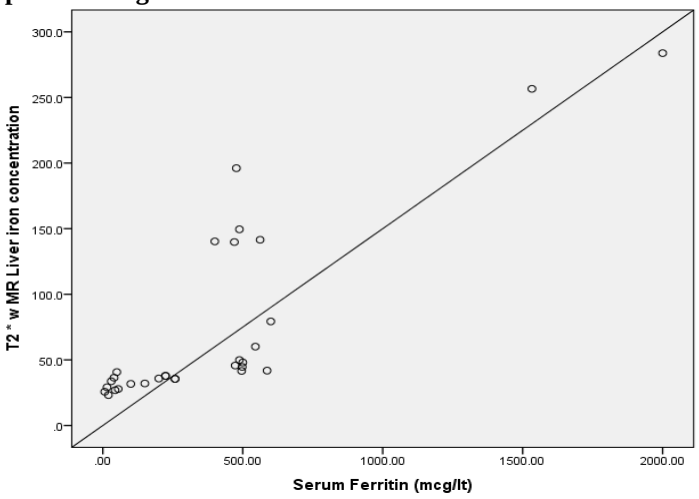


Mean MR Liver iron concentration among cases with normal Serum Ferritin and increased serum ferritin was 39.4±27.4 and 112.7±83.7 µmol/g respectively and this difference was statistically significant (p=0.007).

Table No.14: Correlation between MR Liver Iron concentration and serum ferritin

Correlation of MR Liver iron concentration with	Correlation Co-efficient	P value	Inference
Serum Ferritin	0.840	0.000	Positive, very strong and significant

Graph No.15: Scatter plot showing correlation between MR Liver iron concentration and serum ferritin



The study showed a positive, very strong and significant correlation between MR Liver iron concentration and serum ferritin (r = 0.840, p=0.000).

Table No.15: Comparison of MR Liver iron concentration with TIBC

MR Liver iron concentration (µmol/g)	Normal TIBC	Reduced TIBC	P value
Mean	32.8±7.7	97.2±79.2	0.002

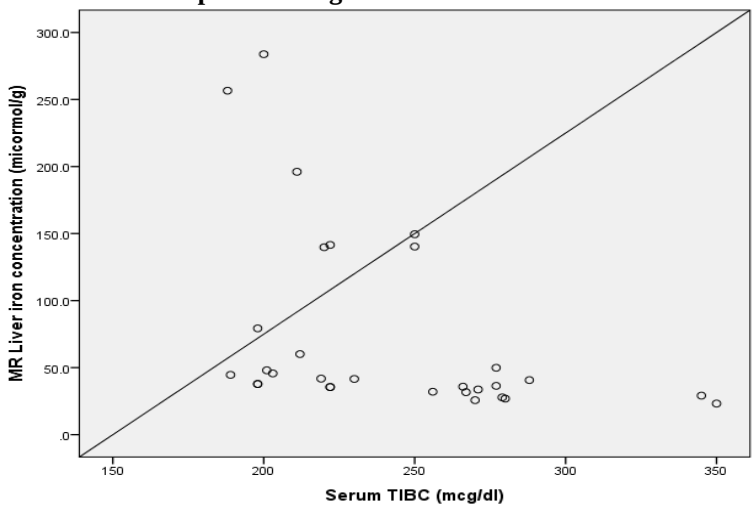
Graph No.16: Box and Whisker plot comparing MR Serum Liver iron concentration and TIBC

Mean MR Liver iron concentration among cases with normal Total iron binding capacity and reduced Total iron binding capacity was 32.8±7.7 and 97.2±79.2 µmol/g respectively and this difference was statistically significant (p=0.002).

Table No.16: Correlation between MR Liver iron concentration and Total iron binding capacity

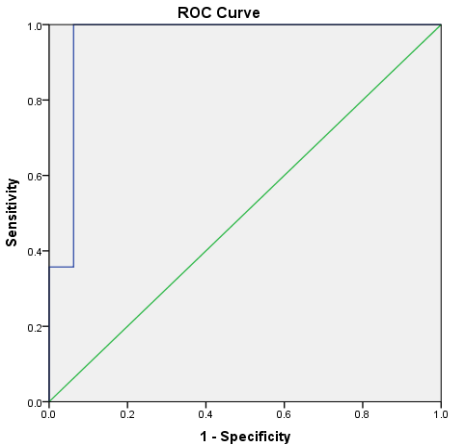
Correlation of MR Liver iron concentration with	Correlation Co-efficient	P value	Inference
TIBC	-0.421	0.021	Negative, medium and significant

Graph No.17: Scatter plot showing correlation between MR LIC and TIBC



The study showed a negative, medium and significant correlation between MR Liver iron concentration and Total iron binding capacity (r = -0.421, p=0.021).

Graph No.18: AUC-ROC curve for differentiating power of MR Liver iron concentration in differentiating normal from increased serum ferritin



The Area under receiver operator characteristic curve (AUC ROC) for differentiating power of MR Liver iron concentration of normal from increased serum ferritin was 140.9 µmol/g with 35.7% sensitivity and 100% specificity and for the value 41.5 µmol/g, it was 100% sensitive and 37% specific.

V. DISCUSSION

Chronic liver disease results in decreased production of hepcidin in liver, leading to increased accumulation of iron. The increased iron overload in the body combines with the reactive oxygen species causing increased hydroxyl radical leading to tissue damage. Studies have shown that the prevalence of iron overload in chronic liver disease ranges from 10-30%. Hence early detection of iron overload and its quantification is of importance and the current study, assessed a non invasive method (MRI) used worldwide in quantifying liver iron concentration.

The current study was done on 30 patients with chronic liver disease on patients ranging from 28-75 yrs with mean age of 55.1 $\pm$ 14.2 yrs. The mean serum ferritin among patients with chronic liver disease ranged from 75 – 2,000 µg/dL with a mean value of 393 $\pm$ 431 µg/Dl.

Similar results of increased serum ferritin with advancement of disease was in with advanced liver fibrosis in treatment-naive autoimmune hepatitis, chronic liver disease and with hepatic steatosis and fibrosis in hepatitis C virus–infected patients. In addition, serum ferritin serves as prognostic factors for mortality and survival in patients with end-stage liver disease.

Finally, liver biopsy has drawbacks of sampling error, invasive nature, increased risk of hospitalization and discomfort. Hence serum ferritin was used as a biomarker for comparison of liver iron concentration in current study. Our study analyzed Total iron binding capacity and its correlation with MR Liver iron concentration. Transferrin is the iron transport protein in the blood and is usually 33% saturated and in iron overload cases, the relative transferrin content compared to iron content reduces leading to reduced TIBC. In the current study, the median TIBC was 226µg/dL with 63% participants having reduced TIBC. However, it should be noted that TIBC may be decreased in chronic liver diseases as transferrin is produced in the liver.

The main aim of the study was to determine the role of MR imaging in detecting liver iron concentration by comparing it with the Serum Ferritin and TIBC. MR imaging is a recognized alternative to serum ferritin and standard liver biopsy for quantification of liver iron concentration. The current study showed that the mean LIC using MRI was 73.6 $\pm$ 70.1 µmol/g. Our study showed that the MRI LIC significantly increased in cases with increased ferritin compared to patients with normal ferritin and the AUC ROC for differentiating power of MR Liver iron concentration of normal from increased serum ferritin was 140.9 µmol/g with 35.7% sensitivity and 100% specificity and for the value 41.5 µmol/g, it was 100% sensitive and 37% specific. Furthermore, there was very strong and significant correlation (r=0.840) between MRI LIC and serum ferritin and a negative, medium and significant correlation with TIBC (r = -0.421).

VI. CONCLUSION

MRI liver iron concentration is a quantitative technique with high correlation to serum ferritin in detection of hepatic iron concentration. The cut off point for detection of increase liver iron was 140.9µmol/g with poor sensitivity but high specificity. It can be used as a non invasive technique in diagnosis and monitoring of serum iron in chronic liver disease.

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