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MRI-Based Iron Load Assessment in Chronic Liver Disease

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Abstract

Background: Chronic liver disease (CLD) is frequently associated with disturbances in iron metabolism and hepatic iron deposition, which may not be accurately reflected by serum biomarkers alone. Magnetic Resonance Imaging (MRI) offers a non-invasive and sensitive method for quantifying liver iron concentration.

Objectives: To evaluate and compare iron metabolism parameters and hepatic iron deposition using MRI-based assessment in patients with chronic liver disease versus healthy controls.

Materials and Methods: This cross-sectional comparative study included 35 patients diagnosed with CLD and 35 age- and gender-matched healthy controls. Clinical evaluation, biochemical parameters including serum ferritin, TIBC, UIBC, serum iron, transferrin saturation, liver enzymes, and albumin were analyzed. MRI was performed to assess liver iron concentration using R^2 mapping, visual impression grading, and structured MRI grading. Statistical comparisons between groups were done using independent samples t-test and Chi-square test, with $p < 0.05$ considered statistically significant.

Results: CLD patients exhibited significantly lower serum ferritin (149.35 ± 286.86 ng/mL vs. 213.48 ± 373.13 ng/mL; $p = 0.001$) and R^2 values (57.82 ± 29.2 vs. 76.43 ± 47.79; $p = 0.012$), suggesting higher hepatic iron overload. UIBC was significantly elevated in CLD ($p = 0.043$), while serum iron and transferrin saturation showed no significant difference. Liver enzymes (SGOT, SGPT) were significantly higher in the CLD group, and serum albumin was lower, though not statistically significant. MRI grading showed a significantly higher proportion of abnormal iron load in CLD ($p = 0.0045$), and light iron overload was more frequently observed in MRI impressions among CLD patients.

Conclusion: Patients with chronic liver disease demonstrate significant disruptions in iron metabolism and increased hepatic iron deposition as detected by MRI. Quantitative MRI, particularly R^2 mapping and structured grading, is a valuable tool for non-invasive assessment of liver iron overload and may be superior to serum markers in detecting early or subclinical hepatic siderosis.

Keywords: Chronic Liver Disease, MRI R^2 Mapping, Hepatic Iron Overload, Serum Ferritin, Iron Metabolism, Non-invasive Imaging, Liver Function Tests.

Introduction

Haemochromatosis is the predominant aetiology of iron excess (1). The fundamental hereditary condition involves increased intestinal absorption of iron, which the body cannot excrete, leading to accumulation in numerous organs and resulting in irreparable harm. In the secondary form, excess iron originates from several blood transfusions or haematological diseases, such as haemolytic anaemia or inadequate erythropoiesis, resulting in gradual buildup in various organs. In both instances, the liver and heart are the most adversely affected organs (2). The process can be reversed with treatment, employing phlebotomies in initial haemochromatosis instances and utilising iron chelating medicines in those with the secondary variant.

While iron is a vital mineral, excessive quantities can be detrimental to human health. Its detrimental effects are believed to result from heightened oxidative stress and the generation of damaging reactive oxygen species. Elevated iron levels in hepatocytes and higher total body iron in hemochromatosis correlate with a heightened risk of cirrhosis and hepatocellular cancer (3). Elevated iron deposition has been linked to several systemic illnesses, such as chronic viral hepatitis, alcoholic liver disease, and nonalcoholic steatohepatitis. Data indicate that hepatic iron accumulation may synergistically interact with alcohol and fat accumulation in facilitating the progression of hepatic fibrosis and cirrhosis, and iron reduction therapy has been proposed to improve the efficacy of interferon treatment for chronic hepatitis C and hepatitis B. Consequently, it is essential to detect the presence of excessive intrahepatic iron. The gold standard for hepatic iron quantification is core liver biopsy (4).

Indirect measures, like serum ferritin and transferrin saturation, have been recognised as sensitive yet nonspecific. The diagnosis of primary haemochromatosis has advanced due to the identification of several related genetic abnormalities in recent years. Nonetheless, the primary metric is the assessment of liver iron content (LIC) (5). Non-invasive methods for assessing hepatic iron, such as the superconducting quantum interference device and MRI, have been detailed. MRI has become an accessible non-invasive method for assessing hepatic iron (6). Spin-echo and gradient-echo techniques utilising extended TEs have been employed to assess tissue transverse relaxation values (R2 and R2*, respectively), which are affected by the paramagnetic effects of iron.

Iron overload is identified on magnetic resonance imaging (MRI) by its reduction of tissue signal intensity. Iron accumulation in the liver and pancreas is indicative of severe hereditary hemochromatosis; a distribution of iron overload in the liver, spleen, and bone marrow pertains to iron within the mononuclear phagocyte system cells; whereas localised liver deposition is typically linked to milder variants of hereditary hemochromatosis or chronic liver conditions (7). Patients with chronic diffuse liver disorders with no history of blood transfusions frequently exhibit a diverse range of iron distribution in the liver, pancreas, spleen, and bone marrow on MRI examinations, indicating that iron accumulation may extend beyond the liver. The quantitative MRI evaluation of liver iron overload through transverse relaxation rates (R2*) relaxometry has become a safe, non-invasive, and precise substitute for liver biopsy in assessing and monitoring patients with hereditary haemochromatosis and transfusion-related haemosiderosis (8). R2* relaxometry employs a sequence of pictures obtained using a multi-echo gradient echo protocol, characterised by progressively rising echo times (TE). This sequence can be executed during a single breath-hold acquisition. The hepatic signal intensity is represented as a multi-exponential periodic complex function of TE, from which the signal decay rate is then computed. A higher quantity of liver iron correlates with an increased relaxation rate (R2*) (9). Compared to alternative MRI techniques, such as R2 relaxometry or signal intensity ratio methods, R2* relaxometry utilising multi-echo chemical

shift-encoded sequences offers the benefit of expedited acquisition and concurrent assessment of hepatic iron and fat.

This field is currently significant: in primary haemochromatosis, genetic mutations, extensively researched in recent years, are not definitive for the diagnosis of the disease (10,11); in the secondary form, the introduction of new iron chelating agents is altering clinical management.

The objective of this study was to compare and assess the efficacy of MRI in quantifying iron load in patients with chronic liver disease vs those without chronic liver disease.

Methodology

Study design: The present cross-sectional study was to evaluate the patients suffering from chronic liver disease and non-chronic liver disease patients

Study population: All Chronic Liver disease patients, of both genders were recruited for the study.

Setting: The study was conducted in the department of medicine, CSS Hospital, Subharti Medical College, Meerut.

Study Unit: All chronic liver disease patients and non-chronic liver disease patients of both genders were recruited from medicine OPD in the study

Study Setting: Medicine OPD CSS Hospital, Subharti Medical College, Meerut.

Study period: The study was done from July 2023 to Feb 2025.

Ethical consent: Informed consent of all participants was obtained after explaining the purpose of the study. Permission to carry out the study was obtained by Institutional Ethical Committee of CSS Hospital, Subharti Medical College, Meerut.

Sample: The study sample was comprised of 70 participants, 35 chronic liver disease patients and 35 non chronic liver disease who were sequentially allocated into study based on their criteria for fulfilling eligibility criteria. The patient scanned by the T2* protocol, without any fasting or contrast ingestion or injections. The sample size was calculated using the formula for estimating a single proportion for a large population: $n = (Z^2 \times P(1-P)) / d^2$, where $Z = 1.96$ (for 95% confidence), $P = 0.96$ (based on prior sensitivity estimates), and $d = 0.15$ (absolute precision). This yielded a minimum required sample size of approximately 59. For robust statistical power and to account for potential attrition, the final sample size was increased to 70, with 35 participants each in the chronic liver disease (CLD) group and the control group.

Inclusion and Exclusion Criteria:

The study included adult patients diagnosed with chronic liver disease. Exclusion criteria comprised patients with malignancies, severe comorbid conditions such as end-stage renal disease or chronic obstructive pulmonary disease, recent blood transfusions (within three months), HIV positivity, dyslipidemia, diabetes mellitus, acute liver failure, and pregnancy. Individuals who declined to provide written informed consent were also excluded from the study.

Method:

The study utilized retrospective data from the hospital's medical records, including laboratory investigations such as serum iron, prolactin, total iron-binding capacity (TIBC), international normalized ratio (INR), liver function tests (LFT), and viral serology. Upper gastrointestinal endoscopy findings for esophageal varices, admission and discharge dates, clinical outcomes (survival or mortality), diagnoses, and associated comorbidities were also recorded. Data sources included personal and demographic information, detailed medical histories, MRI abdomen images, and various laboratory parameters

including complete blood count (CBC) and total iron profile. MRI signal intensity analysis was performed by placing a region of interest (ROI) of 1–1.4 cm² in the right lobe of the liver—avoiding vascular and biliary structures—to ensure measurement accuracy. The Hankins equation was applied using an Excel template to interpret TE signal intensities across imaging slices. For quantifying hepatic iron content, a gradient echo protocol was employed to calculate T2* values and their reciprocal R2* (=1/T2*), offering improved sensitivity over conventional T2 techniques. Due to magnetic field inhomogeneity and susceptibility effects caused by high iron concentration, R2* mapping enabled more precise detection, capturing the rapid signal decay characteristic of iron overload.

Statistical analysis was conducted using SPSS version 21.0. Continuous variables (e.g., age, BMI, liver enzymes, ferritin, iron indices, albumin, and MRI R^2 values) were expressed as mean ± SD and compared between CLD and control groups using independent t-tests. Categorical variables (e.g., gender, BMI category, MRI impression/grading) were analyzed using the Chi-square test. A p-value <0.05 was considered statistically significant. MRI-derived R^2 values were evaluated to quantify hepatic iron, with lower values indicating greater iron overload. Findings were summarized using tables and figures.

Result

The present cross-sectional study was carried out at CSS Hospital, Subharti Medical College, Meerut, from July 2023 to February 2025, including 70 participants—35 with chronic liver disease (CLD) and 35 without (Non-CLD). All subjects underwent clinical evaluation and laboratory tests including liver function tests, complete blood count, serum iron profile, and viral serology. Demographic details such as age, gender, BMI, and alcohol history were documented. Data were analyzed using SPSS v13.2, with t-tests for continuous variables and chi-square tests for categorical data. A p-value <0.05 was considered significant. The analysis primarily compared ferritin, transferrin saturation, and MRI R2* values between groups to evaluate hepatic iron overload.

Table 1: Distribution of Study Groups According to Demographic Variables

Variable	Categories	CLD Cases (n=35)	Controls (n=35)	p-value
Age	Mean ± SD	49.34 ± 12.58	48.79 ± 16.58	0.877
Gender	Male	14 (40.0%)	13 (37.14%)	0.687
	Female	21 (60.0%)	22 (62.86%)	
BMI (kg/m²)	Mean ± SD	23.8 ± 7.1	22.5 ± 6.7	0.087

The demographic comparison between the chronic liver disease (CLD) group and control group showed no statistically significant differences across age, gender, or BMI. The mean age was comparable between CLD cases (49.34 ± 12.58 years) and controls (48.79 ± 16.58 years) with a p-value of 0.877, indicating well-matched age distribution. Similarly, gender distribution was nearly equal, with 40% males and 60% females in the CLD group, compared to 37.14% males and 62.86% females in controls (p = 0.687). The mean BMI was slightly higher in the CLD group (23.8 ± 7.1 kg/m²) than in controls (22.5 ± 6.7 kg/m²), but the difference was not statistically significant (p = 0.087), suggesting homogeneity between groups in terms of baseline demographic characteristics (Table 1).

Table 2: Distribution Of Study Groups According To Iron Metabolism

Iron Metabolism	CASES	CONTROLS	P-VALUE
TIBC (Total Iron Binding Capacity)	285.73±77.28	274.93±93.3	0.05
Ferritin	149.35±286.86	213.48±373.13	0.001
S. Iron (Serum Iron)	67.07±39.16	66.40±38.82	0.658
UIBC (Unsaturated Iron Binding Capacity)	215.04±91.09	204.29±100.46	0.043
Transferrin Saturation	24.12±16.75	26.62±17.77	0.054
Estimated Iron	3.49±7.28	2.25±1.58	0.002

The analysis of iron metabolism parameters between chronic liver disease (CLD) patients and controls revealed several notable differences mentioned in Table 2. Ferritin levels were significantly lower in the CLD group (149.35 ± 286.86 ng/mL) compared to controls (213.48 ± 373.13 ng/mL), with a highly significant p-value (0.001), suggesting altered iron storage in CLD. Estimated iron was also significantly higher in CLD patients (3.49 ± 7.28 µmol/L vs. 2.25 ± 1.58 µmol/L; p = 0.002), indicating potential iron overload despite lower ferritin. UIBC levels were elevated in the CLD group (215.04 ± 91.09 µg/dL) compared to controls (204.29 ± 100.46 µg/dL), reaching statistical significance (p = 0.043). TIBC and transferrin saturation values were comparable between the groups, with marginal p-values (0.05 and 0.054, respectively), while serum iron levels showed no significant difference (p = 0.658).

Table 3: MRI-Based Assessment of Hepatic Iron Overload in Study Groups

MRI Parameter	Categories	CLD Cases (n=35)	Controls (n=35)	Total (n=70)	p-value
Average R^2 Value	Mean ± SD	57.82 ± 29.2	76.43 ± 47.79	–	0.012
MRI Impression	Light	15 (42.86%)	11 (31.4%)	26 (37.14%)	0.400
	Normal	20 (57.14%)	24 (68.6%)	44 (62.86%)	
MRI Grading	Grade 1	10 (28.57%)	8 (22.86%)	–	0.0045
	Grade 2	3 (8.57%)	2 (5.71%)	–	
	Grade 3	1 (2.86%)	1 (2.86%)	–	
	Normal	20 (57.14%)	24 (68.57%)	–	

MRI-based evaluation revealed a significantly lower mean R^2 value in chronic liver disease (CLD) cases (57.82 ± 29.2) compared to controls (76.43 ± 47.79), with a p-value of 0.012, suggesting increased hepatic iron overload in the CLD group. Regarding qualitative MRI impressions, 42.86% of CLD patients showed signs of light iron overload, compared to 31.4% in the control group, though this difference was not statistically significant (p = 0.400). In MRI grading, a higher proportion of CLD patients had abnormal iron load—Grade 1 (28.57%), Grade 2 (8.57%), and Grade 3 (2.86%)—as compared to controls with Grade 1 (22.86%), Grade 2 (5.71%), and Grade 3 (2.86%). Normal MRI findings were more frequent in the control group (68.57%) than in CLD patients (57.14%). The overall difference in MRI grading was statistically significant (p = 0.0045), reinforcing the association between CLD and hepatic iron accumulation (Table 3).

Discussion

The present cross-sectional study was conducted at the Department of Medicine, CSS Hospital, Subharti Medical College, Meerut, from July 2023 to February 2025, and included a total of 70 participants. Among these, 35 patients were diagnosed with chronic liver disease (CLD group) and 35 patients did not have chronic liver disease (Non-CLD group), meeting the eligibility criteria as defined in the study protocol.

In the present study, the mean age of CLD patients (49.34 ± 12.58 years) and controls (48.79 ± 16.58 years) showed no significant difference ($p = 0.877$), aligning with França et al. [12], Hernando et al. [13], and Sirlin & Reeder [93], who noted that liver iron accumulation in CLD is more linked to disease severity than age. However, this contrasts with Tada et al. [14], who observed higher iron overload in older individuals (≥60 years), whereas our population showed substantial representation in the 31–60 age group, indicating earlier onset. Females were predominant in both CLD (60%) and control (62.86%) groups ($p = 0.687$), suggesting gender-neutral distribution. Hernando et al. [13] and Sirlin & Reeder [15] also observed that MRI-based hepatic iron quantification is unaffected by sex. Reeder et al. [100] further supported that MRI methods provide consistent results across genders. The mean BMI was slightly higher in CLD (23.8 ± 7.1) than controls (22.5 ± 6.7) but was not statistically significant ($p = 0.087$), consistent with Imajo et al. [16] and França et al. [12], who noted that BMI does not strongly correlate with hepatic iron overload, reaffirming that MRI quantification is accurate regardless of body composition, as also highlighted by Hernando et al. [13].

In our study, UIBC was significantly elevated in CLD patients (215.04 ± 91.09 µg/dL) compared to controls (204.29 ± 100.46 µg/dL) ($p = 0.043$), indicating altered iron transport. Serum iron levels were similar between groups, and transferrin saturation was marginally lower in CLD (24.12%) than controls (26.62%) ($p = 0.054$). These results align with Tziomalos and Perifanis [17] and Sirlin & Reeder [15], who reported that biochemical markers like serum iron and transferrin saturation may not reliably indicate hepatic iron overload in CLD. França et al. [12] and Hernando et al. [13] also highlighted their limited sensitivity, supporting MRI as a more accurate tool. Our findings suggest impaired iron utilization and support the role of MRI in evaluating hepatic iron more precisely.

In our study, the MRI-derived average R^2 value—a key indicator of hepatic iron content—was significantly lower in CLD patients than in controls (57.82 ± 29.2 vs. 76.43 ± 47.79; $p = 0.012$), indicating greater iron deposition in CLD. This finding is supported by Hernando et al. [13], França et al. [12], and Imajo et al. [16], who emphasized the reliability of R^2 and R2* mapping in detecting hepatic iron overload. These authors confirmed that lower R^2 values correlate with increased liver iron and disease progression, reinforcing MRI's role in liver iron quantification over traditional biochemical markers.

MRI-based impressions showed a higher frequency of light iron overload in CLD patients (42.86%) compared to controls (31.4%), while normal findings were more common among controls (68.6%) than in CLD (57.14%), though the difference was not statistically significant ($p = 0.400$). This pattern supports the trend of mild hepatic siderosis in CLD, consistent with França et al. [12], Sirlin and Reeder [15], and Reeder et al. [18], who highlighted the clinical utility of qualitative MRI impressions, especially when quantitative mapping is unavailable or limited.

Additionally, MRI grading revealed a significant difference between groups ($p = 0.0045$), with more CLD patients exhibiting Grade 1 to 3 hepatic iron overload. These results align with França et al. [12], Hernando et al. [13], and Reeder et al. [18], who emphasized the accuracy of MRI grading in stratifying liver iron burden and its correlation with fibrosis and disease severity. Thus, our findings confirm the diagnostic

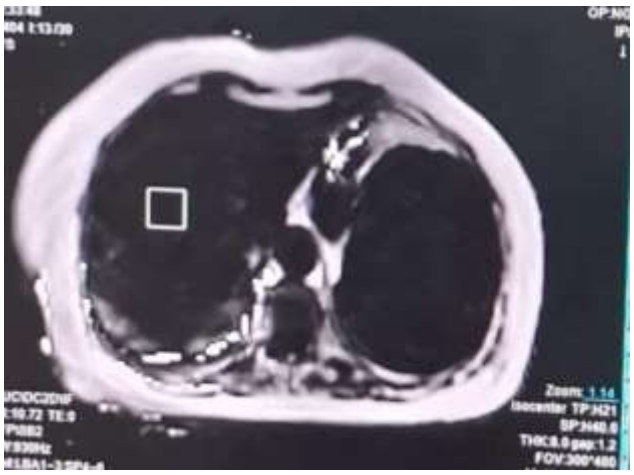
value of MRI-based metrics—including R^2 values, qualitative impressions, and grading—in assessing hepatic iron overload in chronic liver disease.

Conclusion

This study demonstrated that patients with chronic liver disease (CLD) exhibit significant alterations in iron metabolism and hepatic iron deposition compared to non-CLD individuals. Despite comparable age, gender, and BMI distributions, CLD patients showed significantly lower serum ferritin and higher estimated iron levels, suggesting disrupted iron storage and mobilization. Liver function tests revealed elevated transaminases, indicating hepatocellular injury, and a trend toward hypoalbuminemia reflected impaired synthetic capacity. MRI-based assessments further supported increased hepatic iron accumulation in CLD patients, as evidenced by significantly lower average R^2 values and higher grades of iron overload on MRI grading. These findings reinforce the utility of MRI as a non-invasive and reliable tool for quantifying liver iron load and support its role in the clinical evaluation and monitoring of chronic liver disease.

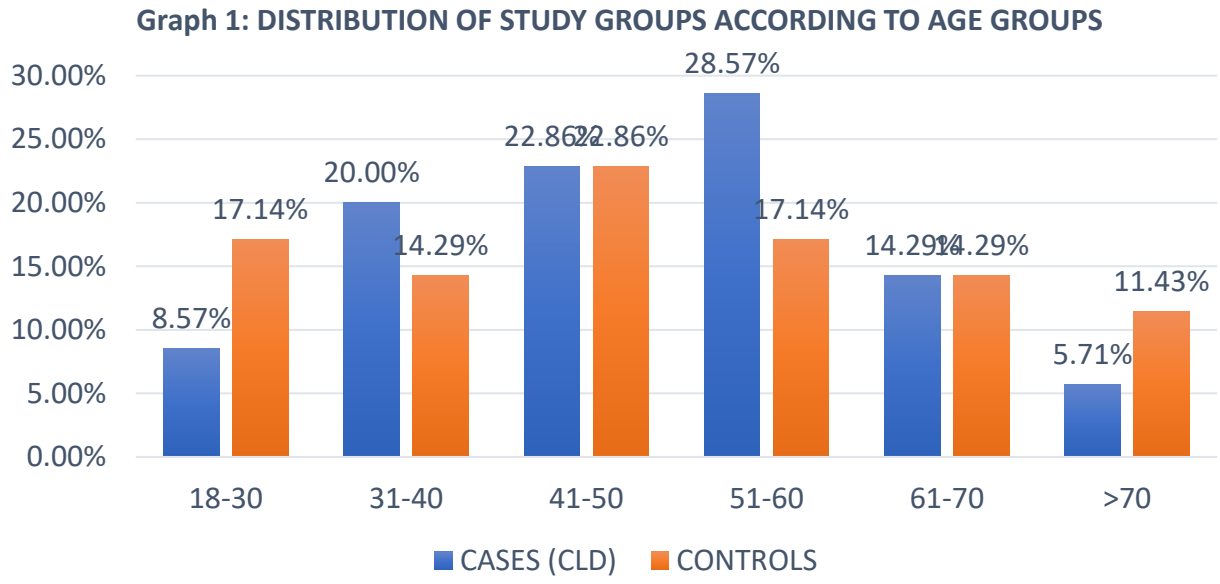
Images

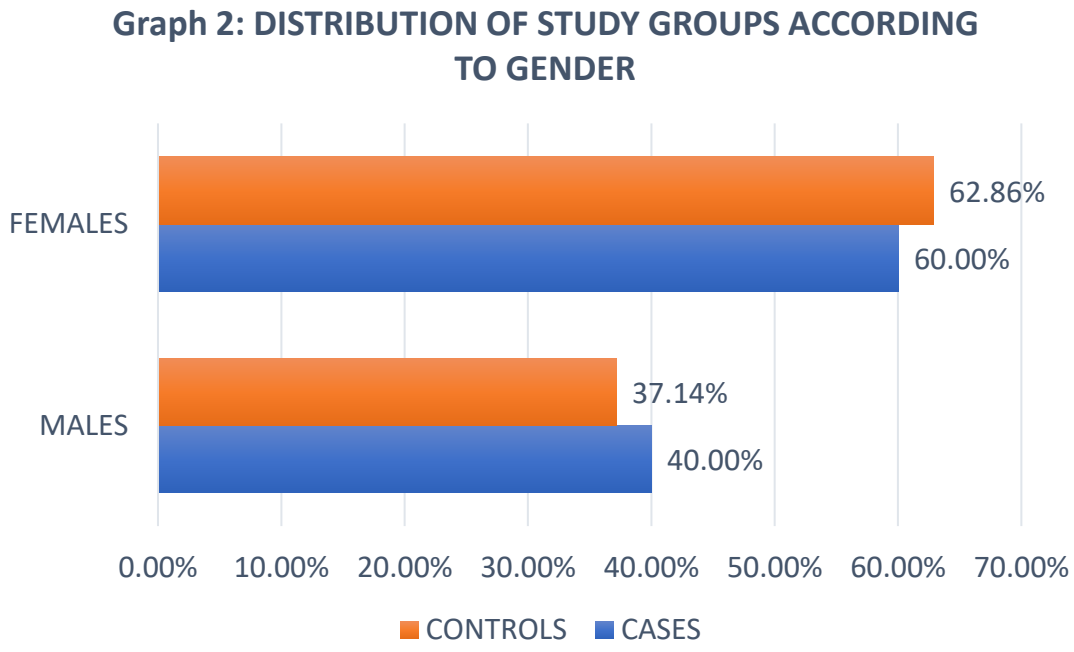
1.MRI image of iron quantification



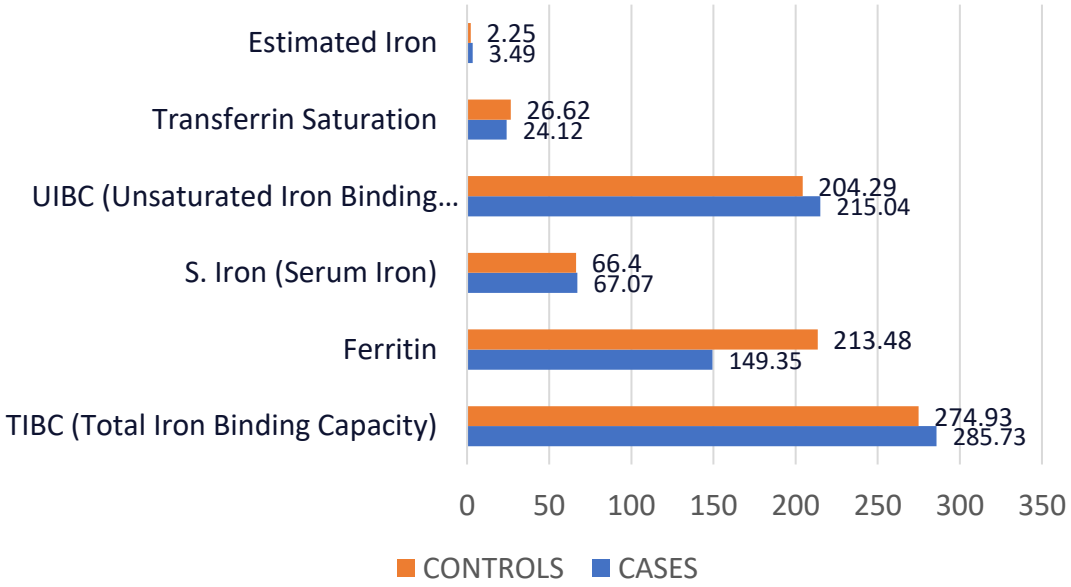
2.MRI image of liver iron quantification

GRAPHS

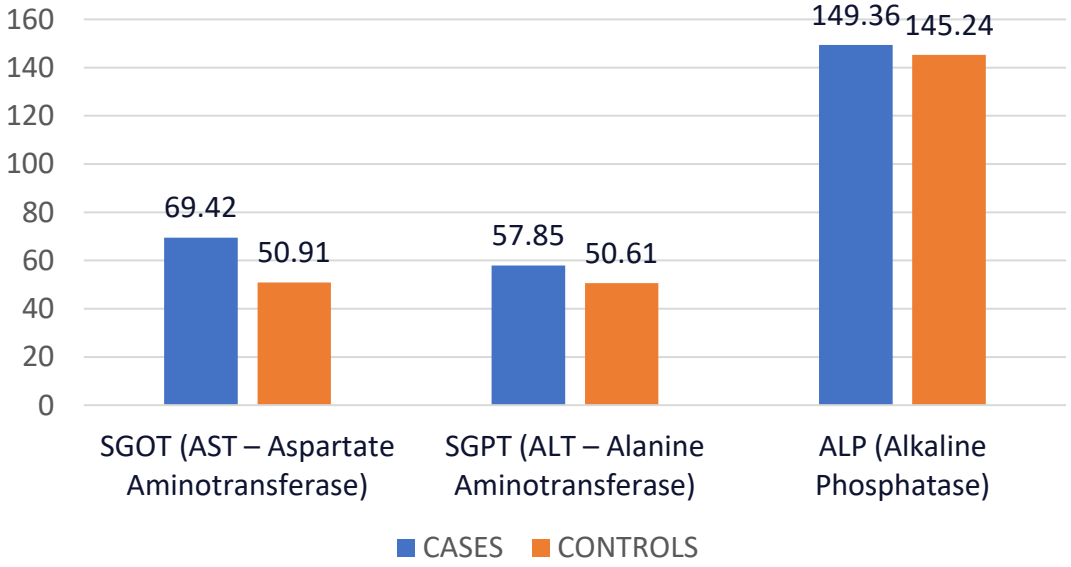

Graph 1: DISTRIBUTION OF STUDY GROUPS ACCORDING TO AGE GROUPS
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
8.57%
17.14%
20.00%
14.29%
22.86%
22.86%
28.57%
17.14%
14.29%
14.29%
5.71%
11.43%
18-30
31-40
41-50
51-60
61-70
>70
CASES (CLD)
CONTROLS


Graph 2: DISTRIBUTION OF STUDY GROUPS ACCORDING TO GENDER
FEMALES
MALES
62.86%
60.00%
37.14%
40.00%
0.00%
10.00%
20.00%
30.00%
40.00%
50.00%
60.00%
70.00%
CONTROLS
CASES

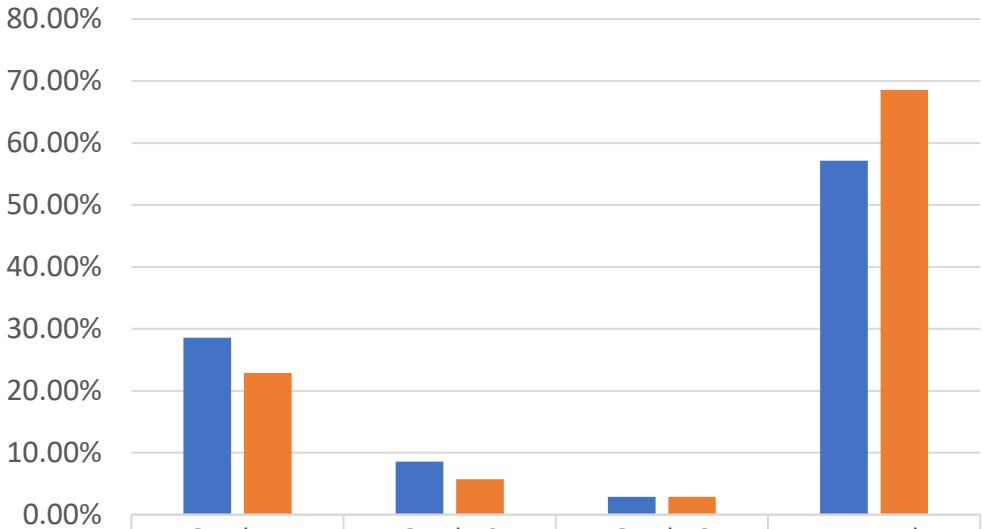
Graph 3:DISTRIBUTION OF STUDY GROUPS ACCORDING TO Iron Metabolism



Graph 4: DISTRIBUTION OF STUDY GROUPS ACCORDING TO Liver Function Tests



Graph 5: DISTRIBUTION OF STUDY GROUPS ACCORDING TO MRI Grading



	Grade 1	Grade 2	Grade 3	Normal
CASES	28.57%	8.57%	2.86%	57.14%
CONTROLS	22.86%	5.71%	2.86%	68.57%

CASES CONTROLS

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