

Analyzing of Serum Iron Markers in Liver Cirrhosis Patients in Basra Province - Iraq

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ABSTRACT

Liver cirrhosis (LC) is a progressive and chronic liver disorder. However, researches on LC are bounded yet. This research aims to explore the relationship between iron markers levels in LC patients comparing with controls in Basra Province-Iraq. In this cross-sectional study, a total of 50 patients with LC and 75 healthy controls was participated. Blood samples were collected from all participants, and serum ferritin, transferrin saturation (TS), serum iron (SI), total iron binding capacity (TIBC), and transferrin were measured using enzyme-linked immunosorbent assay (ELISA) and Automated Analyzer. Serum levels of ferritin and TS are elevated high-significantly ($p < 0.01$), while, SI, transferrin, and TIBC serum levels are decreased high-significantly ($p < 0.01$) in LC patients compared to the controls. The findings illustrate that ferritin and TS have a high significant positive correlation between them, high significant positive correlations with SI, and high significant negative correlations with transferrin and TIBC. Moreover, the area under the receiver operating characteristics (ROC) curve (AUC) was calculated for ferritin, TS, SI, transferrin, and TIBC (AUC = 0.938, 0.952, 0.100, 0.029, and 0.007, respectively) which could be prospective markers in LC patients. The correlations of ferritin and TS with SI, transferrin, and TIBC and the high positive value of AUC for ferritin and TS shows that ferritin and TS could be potential markers for the onset of liver failure in LC patients. These results highlight the importance of serum iron markers in LC and suggest that further researches are needed to explore LC markers.

Keywords: Cirrhosis; Ferritin; Iron; Liver; Transferrin Saturation

INTRODUCTION

The liver is a substantial organ in the human body. It has numerous vital biological functions including maintaining metabolic homeostasis¹. One of the most crucial roles of homeostasis of the liver is iron homeostasis².

Liver cirrhosis (LC) is a progressive and chronic liver disorder. It is characterized by regenerative nodule formation, comprehensive fibrosis, and liver malformation³. Due to liver damage is continued, the functional capacity of liver is progressively diminished. This will lead to many metabolic and biochemical disturbances in the body⁴. LC etiology is usually diverse and multifactorial⁵. One of the fastest rising causes of LC globally is metabolic LC which is substantially connected to modern lifestyle disorders such as obesity⁶.

Iron, an indispensable trace metal, has pivotal roles in different biological actions such as synthesis of DNA, cellular respiration, and transport of oxygen⁷. Nevertheless, iron homeostasis has to be controlled firmly due to the harmful effects of both of iron overload and deficiency in the body⁸. Various fundamental biochemical markers are utilized to evaluate iron in the clinical practice and give a worthy data regarding iron transport, storage and using in the body such as serum levels of ferritin, transferrin saturation (TS), serum iron (SI), transferrin, and total iron binding capacity (TIBC)⁹. The serum levels

of these markers are changed regularly. This could be attributed to impairment of proteins synthesis, chronic inflammation, iron storage mechanisms alterations, and hepatocellular injury¹⁰.

Ferritin, an essential protein in the liver, has a main role in iron storage¹¹. Meanwhile, transferrin, a glycoprotein, synthesizes in the liver and transports iron in the blood¹². Consequently, the serum level of TIBC, the iron binding-capacity of transferrin, is also altered¹³. These variations may affect significantly the serum level of TS, which is the percentage of the serum iron level divided by TIBC, that illustrate the transferrin binding to iron quantity¹⁴.

Although many studies have examined the role of iron markers in liver diseases, the impact of these markers in LC patients still inconspicuous. This research aims to explore the relationship between iron markers levels in the blood of LC patients in comparison with healthy controls in Basra Province - Iraq.

METHODS

Participants Recruitment and Study Design: This study, a case-control clinical study which carried out from November 2025 to April 2026, included patients enrolled in The Gastroenterology Hospital in the Province of Basra, Iraq. LC patients were diagnosed using Child-Pugh Scores criteria by clinicians based on the clinical, laboratory and

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ultrasonography findings^{15,16}. In our study, we focused on LC which caused by metabolic factors such as overweight and lifestyle. The Ethics and Behavioral Research Committee (Scientific Committee) of the Department of Basic Sciences – College of Nursing – University of Basrah – Province of Basra – Iraq (No. 7/54/836 in 30/11/2025) has been confirmed the protocol of this study which was accomplish with Declaration of Helsinki acquiescence. All participants took a clarification for the procedures and signed the informed consent forms. This research required a minimum of 50 LC patients. The number of samples (n) was established through a calculation of the proportion of the population, taking into account the following assumptions: a prevalence of LC of 2.11%, a confidence level of 95%, and a margin of error of 4% (representing absolute precision)¹⁷. The LC patients who were 30–65 years old (class B of Child-Pugh Scores as per the clinician's diagnosis) were shared in this study. LC patients who experienced from inflammatory disorders, chronic kidney dysfunction, vitamin/iron deficiencies, iron supplementation, hemochromatosis, viral hepatitis (B or C), transfusion of red blood cell, and malicious disorders were excluded^{18,19}. The healthy controls aged 30–65 years without LC. The demographic data were obtained by a structured interview conducted when the participants were being visited using a typical self-managed questionnaire. All participants had stable clinical conditions for the last three months.

Samples Collection: Blood samples were gathered from participants after fasting for 12-hours and 30-minutes of resting in the morning time between 08:00 AM and 9:00 AM. Eight mL of venous blood was collected from the participants and moved to a gel tube. It was clotted for 10-minutes. Then, it was rotated for 20 minutes at 402 x g for serum collecting. The serums were directly used for determination of the research markers²⁰.

Measurements of SI, Transferrin, and TIBC by Automated Analyzer: Laboratory kits from Biolabo SAS, Maizy, France were used to assess the serum levels of iron (Cat. No.: K2108, sensitivity: 10 µg/dL), transferrin (Cat. No.: K4208, sensitivity: 50 mg/dL), and TIBC (Cat. No.: 92308, sensitivity: 42 µg/dL) by Kenza Analyzer (240TX, Maizy, France). The sample is automatically mixed with specific reagents, and a chemical reaction is produced that forms a colored complex. The absorbance of light at specific wavelengths (typically 190 to 900 nm) is measured, and the concentration of the marker is calculated²¹.

Table 1. The demographic characteristics of the present study

The Characteristics		LC Patients	Healthy Controls
Total Participants No.		50	75
Age (Years) (Mean ± SD)		49.17 ± 12.72	48.28 ± 14.81
BMI (kg/m ²)		27.54 ± 3.28	28.06 ± 3.61
Duration of LC (Years)		6.09 ± 3.47	0
Demographic Area	Urban	41	68
	Rural	9	7
Educational Background	Learned	44	67
	Illiterate	6	8
Smoking Habits	Smokers	0	0
	Non-Smokers	50	75
Alcohol Consuming	Consumers	0	0
	Non-Consumers	50	75
Food Habits	Vegetarian	13	11
	Non-Vegetarian	37	64
Employment Status	Employed	43	66
	Not Employed	7	9
Participants Gender	Men	24	35
	Women	26	40

Measurement of Ferritin by Enzyme Linked Immunoabsorbance Assay (ELISA): Laboratory kit from BT Laboratory, Shanghai, China was utilized to measure the level of ferritin (Cat. No.: E6766Hu, sensitivity: 0.33 ng/mL) by enzyme-linked immunosorbent assay (ELISA) (Humareader/Human/Germany). The procedure was applied based on the protocol of kit. The plate was read by an ELISA reader (Thermo Fisher Scientific) with 450 nm absorbance. Ferritin levels were assayed plotting a standard curve²².

Measurement of BMI and TS: Body mass index (BMI) was measured by the standard BMI equation²³:

$$\text{BMI (kg/m}^2\text{)} = \text{weight (kg)/height}^2 \text{ (m)}$$

While, TS was measured by dividing SI by TIBC²⁴:

$$\text{TS\%} = (\text{SI} / \text{TIBC}) \times 100\%$$

Statistical Analysis: The statistical analysis was performed by Statistical Package for the Social Sciences (SPSS) version 31 (IBM Corporation, Armonk, NY, USA). Data was normally distributed, and the statistical significance was computed. The scatter plots were drawn and Pearson correlation was applied for constructing the correlations. A value of $p < 0.05$ was referred to statistically significant, while a value of $p < 0.01$ was referred to highly significant²⁵. The 95% confidence interval, specificities, and sensitivities were computed via the receiver operating characteristics (ROC) curve which was created by plotting 1-specificity (x-axis) against sensitivity (y-axis) and determining the area under the curve (AUC). An AUC value close to 1 (or 0) suggested a high diagnostic value. Moreover, binary logistic regression analysis was utilized²⁶.

RESULTS

Participants Characteristics

One hundred and sixty-five (165) participants were participated. Forty participants were excluded as they could not correspond the criteria of inclusion and exclusion. Only 50 LC patients were shared in this research and matched with 75 healthy controls. As demonstrated in Table 1, no significant differences were observed ($p > 0.05$) in age (49.17 ± 12.72 vs. 48.28 ± 14.81 years), gender ratio (men/women) (24/26 vs. 35/40), and BMI (27.54 ± 3.28 vs. 28.06 ± 3.61 kg/m²) of LC patients and healthy controls. Additionally, the data indicated that LC patients had a highly significant decrease ($p < 0.01$) in SI, transferrin, and TIBC

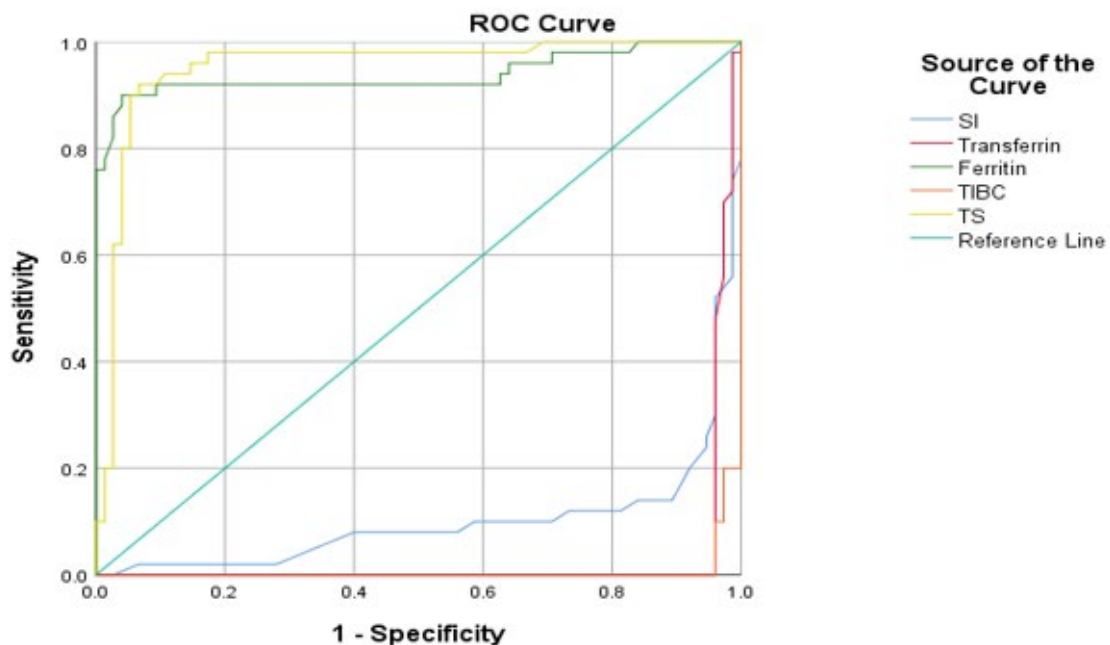
Table 2. Serum levels of markers in LC patients and healthy controls

Biomarker	LC Patients			95% CI		Healthy Controls
	Mean $\pm$ SD	SE	Range	Lower	Upper	Mean $\pm$ SD
Ferritin (ng/mL)	374.06 $\pm$ 111.03**	15.70	62.59-562.18	342.51	405.61	138.83 $\pm$ 70.11
TS (%)	39.87 $\pm$ 12.12**	1.71	23.45-79.57	36.43	43.32	22.72 $\pm$ 6.24
SI (μ g/dL)	71.38 $\pm$ 15.15**	2.14	38.25-110.36	67.07	75.69	94.65 $\pm$ 11.13
Transferrin (mg/dL)	96.26 $\pm$ 27.36**	3.91	58.73-171.12	88.41	104.11	277.96 $\pm$ 65.80
TIBC (μ g/dL)	186.22 $\pm$ 37.21**	5.26	85.76-299.42	175.65	196.79	476.40 $\pm$ 80.44

Table 3. Correlations coefficient of Ferritin and TS with SI, Transferrin, and TIBC in LC patients.

Markers	TS (%)	Ferritin (ng/mL)	SI (μ g/dL)	Transferrin (mg/dL)	TIBC (μ g/dL)
Ferritin (ng/mL)	0.757**	-	0.618**	-0.946**	-0.454**
TS (%)	-	0.757**	0.595**	-0.807**	-0.652**

Tested with Pearson correlations. * ($p < 0.05$): Significant; ** ($p < 0.01$): High-significant.

**Figure 1.** The ROC curve for SI, Transferrin, Ferritin, TIBC, and TS in LC and healthy control patients.

levels compared to controls as shown in Table 2. Furthermore, AUC data, as illustrated in Figure 1, explained that SI, transferrin, and TIBC could be delicate markers in LC patients (AUC = 0.100, 0.029, and 0.007, respectively). Moreover, the binary logistic regression analysis data demonstrated that SI, transferrin, and TIBC had high-significant decreases ($p=0.000$, $p=0.009$ and $p=0.006$, respectively) in comparison with the controls which could be used as tolerable markers in LC patients. In addition, multicollinearity was assessed using variance inflation factor (VIF) and tolerance values. The VIF for ferritin and TS was 2.914, and for transferrin and TIBC was 2.635 indicating low multicollinearity and acceptable independence from other variables. From the other hand, Normality was assessed using the Shapiro–Wilk test and visual inspection of histograms and Q–Q plots. Data were considered normally distributed ($p > 0.05$).

Data are presented as mean $\pm$ standard deviation (SD); SE: Standard Error; Range: is the difference between the highest and lowest values in the set; 95% CI: Confidence Intervals (Lower and Upper); SI: Serum Iron; TIBC: Total Iron Binding Capacity; TS: Transferrin Saturation;

Significance level is indicated by *, Where: $p > 0.05$: p-value not significant, * indicates $p < 0.05$ (p-value significant); ** indicates $p < 0.01$ (p-value highly significant), in comparison with the corresponding control value.

Correlation of Ferritin with Other Markers

The ferritin level in LC patients had a high-significant elevation ($p < 0.01$) in comparison to the controls as shown in Table 2. Furthermore, AUC data, as demonstrated in Figure 1, detected that ferritin could be vigorous marker in LC patients (AUC = 0.938). In addition, the correlations of ferritin with TS and SI was high-significant ($p < 0.01$) positive ($r = 0.757$, $r = 0.618$, respectively), and high-significant ($p < 0.01$) negative with transferrin and TIBC ($r = -0.946$, and $r = -0.454$, respectively), as elucidated in Figure 2. Moreover, the binary logistic regression analysis explicated that ferritin had a high-significant increase ($p=0.002$, OR = 1.016, 95% CI = 1.008 – 1.024) in comparing to the controls which suggesting that it may be used as a marker in LC patients.

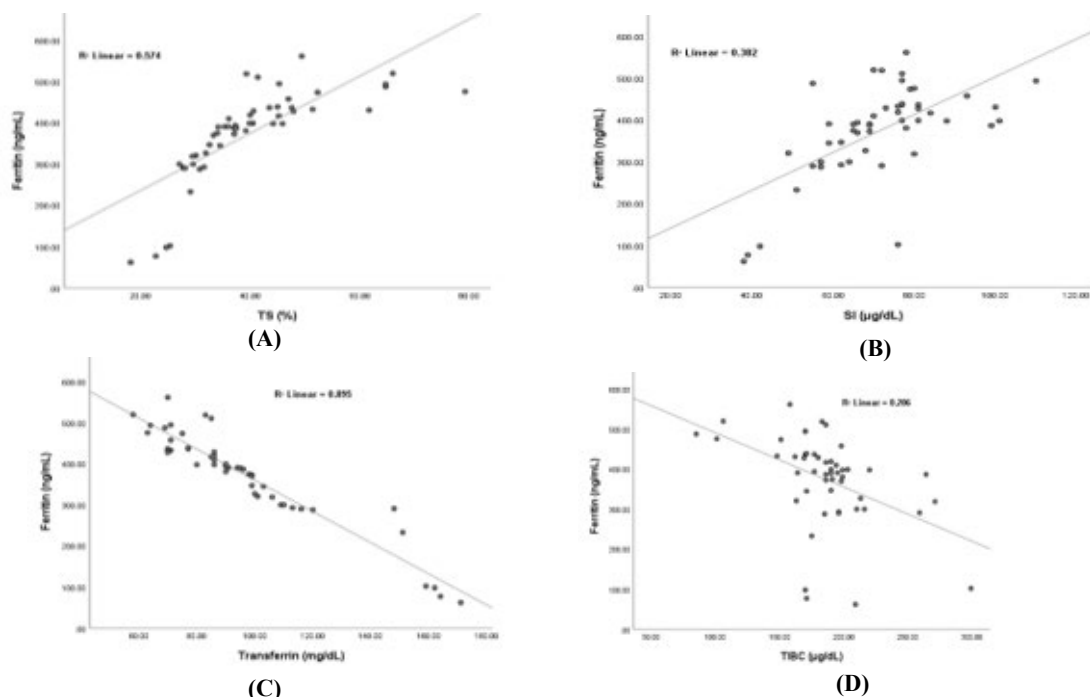


Figure 2. The scatter plot of Ferritin with (A) TS, (B) SI, (C) Transferrin, and (D) TIBC in LC patients.

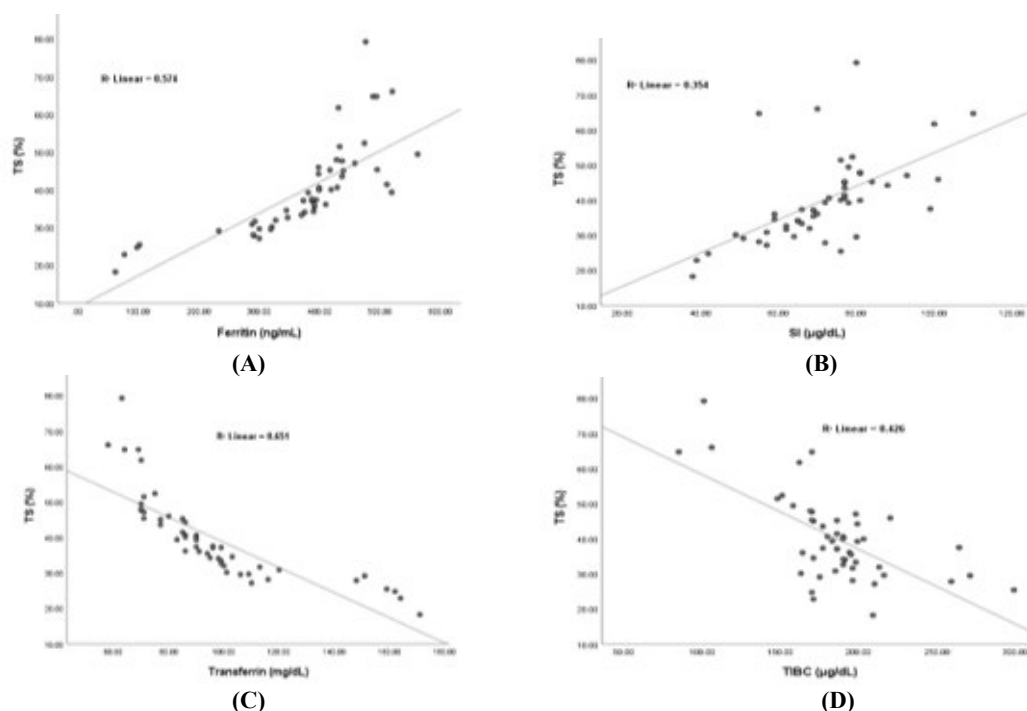


Figure 3. The scatter plot of TS with (A) Ferritin, (B) SI, (C) Transferrin, and (D) TIBC in LC patients.

Correlation of TS with Other Markers

The TS level in LC patients had a high-significant increasing ($p < 0.01$) in comparison to the controls as shown in Table 2. Furthermore, AUC data, as demonstrated in Figure 1, detected that TS could be powerful marker in LC patients (AUC = 0.952). In addition, the correlations of TS with ferritin and SI was high-significant ($p < 0.01$) positive ($r = 0.757$, $r = 0.595$, respectively), and high-significant ($p < 0.01$) negative with transferrin and TIBC ($r = -0.807$, and $r = -0.652$, respectively), as elucidated in Figure 3. Moreover, the binary logistic regression analysis explicated that TS had a high-significant increase ($p = 0.004$, OR = 1.131, 95% CI = 1.034 – 1.238) in comparing to the controls which suggesting that it may be used as a marker in LC patients.

DISCUSSION

In this research, the data showed that all participants were non-consumers for alcohol, non-smokers, most of them from urban, most of them had an education level, and most of them had a good work place²⁷. The genetics, environment, pollution, and variations in food habits are the main variations between urban and rural places people which are increasing in urban regions greatly²⁸.

The results of this study indicated a high-significant positive correlation between ferritin and TS with SI, and high-significant negative correlations with transferrin and TIBC. These findings are in-line with many studies that explored the correlation of ferritin and TS with SI,

transferrin, and TIBC in LC patients^{29,30}. In LC patients, body iron stores increase, ferritin levels increase and more iron may appear in circulation leads to SI increases and decreasing in transferrin and TIBC levels, and consequently, increasing in TS levels³¹. The elevation of ferritin in LC patients may be due to hepatocyte damage, iron overload, and impaired iron metabolism³². Ferritin is stored mainly in liver cells (hepatocytes). However, hepatocyte injury and cell destruction occur in LC patients³³, leading to the release of ferritin into the bloodstream, which increases serum ferritin levels and decreases transferrin levels because its synthesis occurs in the liver, and consequently, TIBC is decreased and TS is increased^{34,35}. Moreover, LC patients may develop hepatic iron accumulation because of intestinal iron absorption increasing³⁶. Therefore, excess iron is stored as ferritin, raising serum levels³⁷. Furthermore, the liver regulates iron metabolism through hepcidin production³⁸. In LC patients, hepcidin regulation becomes abnormal, leading to disturbed iron homeostasis and increased ferritin levels³⁹. Besides, the liver has a key role in ferritin clearance and metabolism⁴⁰. In LC patients, liver dysfunction leads to reduced clearance, causing accumulation of ferritin in the blood⁴¹. Also, LC patients could develop hypersplenism due to portal hypertension⁴². This can cause increased destruction of red blood cells, releasing iron that is stored as ferritin^{43,44}. From the other hand, LC increases oxidative stress⁴⁵, which stimulates ferritin synthesis as a protective mechanism to bind free iron and reduce cellular damage⁴⁶.

The current research was retrospective and cross-sectional in lieu of draw attention to the causals. Furthermore, the LC patients and controls data, in the Province of Basra – Iraq, might not be represent accurately the real state because of the small sample size of the research. Nevertheless, the current research findings may be utilized to develop therapeutic strategies for the diagnosis, alleviation, and management of LC in Province of Basra, Iraq. Further studies with considerable population with different stages of Child-Pugh Scores might be requisite for advocating iron markers roles of in LC, the putative molecular mechanisms at play, the mechanisms regulating the pathogenic process, the regulatory network, the response of patients to treatment, and the relationship between the levels of specific markers and treatment outcomes including those with hepatitis and liver cancer.

CONCLUSION

The correlations of ferritin and TS with SI, transferrin, and TIBC and the high positive value of AUC for ferritin and TS shows that ferritin and TS could be potential markers for the onset of liver failure in LC patients. Furthermore, the biochemical determination of these markers can assist in understanding the pathogenic mechanisms, hepatic dysfunction, and liver cancer in LC patients.

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Competing Interest: None

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REFERENCE

- Sharma R, Zhao W, Zafar Y, et al. Serum hepcidin levels in chronic liver disease: a systematic review and meta-analysis. *Clin Chem Lab Med* 2024;62(3):373–84.
- Al-Hulfi RA, Salem BAMA, Al-Naiema IM. The Effect of Air Pollutants on liver Enzymes and Pituitary Gland Hormones of Smokers and non-smokers of Oil Refinery and Gas Station Workers in Basra/Iraq. *Indian Journal of Forensic Medicine and Toxicology* 2022;16(3):297-301.
- Al-Abedy NMM, Salman ED, Faraj SA. Evaluation of serum hepcidin level in Iraqi patients with chronic hepatitis C and relation to serum iron status. *Indian J Forensic Med Toxicol* 2021;15:1425–30.
- Ali M, Sayed A, Elhawary M, et al. Serum hepcidin in patients with chronic hepatitis C and its relation to treatment with antiviral drugs. *Al-Azhar Int Med J* 2020;1:4.
- Amer A, Aboelmagd Y, Sami M. Study of hepcidin level in Egyptian HCV infected patients and its correlation to ferritin. *Plant Arch* 2020;20:2105–9.
- Gill M, Sharafat J, Ikram F, et al. Correlation between serum ferritin and hepcidin levels in chronic hepatitis C patients. *Cureus* 2021;13:e17484.
- Wahab SEA, Ibrahim IM, El-Gebaly AM, et al. Serum hepcidin level in different stages of hepatitis C induced chronic liver disease. *Egypt J Hosp Med* 2020;81:1634–8.
- Varghese J, Varghese James J, Karthikeyan M, et al. Iron homeostasis is dysregulated, but the iron-hepcidin axis is functional, in chronic liver disease. *J Trace Elem Med Biol* 2020;58:126442.
- Hachim NS, Ali DS, Matoq MA. The role of serotonin, ferritin, and hematological profiles in female pattern hair loss. *Romanian Journal of Medical Practice* 2025;20(3):270–6.
- Cam H, Yilmaz N. Serum hepcidin levels are related to serum markers for iron metabolism and fibrosis stage in patients with chronic hepatitis B: a cross-sectional study. *Arab Journal Gastroenterol* 2020;21:85–90.
- Hasan Y, Brown K. Viral eradication restores normal iron status in chronic hepatitis C patients with abnormal iron studies. *Ann Hepatol* 2020;19:422–6.
- Bloomer SA, Brown KE. Hepcidin and iron metabolism in experimental liver injury. *Am J Pathol* 2021;191:1165–79.
- Lei JJ, Li S, Dong BX, et al. Advances in the treatment of acute hepatic porphyria[J]. *J Clin Hepatol* 2024, 40(4):828-33.
- Linenberger M, Fertrin KY. Updates on the diagnosis and management of the most common hereditary porphyrias: AIP and EPP. *Hematology* 2020 ;2020(1):400–10.
- Bloomer SA, Brown KE. Iron-Induced Liver Injury: A Critical Reappraisal. *Int J Mol Sci* 2019;20:2132.
- Mehta KJ, Farnaud SJ, Sharp PA. Iron and liver fibrosis: Mechanistic and clinical aspects. *World J Gastroenterol* 2019;25:521-38.
- Hashim MF, Sabah FS, Abbas HJ. Study the Association of Fat Mass and Obesity-associated (FTO) Gene Polymorphisms (SNP rs9939609) with Biochemical Markers in Obese Iraqi Patients. *Iraqi Journal of Science* 2024;65(2):684–92.
- Shander A, Muñoz M, Spahn DR. Intravenous iron for severe iron deficiency anaemia. *Br J Anaesth* 2018;121:342-4.
- Abd-Alqader SM, Zearah SA, Al-Assadi IJ. Studying the Effect of Curcumin (Standard & Supplements) and Zinc on the Concentrations of Glucose, Insulin, HOMA-IR, and Anti-Mullerian Hormone in PCOS-Model Rats. *Baghdad Sci.J.* 2024;21(2):0428.
- Alsalmi SA, Al-Fartosy AJM. Association of Serum Programmed Cell Death Protein 1 (PD-1) and Gene Polymorphism with Some Valid Predictors for Systemic Lupus Erythematosus (SLE) Patients in Basra Province, Iraq. *IMJM* 2024;23(3):46-54.
- Zaid MH, Al-Fartusie FS, Pazhang Y, et al. Evaluation of lipid profile, liver function enzymes, and trace elements in Iraqi diabetic nephropathy patients. *Biometals* 37 2024;1565–174.

22. Alsalmi SA, Mashkor IMAA, Al-Fartosy AJM. Osteoprotegerin and Interleukin-37 are Correlated with Liver Diseases in Chronic Hepatitis B Virus (HBV)-infected Subjects. *Indones Biomed. J* 2023;15(3):222-30.
23. Al-Naseem A, Sallam A, Choudhury S, et al. Iron deficiency without anaemia: a diagnosis that matters. *Clinical Medicine* 2021;21(2):107-13.
24. AlAli ZSA, Al Salem BAMAA. The effects of glucose 6-phosphate dehydrogenase deficiency on non-enzymatic antioxidants and kidney function in children in the Basra Governorate, Iraq. *New Zealand Journal of Medical Laboratory Science* 2025;79(1):13-6.
25. Al-Atbi HS, Jaraf HA, Sabti AB. Synthesis, Antibacterial, and Antioxidant Activities of Some Schiff Bases. *Trop J Nat Prod Res* 2022;6(12):1925-9.
26. Soppi E. Iron deficiency without anemia – common, important, neglected. *Clin Case Rep* 2019;5:2-7.
27. Yousif S, Tariq I, Jameel FA. Iron deficiency state prediction in childbearing-age women based on routine laboratory tools: A uni-centre study. *Bahrain Medical Bulletin* 2024;46(2):2055-58.
28. van der Meer P, van der Wal HH, Melenovsky V. Mitochondrial function, skeletal muscle metabolism, and iron deficiency in heart failure. *Circulation* 2019;139:2399-402.
29. Georgieff MK. Iron deficiency in pregnancy. *Am J Obstet Gynecol* 2020;223:516-24.
30. Muñoz M, Gómez-Ramírez S, Besser M, et al. Current misconceptions in diagnosis and management of iron deficiency. *Blood Transfus* 2019;17:191-5.
31. Ati MH, Al-Fartosy AJM, Saleh BA. Synthesis, Biological Evaluation, Molecular Docking, and DFT Analysis of Potential Bioactive Azo-Imines. *Chemistryselect* 2025;10(36):e02660.
32. von Haehling S, Ebner N, Evertz R, et al. Iron deficiency in heart failure. *JACC Heart Fail* 2019;7:36-46.
33. Abdulrehman J, Tang GH, Auerbach M, et al. The safety and efficacy of ferumoxytol in the treatment of iron deficiency: a systematic review and meta-analysis. *Transfusion* 2019;59:3646-56.
34. Scheiner B, Semmler G, Maurer F, et al. Prevalence of and risk factors for anaemia in patients with advanced chronic liver disease. *Liver Int* 2020;40:194-204.
35. Mohammed IM, Alsalmi SA, Al-Fartosy AJM. Trace Elements and Oxidant/Antioxidant Status in Beta-Thalassemia Patients. *Bahrain Med Bull* 2023;45(4):1772-8.
36. Fierro-Fine A, Guerin L, Hicsasmaz H, et al. Clinical Factors Associated with Hepatocellular Iron Deposition in End-stage Liver Disease. *J Clin Transl Hepatol* 2020;8(3):231-9.
37. Mohammed IM, Al. Mashkor IMA, Alsalmi SA, et al. Circulating Serum Caveolin-1 and Interleukin-37 as Predictive Biomarkers in Rheumatoid Arthritis Patients. *J Nat Sc Biol Med* 2024;15:65-72.
38. Faris ST, Al-Fartosy AJM, Al-Fregi AA. Interleukin-37 and Interleukin-18 as Prognostic Biomarkers for End-Stage Renal Disease. *Azerbaijan Medical Journal* 2022;62(4):1429-39.
39. Ibrahim AJ, AL-Saeed AHM. Evaluation of some heart enzymes and Iron levels in β -thalassemia patients in Thi-Qar City, Iraq. *Baghdad Sci.J.* 2023;21(6):10.
40. Rössler J, Schoenrath F, Seifert B, et al. Iron deficiency is associated with higher mortality in patients undergoing cardiac surgery: a prospective study. *Br J Anaesth* 2020;124:25-34.
41. Abbas OM, Khalifa KAE, Makhlof MM, et al. Influence of esophageal variceal bleeding on iron status in chronic hepatitis C patients. *Eur J Gastroenterol Hepatol* 2020;32:616-22.
42. Giannini C, Polidori N, Saltarelli MA, et al. Increased hepcidin levels and non-alcoholic fatty liver disease in obese prepubertal children: a further piece to the complex puzzle of metabolic derangements. *J Pediatr Endocrinol Metab* 2022;35:39-47.
43. Camaschella C. Iron deficiency. *Blood* 2019;133:30-9.
44. Auerbach M, Gafter-Gvili A, Macdougall IC. Intravenous iron: a framework for changing the management of iron deficiency. *Lancet Haematol* 2020;7:e342-50.
45. Nikolaou M, Chrysoshoou C, Georgilas TA, et al. Management of iron deficiency in chronic heart failure: Practical considerations for clinical use and future directions. *Eur J Int Med* 2019;65:17-25.
46. Spahn DR, Schoenrath F, Spahn GH, et al. Effect of ultra-short-term treatment of patients with iron deficiency or anaemia undergoing cardiac surgery: a prospective randomised trial. *Lancet* 2019;393:2201-12.