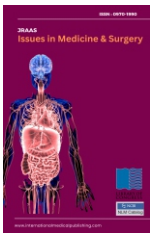
JRAAS
Special Issue in Medicine & Surgery
www.internationalmedicalpublishing.com



Research Article

Section: Medicine

Correlation Between Hyperferritinemia & Microvascular Complications of Type 2 Diabetes Mellitus

Vishal Bohra[1] & Abhiram Chandra Gupta[1*]

[1]Department of General Medicine, Indira Gandhi Employees State Insurance Corporation Hospital, Jhilmil, Delhi, India

HIGHLIGHTS

- Ferritin-linked complications
- Higher in retinopathy nephropathy
- Weak BP correlation
- No glycemic association
- Potential complication marker

Key Words:
Type 2 diabetes mellitus
Serum ferritin
Diabetic retinopathy
Diabetic nephropathy
Microvascular complications
Hyperferritinemia

ABSTRACT

Introduction: Diabetes mellitus is a major metabolic disorder associated with microvascular complications such as retinopathy and nephropathy. Emerging evidence suggests that elevated serum ferritin, a marker of iron stores and inflammation, may play a role in the pathogenesis of these complications. **Aim & Objective:** To assess serum ferritin levels in patients with type 2 diabetes mellitus (T2DM) and evaluate its association with microvascular complications. **Materials & Methods:** This observational cross-sectional study was conducted over 18 months in the Department of General Medicine, IG ESIC Hospital, Jhilmil, Delhi. A total of 351 patients with T2DM were included based on predefined inclusion and exclusion criteria. After obtaining informed consent, detailed clinical history, examination, and investigations including serum ferritin, urine albumin–creatinine ratio, and fundoscopy were performed. Data was analyzed using SPSS, with appropriate statistical tests applied. A p-value <0.05 was considered significant. **Results:** A total of 351 patients with T2DM were included, with a mean age of 56.84±7.29 years and a male predominance (57.1%). Over half of the participants were overweight (52%). The prevalence of diabetic retinopathy and nephropathy was 23.7% and 22.6%, respectively. The mean serum ferritin level was 376.70±94.56 mcg/L. Significantly higher ferritin levels were observed in patients with diabetic retinopathy (416.93±106.15 vs 364.19±87.19 mcg/L; p=0.001) and nephropathy (400.53±112.68 vs 369.75±87.64 mcg/L; p=0.01). No significant association was found between serum ferritin and HbA1c levels (p=0.26). However, weak but statistically significant positive correlations were noted between ferritin and systolic blood pressure (r=0.12), diastolic blood pressure (r=0.11), platelet count (r=0.24), and urea levels (r=0.12). **Conclusion:** Hyperferritinemia is common in patients with T2DM and shows a weak association with diabetic retinopathy and nephropathy. Serum ferritin may serve as a potential marker for microvascular complications, although its relationship with glycemic control remains unclear.

*** Corresponding Author**: Vishal Bohra & Abhiram Chandra Gupta, e-mail: vishal.bohra2906@gmail.com
Article History: Received 21 Februray 2026; Received in Revised form 27 March 2026; Accepted 03 April 2026
How To Cite: Vishal Bohra & Abhiram Chandra Gupta. Correlation Between Hyperferritinemia & Microvascular Complications of Type 2 Diabetes Mellitus. *JRAAS : Special Issue in Medicine & Surgery.* 2026;41(1):1-10. https://doi.org/10.71393/ray7v284

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistently elevated blood glucose levels resulting from defects in insulin secretion, insulin action, or both. It encompasses several categories, including type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM), maturity-onset diabetes of the young (MODY), gestational diabetes, neonatal diabetes, and secondary forms associated with endocrinopathies or drug use such as corticosteroids. Among these, T1DM and T2DM are the most common subtypes, with T1DM primarily resulting from autoimmune destruction of pancreatic β-cells leading to absolute insulin deficiency, and T2DM arising from insulin resistance combined with relative insulin deficiency. T1DM is typically presents in childhood or adolescence, whereas T2DM is more commonly seen in middle-aged and older adults, largely due to prolonged exposure to unhealthy lifestyle and dietary habits.

Diabetes mellitus represents one of the most prevalent metabolic diseases globally and poses a major public health challenge. The burden of diabetes has been rising rapidly, particularly in low- and middle-income countries, driven by urbanization, lifestyle modifications, and increasing rates of obesity and physical inactivity [1]. According to global estimates, approximately 422 million people are affected by diabetes worldwide, with the majority residing in developing regions. Notably, nearly 90% of these cases are attributed to T2DM. The prevalence of T1DM, although lower, has also been increasing globally, with reported annual increases of 2–5% in regions such as Europe, the Middle East, and Australia [2–7]. In the United States, incidence rates have shown a steady rise of approximately 2% annually across most age groups and ethnicities [8], although some data suggest a plateau in incidence during certain periods [9].

Future projections indicate a substantial rise in global diabetes prevalence, with an estimated 578 million individuals affected by 2030 and nearly 700 million by 2045. Diabetes-related complications contribute significantly to morbidity and mortality, with over four million deaths annually reported among individuals aged 20–79 years [10].

Chronic hyperglycemia in diabetes leads to a wide spectrum of complications, broadly categorized into microvascular and macrovascular complications. Microvascular complications, including diabetic nephropathy, neuropathy, and retinopathy, arise from structural and functional changes in small blood vessels and tend to develop earlier and more frequently than macrovascular complications [11]. However, current data on trends and emerging patterns of these complications remain limited, making it difficult to draw definitive conclusions regarding their progression [11]. The rising prevalence of T2DM has been accompanied by a proportional increase in microvascular complications. Persistent hyperglycemia, along with genetic susceptibility, renders the microvasculature highly vulnerable to damage, affecting vital organs such as the kidneys, eyes, and peripheral nerves. Diabetic retinopathy is a leading cause of blindness, diabetic neuropathy contributes significantly to foot ulcers and amputations, and diabetic nephropathy remains a major cause of chronic kidney disease [12].

Serum ferritin, a marker of total body iron stores, has gained attention in recent years for its potential role in the pathophysiology of T2DM and its complications. Elevated serum ferritin levels indicate increased iron stores and are associated with enhanced production of reactive oxygen species, leading to oxidative stress [13,14]. Iron-induced oxidative stress has been implicated in the development of several clinical conditions, including T2DM, cardiovascular diseases, and malignancies

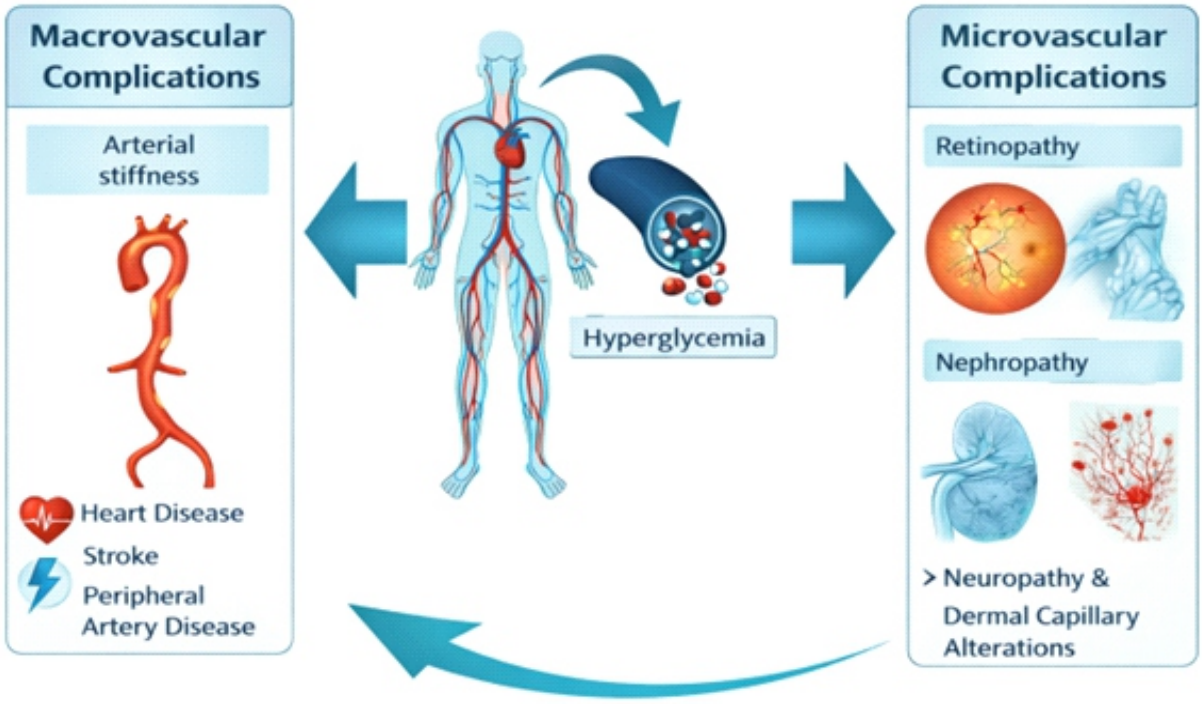


Figure 1: Hyperferritinemia correlates with microvascular complications in type 2 diabetes mellitus. Adopted from [36]

[15,16]. Additionally, serum ferritin acts as an acute-phase reactant and reflects systemic inflammation, with hyperferritinemia being associated with both pro-inflammatory and immunosuppressive states [17].

Oxidative stress and chronic inflammation are key mechanisms underlying the development and progression of T2DM and its complications. Despite growing interest, the relationship between serum ferritin levels and diabetic complications remains controversial. Some studies have reported a protective association, suggesting that higher ferritin levels may be linked to reduced cardiovascular risk in patients with T2DM [18,19]. In contrast, other studies have demonstrated a positive correlation between elevated serum ferritin levels and the risk and severity of coronary artery disease [20]. Furthermore, increased serum ferritin has been significantly associated with microvascular complications such as diabetic retinopathy, neuropathy, and nephropathy [20,21].

Given these conflicting findings and the limited data available, further research is warranted to better understand the role of serum ferritin in T2DM and its complications. Evaluating this association may provide valuable insights into disease mechanisms and help identify potential biomarkers for early detection and risk stratification of diabetic complications.

MATERIALS & METHODS

This observational cross-sectional study was conducted over 18 months in the Medicine OPD of IGESIC Hospital, Delhi, among T2DM patients aged 40–70 years with disease duration >5 years. Patients with T1DM, recent T2DM, major comorbidities, alcoholism, smoking, or relevant drug history were excluded. A total of 351 patients were included (calculated sample size: 350). After informed consent, detailed history, clinical examination, and investigations including serum ferritin, urine albumin creatinine ratio, and fundoscopy were performed. Data were analyzed using SPSS; continuous variables were expressed as mean±SD and compared using t-test/regression, while categorical data were analyzed using chi-square/logistic regression. A p-value <0.05 was considered significant.

RESULT

The present study was conducted in the Department of General Medicine, IG ESIC Hospital Jhilmil, Delhi, to estimate serum ferritin levels in patients with Type 2 Diabetes mellitus. A total of 351 patients were included in the study. **Table 1** shows the distribution of patients according to age.

More than one-third of patients were between 51 and 60 years of age (47.4%), followed by 61-70 (30%) and 40-50 (22.6%). The mean age of patients was 56.84±7.29 years. **Table 2** presents the distribution of patients by gender. More than half of the patients were males (57.1%). **Figure 2** shows the distribution of patients according to BMI. More than half of patients had a BMI of 25.00-29.99 kg/mtr2 (52%), followed by 18.50-24.99 kg/mtr2 (40.6%) and ≥30.00 kg/mtr2 (7.4%). **Figure 3** shows the distribution of patients according to the prevalence of diabetic retinopathy and nephropathy. The prevalence of these conditions was 23.7% and 22.6%, respectively. **Figure 4** shows the distribution of blood pressure. The mean SBP and DBP were 130.52±7.12 and 79.09±2.62, respectively. **Figure 5** shows the distribution of CBC parameters. The mean Hb, TLC and platelet count were 12.85±0.98, 6874.89±1698.11 and 225.42±76.81, respectively. **Figure 6** show the distribution of liver function tests. The mean urea, creatinine and total bilirubin were 27.09±6.98, 0.79±0.15 and 0.74±0.36, respectively. The mean SGOT, SGPT and albumin were 35.05±16.56, 44.33±34.42 and 4.49±0.32, respectively. **Figure 7** shows the distribution of blood sugar. The mean FBS and PPBS were 277.41±52.98 and 314.04±40.90, respectively. **Figure 8** show the distribution of HbA1c among diabetic patients. HbA1c <6.5 was among more than half of patients (69.1%) followed by 7.6-9 (11.4%), 6.5-7.5 (10.9%) and >9 (8.6%). The mean HbA1c was 6.36±1.39. **Table 3** shows the distribution of the lipid profile among diabetic patients. The mean triglyceride, HDL, and LDL were 248.59±47.85, 44.55±7.05, and 141.42±9.77, respectively. **Table 4** compare ferritin levels in T2DM patients with and without diabetic retinopathy. Serum ferritin level was significantly (p=0.001) higher among patients with diabetic retinopathy (416.93±106.15 mcg/l) than among patients without diabetic retinopathy (364.19±87.19 mcg/l). **Table 5** compares ferritin levels in T2DM patients with and without diabetic nephropathy. Serum ferritin level was significantly (p=0.01) higher among patients with diabetic nephropathy (400.53±112.68 mcg/l) than among patients without diabetic nephropathy (369.75±87.64 mcg/l). **Table 6** compares ferritin levels with HbA1c levels. The analysis of variance revealed no significant difference in ferritin levels across HbA1c levels (p> 0.05). The total serum ferritin level was 376.70±94.56 mcg/l. **Table 7** shows the correlation of ferritin levels with various factors. SBP, DBP, platelet count and urea were poorly significantly (p<0.05) positively correlated with serum ferritin

Table 1: Distribution of patients according to age

Age in years	Frequency (n=350)	Percentage (%)
40-50	79	22.6%
51-60	166	47.4%
61-70	105	30%
Mean ± SD	56.84±7.29	

Table 2: Distribution of patients according to gender

Gender	Frequency (n=350)	Percentage (%)
Male	200	57.1
Female	150	42.9

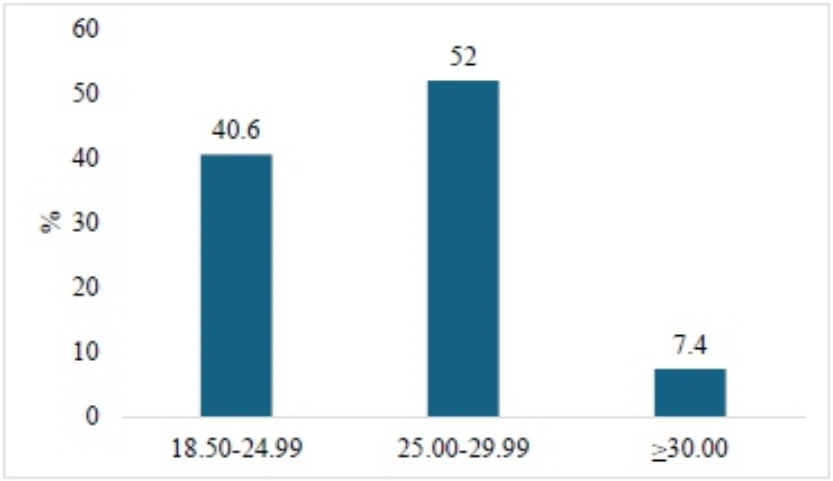


Figure 2: Distribution of patients according to BMI

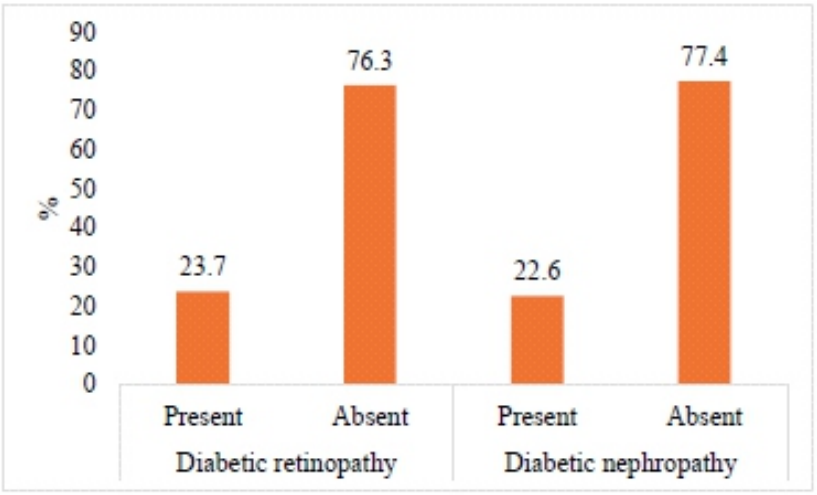


Figure 3: Distribution of Prevalence of diabetic retinopathy and nephropathy

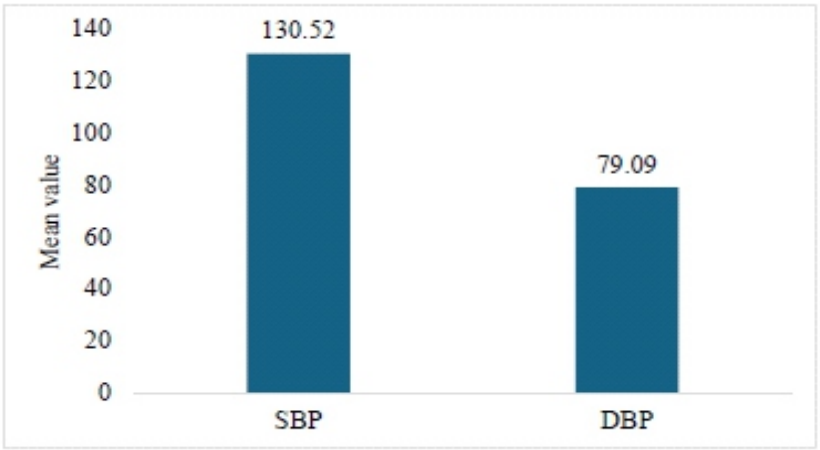


Figure 4: Distribution of blood pressure among diabetic patients

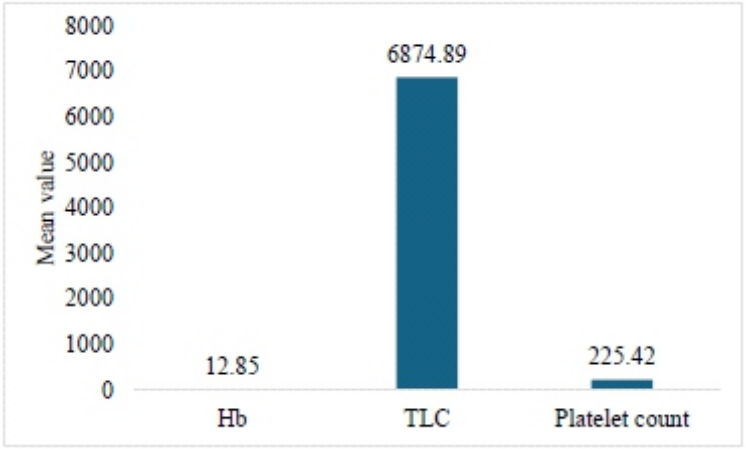


Figure 5: Distribution of CBC parameters among diabetic patients

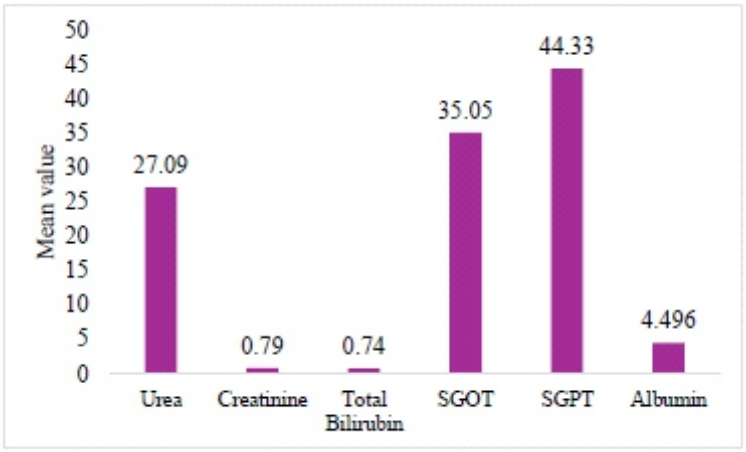


Figure 6: Distribution of liver function tests among diabetic patients

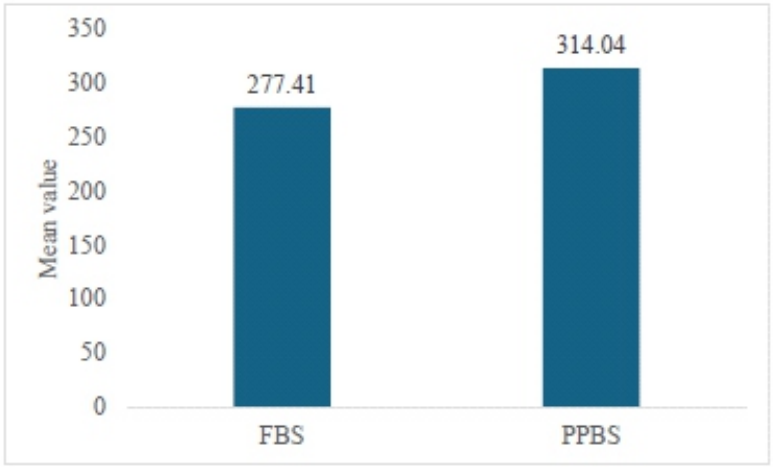


Figure 7: Distribution of blood sugar among diabetic patients

Table 3: Distribution of Lipid profile among diabetic patients

Lipid profile	(Mean ± SD)
Triglyceride	248.59±47.85
HDL	44.55±7.05
LDL	141.42±9.77

Table 4: Comparison of ferritin level in T2DM patients with and without diabetic retinopathy

Diabetic retinopathy	Ferritin level (mcg/l) (Mean±SD)
Present	416.93±106.15
Absent	364.19±87.19
p-value[1]	0.001*

[1]Unpaired t-test, *Significant

Table 5: Comparison of ferritin level in T2DM patients with and without diabetic nephropathy

Diabetic nephropathy	Ferritin level (mcg/l) (Mean ± SD)
Present	400.53±112.68
Absent	369.75±87.64
p-value[1]	0.01*

[1]Unpaired t-test, *Significant

Table 6: Comparison of ferritin level with HbA1c level

HbA1c	Ferritin level (mcg/l) (Mean ± SD)
<6.5	375.93±95.24
6.5-7.5	353.95±94.99
7.6-9	390.25±81.63
>9	393.67±102.83
Total	376.70±94.56
p-value[1]	0.26

[1]ANOVA test

Table 7: Correlation of ferritin level with various factors

Factors	Ferritin level	
	Correlation coefficient	**p-value[1]**
BMI	-0.03	0.51
SBP	0.12	0.01*
DBP	0.11	0.03*
Hb	-0.08	0.09
TLC	-0.05	0.33
Platelet count	0.24	0.001*
Urea	0.12	0.02*
Creatinine	0.07	0.14
Total bilirubin	0.03	0.47
SGOT	-0.03	0.48
SGPT	0.01	0.80
Albumin	-0.01	0.80
FBS	0.05	0.33
PPBS	0.03	0.49
HbA1c	0.06	0.25
Triglyceride	-0.14	0.008*
HDL	0.002	0.97
LDL	-0.05	0.27

[1]Pearson correlation, *Significant

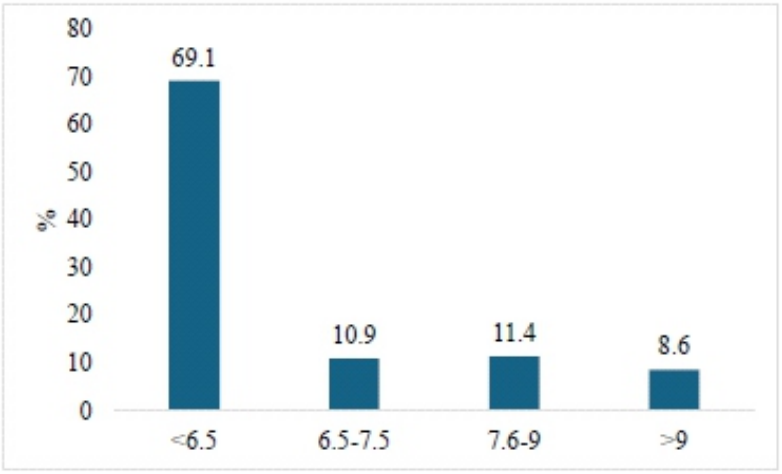


Table 6: Distribution of cases according to clinical outcome. (N = 89)

DISCUSSION

Diabetes mellitus has emerged as a major global health concern due to its wide spectrum of complications. Chronic hyperglycemia plays a key role in the development of these complications, leading to metabolic and hemodynamic disturbances such as increased formation of advanced glycation end products (AGEs), generation of reactive oxygen species (ROS), activation of protein kinase C (PKC), and stimulation of the polyol and renin angiotensin systems. These mechanisms contribute to the structural and functional vascular changes seen in diabetes [29].

Hyperglycemia, oxidative stress, and lipid peroxidation further accelerate the process of advanced glycation, resulting in activation of intracellular signaling pathways and release of proinflammatory and profibrotic cytokines, thereby promoting diabetic vascular complications [30,31]. Additionally, hyperglycemia in diabetes results from increased hepatic glucose production and reduced peripheral glucose utilization, often accompanied by elevated glucagon levels.

Iron metabolism has also been implicated in glucose dysregulation. Even without overt iron overload, increased tissue iron enhances oxidative stress and inflammatory processes. Serum ferritin, an intracellular iron storage protein found in organs such as the liver, spleen, bone marrow, heart, pancreas, and kidneys, serves as a marker of body iron stores and inflammation [34].

The present study was conducted at IG ESIC Hospital, Delhi, to evaluate serum ferritin levels in patients with type 2 diabetes mellitus, including a total of 351 participants. Most patients were aged 51–60 years (47.4%), with a mean age of 56.84±7.29 years, comparable to findings by **Tummalacharla et al**. [28] and **Chawla et al.** [23]. Male predominance (57.1%) was observed, like studies by **Arnold et al.** [22], though contrasting with **Al Argan et al**. [33], where females predominated.

Most patients had a BMI in the overweight range (25–29.99 kg/m²). The prevalence of diabetic retinopathy and nephropathy was 23.7% and 22.6%, respectively, aligning with previous studies reporting similar rates of microvascular complications [22-24].

Several studies, including those by **Al Miraj and Khan** have reported significantly higher ferritin levels in diabetic patients compared to controls [26]. Similarly, **Aruna Kumari et al.** [34] and **Chawla et al.** also observed elevated ferritin levels in diabetes [23].

In this study, serum ferritin levels were significantly higher in patients with diabetic retinopathy and nephropathy, suggesting its association with microvascular complications. Similar observations were reported in other studies, and large population-based research by Kang et al. demonstrated an association between elevated ferritin levels and chronic kidney disease, particularly in men [35].

No significant association was observed between serum ferritin and HbA1c levels in this study, which is consistent with findings by **Al Argan et al.** [33]. However, other studies have reported a positive correlation between ferritin and glycemic parameters, including HbA1c, fasting, and postprandial glucose levels [25-27].

A weak but significant positive correlation was noted between serum ferritin and parameters such as systolic blood pressure, diastolic blood pressure, platelet count, and urea. However, no significant association was found with other variables. In contrast, studies by Vinodkumar et al. and Diwan et al. reported stronger correlations between ferritin and multiple metabolic parameters, including glucose levels, lipid profile, and insulin resistance markers [27].

Overall, the findings suggest that elevated serum ferritin may be associated with diabetic complications, particularly microvascular involvement, though its relationship with glycemic control remains inconsistent across studies.

CONCLUSION

The present study conducted in the Department of General Medicine at IG ESIC Hospital, Jhilmil, Delhi, among patients with type 2 diabetes mellitus demonstrated a high prevalence of elevated serum ferritin levels. A considerable proportion of patients were overweight, and notable occurrences of diabetic retinopathy and nephropathy were observed. More than half of the patients had relatively controlled glycemic status. Serum fe-

rritin levels were significantly higher in patients with diabetic retinopathy and nephropathy, while no significant association was found with HbA1c levels. A weak positive correlation was observed between serum ferritin and blood pressure, platelet count, and urea levels, whereas no association was noted with other parameters. Overall, the study suggests that hyper-ferritinemia is common in T2DM and shows a weak association with microvascular complications such as diabetic retinopathy and nephropathy.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multicentre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic and management approaches. Such efforts will improve overall patient care and help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical healthcare applications. It emphasizes the importance of trans-lating scientific observations into meaningful improve-ments in patient care, diagnosis, and treatment outcomes. By highlighting real-world relevance, the study contributes to evidence-based medical practice and supports informed clinical decision-making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

SF: Serum Ferritin
T2DM: Type 2 Diabetes Mellitus
DR: Diabetic Retinopathy
DN: Diabetic Nephropathy
HbA1c: Glycated Hemoglobin
MC: Microvascular Complications

AUTHOR INFORMATION

Dr. Vishal: Senior Resident
Dr. Abhiram Chandra Gupta: Senior Consultant

AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

ACKNOWLEDGEMENT

The authors sincerely acknowledge the seniors of the Department of General Medicine, Indira Gandhi Employees State Insurance Corporation Hospital, Jhilmil, Delhi, India. We are grateful to our college for providing the necessary resources to carry out this work. We also extend our heartfelt thanks to our colleagues and technical staff for their valuable assistance during the study.

CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

FUNDING

None

ETHICAL APPROVAL & CONSENT TO PARTICIPATE

All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a re-asonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in thewriting or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research directions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

PUBLISHER'S NOTE

All statements made in this article are the sole responsibility of the authors and do not necessarily reflect the views of the publisher, editors, or reviewers. The journal maintains a neutral stance regarding jurisdictional claims in institutional affiliations presented in published work.

ARCHIVING INFORMATION

- zenodo
- Self-archiving on Google and Amazon Web Services (AWS) cloud servers, as well as on three dedicated in-house servers.

REFERENCE

1. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. Nat Rev Endocrinol. 2018;14:88–98. doi:10.1038/nrendo.2017.151
2. Felner EI, Klitz W, Ham M, Lazaro AM, Stastny P, Dupont B, et al. Genetic interaction among three genomic regions creates distinct contributions to early- and late-onset type 1 diabetes mellitus. Pediatr Diabetes. 2005;6:213–220. doi:10.1111/j.1399-5448.2005.00128.x
3. Dabelea D, Bell RA, D'Agostino RB, Imperatore G, Johansen JM, et al. Incidence of diabetes in youth in the United States. JAMA. 2007;297:2716–2724. doi:10.1001/jama.297.24.2716
4. Gale EA, Gillespie KM. Diabetes and gender. Diabetologia. 2001;44:3–15. doi:10.1007/s001250051573
5. Mamoulakis D, Galanakis E, Bicouvarakis S, Paraskakis E, Sbyrakis S. Epidemiology of childhood type 1 diabetes in Crete, 1990–2001. Acta Paediatr. 2003;92:737–739. doi:10.1111/j.1651-2227.2003.tb00606.x
6. Tuomilehto J. The emerging global epidemic of type 1 diabetes. Curr Diab Rep. 2013;13:795–804. doi:10.1007/s11892-013-0433-5
7. Patterson CC, Dahlquist GG, Gyürüs E, Green A, Soltész G. Incidence trends for childhood type 1 diabetes in Europe during 1989–2003 and predicted new cases 2005–20. Lancet. 2009;373:2027–2033. doi:10.1016/S0140-6736(09)60568-7
8. Vehik K, Hamman RF, Lezotte D, Norris JM, Klingensmith G, Bloch C, et al. Increasing incidence of type 1 diabetes in 0–17-year-old Colorado youth. Diabetes Care. 2007;30:503–509. doi:10.2337/dc06-1837
9. Rush T, McGeary M, Sicignano N, Buryk MA. A plateau in new onset type 1 diabetes. Pediatr Diabetes. 2018;19:917–922. doi:10.1111/pedi.12670
10. Saeedi P, Petersohn I, Salpea P, Malanda B, Karuranga S, Unwin N, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045. Diabetes Res Clin Pract. 2019;157:107843. doi:10.1016/j.diabres.2019.107843
11. Harding JL, Pavkov ME, Magliano DJ, Shaw JE, Gregg EW. Global trends in diabetes complications. Diabetologia. 2019;62:3–16. doi:10.1007/s00125-018-4711-2
12. Guariguata L, Whiting DR, Hambleton I, Beagley J, Linnenkamp U, Shaw JE. Global estimates of diabetes prevalence for 2013 and projections for 2035. Diabetes Res Clin Pract. 2014;103:137–149. doi:10.1016/j.diabres.2013.11.002
13. Recalcati S, Gammella E, Buratti P, Cairo G. Molecular regulation of cellular iron balance. IUBMB Life. 2017;69:389–398. doi:10.1002/iub.1628
14. Brissot P, Ropert M, Le Lan C, Loréal O. Non-transferrin bound iron. Biochim Biophys Acta. 2012;1820:403–410. doi:10.1016/j.bbagen.2011.07.014
15. Altamura S, Kopf S, Schmidt J, Müdder K, da Silva AR, Nawroth P, et al. Uncoupled iron homeostasis in type 2 diabetes mellitus. J Mol Med (Berl). 2017;95:1387-1398. doi:10.1007/s00109-017-1575-5
16. Kobayashi M, Suhara T, Baba Y, Kawasaki NK, Higa JK, Matsui T. Pathological roles of iron in cardiovascular disease. Curr Drug Targets. 2018;19:1068–1076. doi:10.2174/1389450118666170602093831
17. Kernan KF, Carcillo JA. Hyperferritinemia and inflammation. Int Immunol. 2017;29:401–409. doi:10.1093/intimm/dxx031
18. Suárez-Ortegón MF, McLachlan S, Price AH, Fernández-Balsells M, Franch-Nadal J, Mata-Cases M, et al. Decreased iron stores and cardiovascular disease in type 2 diabetes. Atherosclerosis. 2018;272:193–199. doi:10.1016/j.atherosclerosis.2018.03.021
19. Hermans MP, Ahn SA, Amoussou-Guenou KD, Balde NM, Rousseau MF. Do high ferritin levels confer lower cardiovascular risk? Diabet Med. 2010;27:417–422. doi:10.1111/j.1464-5491.2010.02956.x
20. Mokhtari H, Bagheri B, Rasouli M. Iron hypothesis and coronary artery disease in geriatric patients. Arch Physiol Biochem. 2020;126:17-22. doi:10.1080/13813455.2018.1437753
21. Ozougwu JC, Obimba KC, Belonwu DC, et al. The pathogenesis and pathophysiology of type 1 and type 2 diabetes mellitus. J Physiol Pathophysiol. 2013;4:46–57.
22. Arnold SV, Khunti K, Tang F, Chen H, Cid-Ruzafa J, Cooper A, et al. Incidence rates and predictors of complications in type 2 diabetes. Am Heart J. 2022;243:232–239. doi:10.1016/j.ahj.2021.10.004
23. Chawla R, Bhoopathi A, Punyani H. Ferritin and serum iron as markers of poor glycemic control. Int J Diabetes Dev C tries. 2019;39:362–368. doi:10.1007/s13410-018-0666-5
24. Faselis C, Katsimardou A, Imprialos K, Deligkaris P, Kallistratos M, Dimitriadis K. Microvascular complications of type 2 diabetes mellitus. Curr Vasc Pharmacol. 2020;18:117-124. doi:10.2174/1570161117666190502103733
25. Kumari TA, Kumar GA, Jyotsna A, Kathyaini R. Serum ferritin, HbA1c, nitric oxide, uric acid in T2DM. Int J Health Clin Res. 2021;4(15):215–218.
26. Al-Miraj AK, Khan MIH. Correlation between serum ferritin and HbA1c in T2DM. J Diabetes Metab. 2021;12:1-3. doi:10.35248/2155-6156.21.12.851
27. Vinodkumar RP, Ravisekar P, Kalaiselvi R. Correlation of serum ferritin with HbA1c. Int J Pharm Clin Res. 2024;16:613-616.

28. Tummalacharla SC, Pavuluri P, Maram SR, Vadakedath S, Kondu D, Karpay S, Kandi V. Serum activities of ferritin among controlled and uncontrolled type 2 diabetes mellitus patients. Cureus. 2022;14(5):1-12. doi:10.7759/cureus.24755
29. Yamagishi S, Nakamura K, Matsui T, Noda Y, Imaizumi T. RAGE as therapeutic target in diabetic complications. Curr Pharm Des. 2008;14:487–495. doi:10.2174/138161208783885735
30. Goh SY, Cooper ME. Advanced glycation end products in diabetes. J Clin Endocrinol Metab. 2008;93:1143–1152. doi:10.1210/jc.2007-1817
31. Ceriello A. Oxidative stress and diabetes complications. Endocr Pract. 2006;12 Suppl 1:60–62. doi:10.4158/EP.12.S1.60
32. Fernández-Real JM, López-Bermejo A, Ricart W. Cross-talk between iron metabolism and diabetes. Diabetes. 2002;51:2348-2354. doi:10.2337/diabetes.51.8.2348
33. Al Argan R, Alkhafaji D, Al Elq A, Albaker W, Elamin Y, Alwaheed A, et al. Ferritin and bilirubin with glycemic control in T2DM. J Med Life. 2023;16:1670–1677. doi:10.25122/jml-2023-0123
34. Petimani MS, Adake P, Babu PS**.** Comparison of plasma glucose, serum ferritin, HbA1c and serum nitric oxide levels between diabetic and non diabetic individuals: an Indian scenario. Int J Biochem Res Rev. 2015;8(3):1–9.
35. Kang HT, Linton JA, Kwon SK, Park BJ, Lee JH. Ferritin and chronic kidney disease. Int J Environ Res Public Health. 2016;13:1–9. doi:10.3390/ijerph13030246
36. Partalidou S, Patoulias D, Pantekidis I, Kefas A, Doumas M, Gkaliagkousi E, Rizzo M, Dimitroulas T, Anyfanti P. The cross-talk between arterial stiffness and microvascular complications in diabetes mellitus: a systematic review of the literature. J Diabetes Metab Disord. 2025;24(2):1-15.