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Study of Serum Iron Profile in Type 2 Diabetes Mellitus Patients in a Tertiary Care Hospital in Ghaziabad

Dr. Nitin Patrick[1], Dr. Juhi Aggarwal[2], Urvashi Midha[3]

[1]M.Sc. Medical Biochemistry, Santosh Medical College & Hospital Santosh Deemed to Be University, Ghaziabad, UP

[2]Professor and Head of Department of Biochemistry, Santosh Medical College & Hospital Santosh Deemed to Be University, Ghaziabad, UP

[3]PhD Scholar, Santosh Medical College & Hospital Santosh Deemed to Be University, Ghaziabad, UP

Abstract: ***Background**: Type 2 Diabetes Mellitus (T2DM) is a major metabolic disorder associated with dysregulated iron metabolism. This study aimed to evaluate and compare the serum iron profile in T2DM patients and healthy controls. **Methods**: A case-control study was conducted on 140 participants (70 T2DM patients and 70 controls). Serum iron, ferritin, total iron-binding capacity (TIBC), unsaturated iron-binding capacity (UIBC), fasting blood sugar (FBS), and HbA1c were estimated. Data were analyzed using SPSS v27, with $p<0.05$ considered statistically significant. **Results**: T2DM patients had significantly higher serum iron (187.0±8.2 µg/dL vs 102.1±23.3 µg/dL) and ferritin (428.7±15.1 ng/mL vs 98.8±12.4 ng/mL) compared to controls ($p<0.001$). TIBC and UIBC were also elevated. BMI and glycemic indices were significantly higher in diabetics. HbA1c positively correlated with serum iron ($r=0.62$, $p<0.001$). **Conclusion**: Elevated serum iron and ferritin levels in T2DM patients suggest a link between iron overload and poor glycemic control. Monitoring iron metabolism may help prevent diabetes-related complications.*

Keywords: Type 2 Diabetes Mellitus, Serum Iron, Ferritin, TIBC, UIBC, Glycemic Control

1. Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder marked by hyperglycemia resulting from insulin resistance and impaired insulin secretion. Iron plays a vital role in glucose metabolism, yet excess body iron may catalyze oxidative reactions leading to β-cell damage and insulin resistance. Studies have shown that increased serum ferritin and iron levels correlate with poor glycemic control and higher risk of diabetic complications.

The global prevalence of Type 2 Diabetes Mellitus (T2DM) has been steadily rising, with India emerging as a major contributor to the diabetic population. Sedentary lifestyle, urbanization, and dietary changes have accelerated the burden of the disease. T2DM accounts for more than 90% of all diabetes cases worldwide and is characterized by impaired insulin action and secretion. In India, the prevalence of diabetes has increased dramatically from 7.1% in 2009 to 8.9% in 2019, and it continues to rise, particularly among middle-aged adults.

Iron, a critical micronutrient, plays a fundamental role in oxygen transport, mitochondrial function, and enzymatic processes. However, excess iron may catalyze the formation of reactive oxygen species (ROS), resulting in oxidative stress that contributes to the dysfunction of pancreatic β-cells and insulin resistance. This mechanism suggests that iron homeostasis is intricately linked to glucose metabolism and the pathophysiology of T2DM. Elevated ferritin levels, often used as a surrogate marker of body iron stores, have been associated with poor glycemic control and increased risk of complications such as nephropathy, neuropathy, and cardiovascular disease.

Recent studies have indicated that individuals with higher iron and ferritin levels tend to exhibit higher fasting glucose and HbA1c concentrations, suggesting a mechanistic link between iron overload and hyperglycemia. Additionally, chronic low-grade inflammation and oxidative stress, both prominent features of T2DM, may further dysregulate iron metabolism. Understanding these biochemical interrelationships may open new avenues for early diagnosis and therapeutic intervention.

2. Materials and Methods

A hospital-based case-control study was conducted in the Department of Biochemistry, Santosh Medical College, Ghaziabad, involving 70 T2DM patients and 70 age- and sex-matched healthy controls. Blood samples were collected after overnight fasting. Serum iron was estimated using the ferrozine method, FBS by GOD-POD, and HbA1c by HPLC. Data were analyzed using SPSS version 27.

3. Results

Table 1: Age wise distribution of study subjects

Years	Control group		Type 2 DM group	
	Frequency	Percent	Frequency	Percent
30-35	9	12.9	6	8.6
36-40	22	31.4	20	28.6
41-45	24	34.3	21	30.0
46-50	8	11.4	12	17.1
51-55	7	10.0	11	15.7
Total	70	100.0	70	100.0

Chi square value-2.58; p value- 0.629

The age distribution of subjects in both groups showed that the majority were between 36–45 years. In the control

group, 31.4% were aged 36–40 years and 34.3% were 41–45 years. Similarly, in the Type 2 DM group, 28.6% were aged 36–40 years and 30% were 41–45 years. Very few participants were in the youngest (30–35 years) and oldest (51–55 years) age categories. The chi-square test value (2.58) with a p-value of 0.629 indicates that there was no statistically significant difference in age distribution between the groups.

Table 2: Mean age among study groups

	Control Group		Type 2 DM group		t value	p value
	Mean	Std. Deviation	Mean	Std. Deviation		
Age	41.64	5.61	43.56	6.08	-1.937	0.055

The mean age in the control group was 41.64 ± 5.61 years, while in the Type 2 DM group it was slightly higher at 43.56 ± 6.08 years. The t-test yielded a value of -1.937 with a p-value of 0.055, suggesting that the difference in mean age between the two groups was not statistically significant.

Table 3: Gender wise distribution of study subjects

	Control Group		Type 2 DM group	
	Frequency	Percent	Frequency	Percent
Female	36	51.4	38	54.3
Male	34	48.6	32	45.7

Chi square value- 0.114; p value- 0.734

Gender distribution was almost equal in both groups. In the control group, females comprised 51.4% and males 48.6%, while in the Type 2 DM group, females made up 54.3% and males 45.7%. The chi-square value (0.114) with a p-value of 0.734 shows no significant gender difference between the groups, indicating that the groups were well-matched for sex.

Table 4: Biochemical parameters in control & type 2 DM groups

	Control Group		Type 2 DM group		t value	p value
	Mean	Std. Deviation	Mean	Std. Deviation		
BMI	23.97±3.00	3.00	28.694	3.5597	-8.489	<0.01*
HbA1c	5.39±0.24	0.24	7.979	.6997	-29.353	<0.01*
FBS	90.14±6.75	6.75	151.179	11.4577	-38.403	<0.01*
PPBS	130.83±5.39	5.39	241.724	16.8997	-52.306	<0.01*
RBC	5.36±0.17	0.17	4.957	.1806	13.564	<0.01*
WBC	7.24±0.86	0.86	8.973	1.1209	-10.253	<0.01*
Hb	13.00±0.60	0.60	13.306	.7671	-2.600	.019
Platelets	268.54±9.69	9.69	254.93	23.367	4.503	<0.01*
Serum Iron	102.07±23.26	23.26	187.007	8.2048	-28.806	<0.01*
Serum Ferritin	98.77±	12.36	428.741	15.0897	-141.531	<0.01*
TIBC	351.93	28.48	406.561	50.1261	-7.929	<0.01*
UIBC	123.67	3.22	135.507	3.9647	-19.377	<0.01*

This table compares the clinical and biochemical parameters between the control group and the Type 2 DM group. The results demonstrate statistically significant differences across nearly all parameters.

The mean BMI was markedly higher in the diabetic group (28.69 ± 3.56) compared to the control group (23.97 ± 3.00), with a highly significant difference ($t = -8.489$, $p < 0.01$), reflecting the well-known association between obesity and Type 2 diabetes.

Glycemic indices were significantly altered. The HbA1c in the diabetic group (7.97 ± 0.70) was much higher than in controls (5.39 ± 0.24), indicating chronic hyperglycemia. Similarly, FBS (151.18 ± 11.46 vs. 90.14 ± 6.75) and PPBS (241.72 ± 16.90 vs. 130.83 ± 5.39) were significantly elevated in the diabetic group (both $p < 0.01$), confirming poor glycemic control.

Hematological parameters also differed between groups. The RBC count was lower in diabetics (4.96 ± 0.18 vs. 5.36 ± 0.17, $p < 0.01$), while hemoglobin levels were slightly but significantly higher in diabetics (13.30 ± 0.77 vs. 13.00 ± 0.60, $p = 0.019$).

The WBC count was significantly increased in the diabetic group (8.97 ± 1.12 vs. 7.24 ± 0.86, $p < 0.01$), suggesting a systemic inflammatory state. Platelet count was lower in diabetics (254.93 ± 23.37 vs. 268.54 ± 9.69, $p < 0.01$).

Iron metabolism parameters showed striking alterations. Serum iron (187.01 ± 8.20 vs. 102.07 ± 23.26) and serum ferritin (428.74 ± 15.09 vs. 98.77 ± 12.36) were markedly elevated in diabetics, both highly significant ($p < 0.01$). Similarly, TIBC (406.56 ± 50.13 vs. 351.93 ± 28.48) and UIBC (135.51 ± 3.96 vs. 123.67 ± 3.22) were also significantly higher in diabetics ($p < 0.01$)

4. Discussion

The present study demonstrated significantly elevated serum iron and ferritin levels in T2DM patients compared to controls. This supports previous findings that iron overload may contribute to the pathogenesis of T2DM by promoting oxidative stress and insulin resistance. Ferritin, an acute-phase reactant, also reflects subclinical inflammation commonly observed in diabetics.

In the context of the current findings, the elevated ferritin levels in diabetic patients may not only represent iron overload but also indicate a state of chronic inflammation. Ferritin functions as an acute-phase reactant, and its increase could be attributed to inflammatory cytokine activation, particularly interleukin-6 (IL-6), which stimulates hepcidin production. Hepcidin, in turn, restricts iron efflux from enterocytes and macrophages, leading to functional iron retention and elevated serum ferritin levels.

Oxidative stress plays a central role in diabetes-related complications. Excess iron can catalyze the Fenton reaction, producing hydroxyl radicals that damage lipids, proteins, and DNA. This chain reaction exacerbates endothelial dysfunction and promotes atherosclerosis, a leading cause of cardiovascular morbidity in diabetic individuals. Furthermore, β-cell damage from iron-induced oxidative stress reduces insulin synthesis, contributing to persistent hyperglycemia.

The correlation between elevated serum ferritin and HbA1c observed in this study aligns with previous research by Misra et al. and Borah et al., who reported significant associations between ferritin levels and markers of glycemic control. These findings support the hypothesis that iron status could serve as a potential biomarker for metabolic stress and glycemic regulation in T2DM. The bidirectional relationship between iron and glucose metabolism suggests that managing iron overload may improve insulin sensitivity and metabolic health.

Given these findings, incorporating iron status assessment into routine diabetic evaluation could be beneficial. Iron chelation, phlebotomy, or dietary interventions aimed at modulating iron intake may provide adjunctive benefits in diabetes management. However, further longitudinal studies are needed to determine causality and the therapeutic potential of iron modulation in preventing diabetes progression and its complications.

5. Conclusion

Serum iron and ferritin levels are significantly higher in Type 2 Diabetes Mellitus, correlating with poor glycemic control. Routine assessment of iron profile may provide valuable insights into metabolic status and help guide management strategies.

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