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Research Paper

Correlation between serum ferritin levels and glycemic control in non-anemic type 2 diabetic patients: a comparative hospital-based study

Dr. Priyadharshini P.*
*Assistant Professor, Department of Community Medicine, KMCH Institute of Health Sciences and Research, Tamil Nadu, India.

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ABSTRACT

Background: Serum ferritin, a marker of iron storage, is increasingly recognized as an independent risk factor for insulin resistance and poor glycemic control in type 2 diabetes mellitus (T2DM). However, its relationship with glycemic indices in non-anemic diabetic patients in the Indian population remains underexplored.
Objective: To evaluate serum ferritin levels in non-anemic T2DM patients and assess their association with glycemic control parameters, including fasting blood glucose (FBG), postprandial blood glucose (PPBG), glycated hemoglobin (HbA1c), fasting insulin, and HOMA-IR.
Methods: This comparative cross-sectional study included 120 participants: 60 non-anemic T2DM patients (Group A) and 60 age- and sex-matched healthy controls (Group B). Individuals with anemia, inflammatory, hepatic, or renal disorders, or those on iron supplementation were excluded. Serum ferritin was measured using electrochemiluminescence immunoassay (ECLIA). Statistical analysis was performed using SPSS version 25.0, applying Pearson correlation and one-way ANOVA.
Results: Mean serum ferritin levels were significantly higher in diabetic patients (186.4 ± 74.2 ng/mL) compared to controls (94.3 ± 38.6 ng/mL) ($p<0.001$). Significant positive correlations were observed between serum ferritin and HbA1c ($r=0.693$), HOMA-IR ($r=0.641$), FBG ($r=0.612$), PPBG ($r=0.587$), and fasting insulin ($r=0.524$). Ferritin levels increased progressively across HbA1c categories, with statistically significant differences ($p<0.001$).
Conclusion: Elevated serum ferritin is strongly associated with poor glycemic control and increased insulin resistance in non-anemic T2DM patients. It may serve as a useful adjunct biomarker for monitoring metabolic control in type 2 diabetes.

Corresponding Author:
Dr. Priyadharshini P.
Assistant Professor, Department of Community Medicine, KMCH Institute of Health Sciences and Research, Tamil Nadu, India.

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Email: drpriya.cm@gmail.com

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1. INTRODUCTION

Diabetes mellitus Type 2 diabetes mellitus (T2DM) is a metabolic condition that is chronic and gradually progressive in nature due to insulin resistance on top of an insulin-resistant background. It is one of the topical international health issues of the twenty-first century. The International Diabetes Federation (IDF) estimates the global adult population with diabetes as 537 million in 2021 and is expected to increase significantly during the decades [1]. Disproportionately, India, the so-called diabetes capital of the world, has to carry the epidemic burden, especially in the areas of urban and semi-urban populations [2], [3].

Although the main focus of most research has been placed on the conventional glycemic indices like the fasting blood glucose (FBG), postprandial blood glucose (PPBG) and glycated hemoglobin (HbA1c), the possible contribution of the iron metabolism in the pathophysiology of T2DM has been growing. Iron is a very vital micronutrient and is important in the transport of oxygen, in electron transfer processes in the mitochondria as also in enzyme activities. But when overloaded, iron may be a powerful pro-oxidant to produce reactive oxygen species (ROS) during the Fenton reaction and consequently cause oxidative stress to pancreatic beta-cells to develop insulin resistance [4], [5].

Ferritin, which is the major intracellular stores of iron, is a good surrogate measure of iron in the entire body. At physiological states, high levels of serum ferritin indicate the existence of higher levels of iron stores. Nevertheless, it is a reactant of acute phase as well, and in the case of inflammation, liver damage, and metabolic disregulation (all of which are typical signs of T2DM) [6], [7]) the levels of this chemical increase. Thus, to unravel the association between iron overload and glycemic dysfunction, the patient should be closely screened against anemia and other related inflammatory diseases.

Various epidemiological and clinical reports have shown that a high serum ferritin level is a risk factor that contributes to the development of T2DM and that it is also parallel with the indicators of insulin resistance [8], [9], [10]. Nevertheless, the majority of the available researches have not specifically targeted populations of non-anemic diabetics and it is hard to isolate the role of stores of iron per se and confounders related to hemoglobin. More so, there is limited data available on Indian based populations in hospitals on ferritin in comparison with glycemic control levels [11], [12].

This paper, therefore, set out to measure systematically serum ferritin levels in non-diagnostic T2DM patients and compare with non-diagnostic healthy controls and to determine the strength and the significance of correlations between serum ferritin with various glycemic parameters such as FBG, PPBG, HbA1c, fasting insulin, and HOMA-IR. The study was also interested in finding whether there is a statistically significant difference in ferritin levels in HbA1c-based glycemic control subgroups. The results of this study can be used in the formation of the idea that serum ferritin is a clinically applicable adjunct biomarker to monitor T2DM patient's metabolism.

2. RELATED WORK

The relationship between iron metabolism and glucose homeostasis, has been a matter of scientific interest over a number of decades. Were the pioneers in their systematic report to show a relationship between serum ferritin and insulin resistance that high levels of ferritin were indeed highly related to hyperinsulinemia and the metabolic syndrome factors [3]. This was later supported by population-based research which associated high iron stores with a greater likelihood of becoming diabetic (T2DM).

Used a prospective cohort study in women and found that high dietary heme iron content was significantly linked with a high risk of contracting T2DM with no regard to other nutritional and lifestyle

factors [8]. This publication involved iron-mediated mechanisms in the pathogenesis of diabetic derangements of metabolites over time. These associations were once again supported in clinical study contexts with greater ferritin and transferrin saturation in T2DM patients than in controls and there was a significant correlation with glycemic dysregulation measures [6].

Mechanistic nature of these associations has been examined by different researchers. Suggested that the insulin receptor signaling could be affected by iron overload-driven oxidative stress, which can help to explain the peripheral insulin resistance [4]. Have found that serum ferritin was a meaningful positive correlate of HOMA-IR in Korean adults but not age, BMI and inflammatory markers [9]. On the same note, showed that insulin resistance was highly correlated with ferritin despite the fact that C-reactive protein was also taken into account, implying that it is not just the result of inflammation [13].

Indian populations in particular, identified serum ferritin independently predicted T2DM in urban south Indians, supporting the importance of study of iron metabolism in Asian people having a unique genetic predisposition to metabolic diseases [14]. Swaminathan found that serum ferritin was positively correlated to a great extent with HbA1c in T2DM patients, indicating that serum ferritin could be a good biomarker of long-term glycemic control [11].

More of the studies that stratify ferritin according to control groups of HbA1c. The ferritin levels tended to be most significant among patients with HbA1c >9% that showed a gradient relationship between poor glycemic control and iron stores [12]. Similar results were also reported. in Iran that serum ferritin was also observed to be much higher in diabetics with poor control than those with better control [15]. In a hospital-based study conducted by Kim and Kim, among non-anemic diabetic T2DM patients, they demonstrated significant correlation between ferritin and HbA1c, FBG and PPBG, which indicated the targeted approach taken in the present study [16].

The association between ferritin and lipid parameters is another association investigated. Inverse correlations between ferritin and HDL- C in T2DM patients were reported by Ford and Cogswell [17], reported ferritin to be a part of the metabolic syndrome cluster, which is associated with high levels of triglycerides and a lack of tolerance to glucose [18]. Taken together, available literature suggests that there is a biologically plausible and clinically meaningful association between increased iron stores and various phenomena of metabolic dysfunction in T2DM.

3. METHODOLOGY

3.1. Study Design and Setting

It is a cross-sectional, 18 months (January 2022 to June 2023) hospital-based, comparative study in Department of Medicine and Biochemistry of a tertiary care teaching hospital in the central part of India. The appropriate ethics committee of the institution (Institutional Ethics Committee, IEC) approved the study plan and informed consent in writing was signed by all participants before they were put into the study.

3.2. Study Population

Inclusion Criteria (Group A – Cases): Patients that are aged 30-70 years with a confirmed diagnosis of T2DM according to WHO/ADA diagnostic criteria (FBG 126mg/dl and PPBG 200mg/dl or HbA1c 6.5%at two separate occasions) and have hemoglobin level greater than 12g/dl (in females) [19], [20].

Inclusion Criteria (Group B – Controls): The controls were age and sex-adjusted, healthy people with no diabetes, normal fasting glucose levels (less than 100 mg/dL) and normal hemoglobin levels.

Exclusion Criteria: Both groups excluded patients with anemia (hemoglobin less than the specified levels), known inflammatory conditions, hepatic conditions, chronic kidney disease (serum creatinine above 1.5 mg/dL), malignancy, hemochromatosis, thalassemia, recent blood transfusion, iron supplementation during the last three months, pregnancy, or type 1 diabetes mellit.

3.3. Sample Size

A sample size of 55 per with a 80% power and significance level at 5% (two tails) was calculated based on a previous study that reported an average difference in serum ferritin of 80 ng/mL between the

T2DM patients and the controls (SD 70 ng/mL). In order to take into consideration, the dropouts, 60 people were recruited in each group to get a total of 120 individuals.

3.4. Data Collection and Laboratory Investigations

Demographic information was entered using the structured proforma, anthropometric measurements (height, weight, BMI) and blood pressure, diabetes time, and medications. After a period least of 10 hours of overnight fasting, the blood samples (8 mL) were fasted in the vein. Centrifugation of serum was used to separate serum at 3000 rpm during 10 minutes and kept in -20 C until analysis.

The subsequent tests were conducted: fasting blood glucose (enzymatic colorimetric method), postprandial blood glucose (two hours after 75g glucose load) fasting serum insulin (CLIA method), and lipid profile (enzymatic methods). The calculation of HOMA-IR was made by the following equation: HOMA-IR = [Fasting Insulin (μIU/mL) 250 FBG (mg/dL)/405].

3.5. Statistical Analysis

An IBM SPSS statistics version 25.0 (Chicago, IL, USA) was used to enter and analyze all data. Mean and standard deviation (SD) were used to indicate the continuous variables. Categorical data were summarized in terms of frequencies and percentages. Comparison of means of two groups was done by use of student independent t-test. The strengths and directions of the linear associations between glycemic parameters and serum ferritin were evaluated using Pearson correlation coefficient (r). Ferritin was compared using a one-way version of ANOVA with post-hoc test and Tukey. This was taken to be a statistically significant p-value of less than 0.05.

4. RESULTS AND DISCUSSION

4.1. Demographic and Clinical Characteristics

Table 1 shows the demographics and clinical parameters of two groups at the baseline. The two groups were similar in terms of age (p=0.682) as well as in terms of sex distribution (p=0.721) which shows they were successfully matched. However, cases had significantly higher BMI (27.6 ± 3.4 vs. 24.1 ± 2.9 kg/m^2, p=0.003), systolic blood pressure (134.2 ± 12.3 vs. 122.4 ± 10.1 mmHg, p=0.001), and diastolic blood pressure compared to controls. Both groups had similar values of hemoglobin (13.8 ± 1.2 vs. 14.1 ± 1.0 g/dl, p=0.143), indicating that neither was anemic.

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

Parameter	Group A (Cases) n=60	Group B (Controls) n=60	p-value
Age (years, mean±SD)	52.4 ± 9.1	51.8 ± 8.7	0.682
Males / Females	34 / 26	32 / 28	0.721
BMI (kg/m^2)	27.6 ± 3.4	24.1 ± 2.9	0.003*
Duration of DM (years)	8.2 ± 4.6	—	—
Systolic BP (mmHg)	134.2 ± 12.3	122.4 ± 10.1	0.001*
Diastolic BP (mmHg)	84.6 ± 8.7	79.1 ± 7.5	0.002*
Hemoglobin (g/dL)	13.8 ± 1.2	14.1 ± 1.0	0.143
Serum Creatinine (mg/dL)	0.91 ± 0.18	0.88 ± 0.16	0.317

** Statistically significant (p<0.05). SD: Standard Deviation; BP: Blood Pressure; BMI: Body Mass Index.*

4.2. Serum Ferritin and Glycemic Parameters

Table 2 reveals that in diabetic patients (186.4±74.2 ng/mL) serum ferritin levels were heavily elevated unlike the controls (94.3±38.6 ng/mL) with the difference being highly significant (p<0.001). This observation is related to previous reports by Swaminathan [11], Kim and Kim [16], [12], who also reported high ferritin in T2DM patients compared to the patients with normal sugar levels.

Likewise, FBG, PPBG and HbA1c, fasting insulin, and HOMA-IR (glycemic parameters) were also significantly elevated in the case group (p<0.001). Pearson's correlation analysis within the diabetic group revealed significant positive correlations between serum ferritin and all glycemic indices: HbA1c (r=0.693, p<0.001), HOMA-IR (r=0.641, p<0.001), FBG (r=0.612, p<0.001), PPBG (r=0.587, p<0.001), and fasting insulin (r=0.524, p<0.001).

Table 2. Comparison of Serum Ferritin and Glycemic Parameters between Cases and Controls

Parameter	Cases (Mean±SD)	Controls (Mean±SD)	P-Value	R Value
Serum Ferritin (ng/mL)	186.4 ± 74.2	94.3 ± 38.6	<0.001*	—
FBG (mg/dL)	168.7 ± 42.1	92.4 ± 10.2	<0.001*	0.612*
PPBG (mg/dL)	234.5 ± 58.3	126.8 ± 18.4	<0.001*	0.587*
HbA1c (%)	8.6 ± 1.4	5.3 ± 0.4	<0.001*	0.693*
Fasting Insulin (µIU/mL)	18.4 ± 6.2	10.1 ± 3.5	<0.001*	0.524*
HOMA-IR	7.6 ± 2.8	2.3 ± 0.9	<0.001*	0.641*

**Statistically significant (p<0.05). FBG: Fasting Blood Glucose; PPBG: Postprandial Blood Glucose; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; r: Pearson's correlation coefficient.*

Correlations among ferritin HbA1c (r=0.693) yielded the greatest correlation, indicating cumulative glycemic exposure over three months is most correlated with iron stores. This is consistent with the hypothesis suggesting that iron overload can have chronic adverse effects on insulin-signaling pathways both in the extent that it negatively affects beta-cell function as well as in the extent that it contributes to peripheral insulin resistance [7], [5], [10]. The strong association with HOMA-IR (r=0.641) further supports the mechanistic interrelationship between high levels of iron stores and insulin resistance. Figure 1 indicates the strength of correlation between serum ferritin and some glycemic parameters in the diabetic population. The gradient representation shows that HbA1c and HOMA-IR exhibit the best associations with ferritin, then FBG, PPBG and fasting insulin.

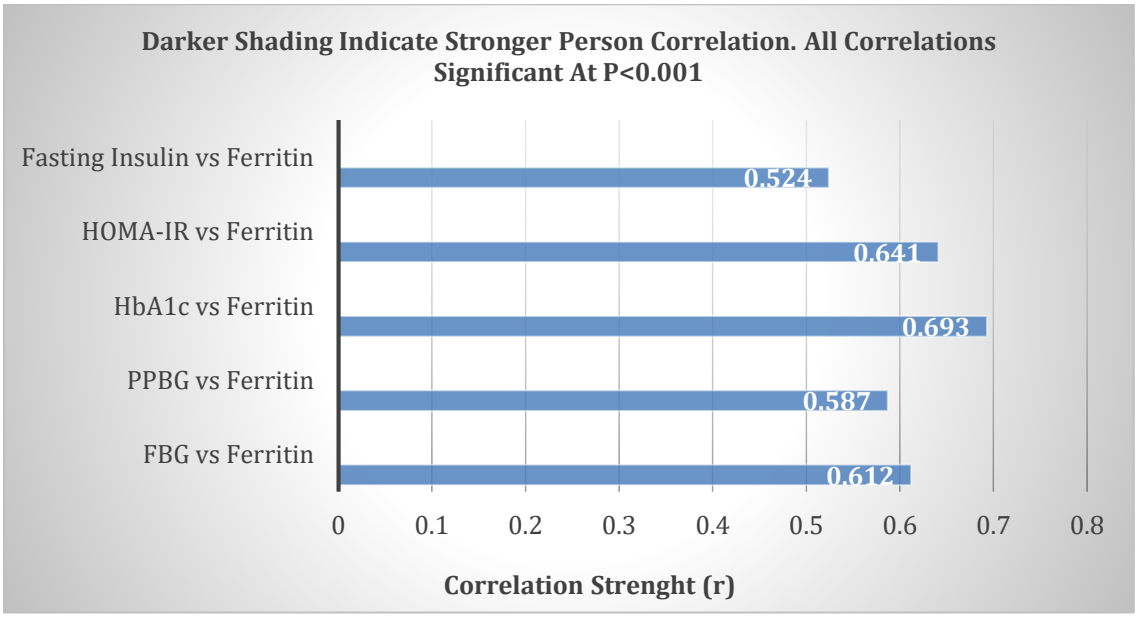

Figure 1. Correlation Strength (r values) Between Serum Ferritin and Glycemic Parameters in Type 2 Diabetic Patients

4.3. Ferritin Levels Across HbA1c-Defined Glycemic Control Groups

Table 3 a statistical analysis indicated a statistically significant increase in serum ferritin in good control (128.4 + 42.3 ng/mL), fair control (172.6 + 58.7 ng/mL), and poor control (226.8 + 82.4 ng/mL) resulted in statistically significant increase (p<0.001) to show that a progressive increase This dose-

response pattern indicates a graded relationship that is assumed to be biologically plausible, with causal association. [12], [15] conducted similar stratified analyses, which found similar trends. This premise suggests that progressive iron build-up might be linked with the impairment of metabolic regulation, which could lead to a vicious circle of oxidative stress, beta-cell damage, and additional metabolic decline [5], [10].

Table 3. Serum Ferritin and Glycemic Parameters Across HbA1c-Defined Control Groups

HbA1c Group	n	Serum Ferritin (ng/mL, mean±SD)	FBG (mg/dL)	PPBG (mg/dL)
Good control (<7%)	12	128.4 ± 42.3	118.6 ± 22.1	168.2 ± 30.4
Fair control (7–8%)	21	172.6 ± 58.7	152.4 ± 36.5	218.7 ± 48.2
Poor control (>8%)	27	226.8 ± 82.4	204.3 ± 52.7	276.5 ± 66.8
p-value (ANOVA)	—	<0.001*	<0.001*	<0.001*

**Statistically significant by one-way ANOVA (p<0.001). Post-hoc Tukey's test showed significant differences between all pairs of subgroups.*

Figure 2 shows that the mean serum ferritin levels increase gradually as one goes up an order in the order of the control group, good, fair, poor glycemic control subgroups and this gives a very clear picture of the dose-response curve.

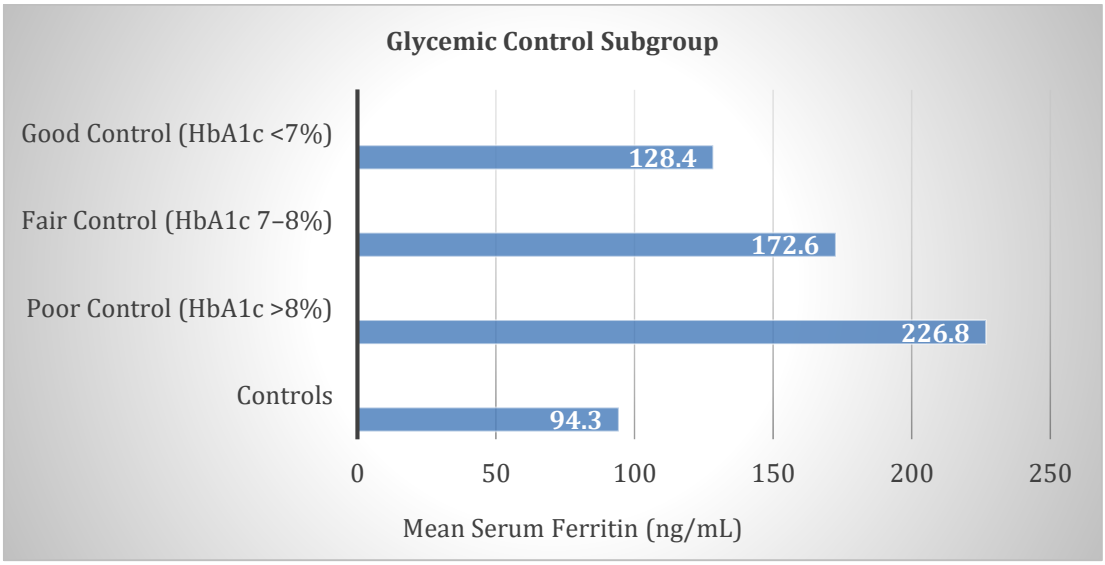

Figure 2. Mean Serum Ferritin Levels Across Glycemic Control Subgroups and Controls

4.4. Serum Ferritin and Lipid Profile

Table 4 shows that the lipid profiles of diabetic patients were significantly different, as compared to those of controls. The diabetic group has significant positive correlations between serum ferritin and total cholesterol (r=0.412), triglycerides (r=0.536), LDL-C (r=0.388), and VLDL-C (r=0.501), and a significant negative correlation between serum ferritin and HDL-C (r=−0. These results build on the metabolic correlations of high levels of ferritin beyond the glycemic factors to lipid misregulation, casting the iron overload in a wider cardiometabolic risk picture [17], [18], [21].

Table 4. Comparison of Lipid Profile Parameters and Correlation with Serum Ferritin in the Diabetic Group

Lipid Parameter	Cases (mean±SD)	Controls (mean±SD)	p-value	r (Ferritin)
Total Cholesterol (mg/dL)	214.6 ± 38.2	178.4 ± 26.1	0.001*	0.412*
Triglycerides (mg/dL)	186.3 ± 52.4	128.6 ± 34.2	<0.001*	0.536*
LDL-C (mg/dL)	138.4 ± 32.6	106.2 ± 24.8	0.001*	0.388*
HDL-C (mg/dL)	42.1 ± 6.8	52.4 ± 7.2	0.001*	−0.421*

VLDL-C (mg/dL)	37.2 ± 10.5	25.7 ± 6.9	<0.001*	0.501*

**Statistically significant (p<0.05). LDL-C: Low-Density Lipoprotein Cholesterol; HDL-C: High-Density Lipoprotein Cholesterol; VLDL-C: Very Low-Density Lipoprotein Cholesterol.*

4.5. Mechanistic Insights and Clinical Implications

The mechanisms by which high levels of iron stores diminish glycemic control are multifactorial and biological in nature. Oxidative stress caused by iron through Fenton chemistry results in hydroxyl radicals, which cause harm to pancreatic beta-cell membrane and mitochondria, decreasing insulin secretory ability [5]. Moreover, hepatic lipid peroxidation, which is caused by iron, disrupts the phosphorylation of insulin receptor substrate, which is also the cause of hepatic insulin resistance [22]. Direct pro-inflammatory signaling, per se, by ferritin itself may further increase cytokine-mediated insulin resistance [10].

Clinically, the current results indicate that serum ferritin estimation can offer more prognostic data than the traditional glycemic indices in the management of T2DM. There is limited evidence on the targeted iron reduction interventions in T2DM utilizing randomized controlled trials [23]. The strengths of the present study are the absence of anemic patients, a broad spectrum of glycemic and lipid parameters, and hospital-based comparative design. This has limitations such as cross-sectional design, and relatively small sample size, as well as being constrained to a single center.

5. CONCLUSION

This cross-sectional and comparative hospital-based research has shown that serum ferritin has strongly positive relationships with various glycemic and measures such as HbA1c, FBG, PPBG, fasting insulin and HOMA-IR. It had the highest correlation with HbA1c (r=0.693), which emphasizes the connection between long-term glycemic dysregulation and iron accumulation. An increasing gradient of ferritin concentration between HbA1c-defined subgroups of glycemic control further points to a dose-response relationship.

Moreover, ferritin was positively associated with parameters of lipid dysregulation, indicating its extended cardiometabolic risk.

These data suggest that serum ferritin is a useful adjunct biomarker to facilitate metabolic tracking in T2DM. To determine whether controlled decrease of iron stores can lead to better glycemic levels and lower cardiovascular risk in type 2 diabetic patients, future longitudinal and interventional studies are justified.

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Author Contributions Statement

Name of Author	C	M	So	Va	Fo	I	R	D	O	E	Vi	Su	P	Fu
Dr. Priyadharshini P.	✓	✓	✓	✓		✓		✓	✓	✓	✓			

C : **C**onceptualization
M : **M**ethodology
So : **So**ftware
Va : **Va**lidation
Fo : **Fo**rmal analysis
I : **I**nvestigation
R : **R**esources
D : **D**ata Curation
O : Writing - **O**riginal Draft
E : Writing - Review & **E**diting
Vi : **Vi**sualization
Su : **Su**pervision
P : **P**roject administration
Fu : **Fu**nding acquisition

Conflict of Interest Statement

The authors declare that there are no conflicts of interest regarding the publication of this paper.

Informed Consent

All participants were informed about the purpose of the study, and their voluntary consent was obtained prior to data collection.

Ethical Approval

Not applicable.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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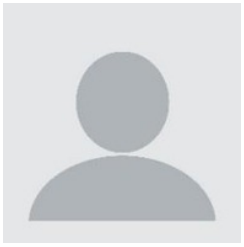
BIOGRAPHIE OF AUTHOR



Dr. Priyadharshini P., is an Assistant Professor in the Department of Community Medicine at KMCH Institute of Health Sciences and Research, Tamil Nadu, India. She holds an MBBS degree along with MD and DNB qualifications in Community Medicine. Her academic and professional interests are centered on community medicine, preventive healthcare, epidemiology, and public health research. She is involved in teaching, clinical training, and research activities aimed at improving population health and strengthening evidence-based healthcare practices. Email: drpriya.cm@gmail.com