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Original article

Serum ferritin in Cardiovascular Disease – Study of Inflammation driven anaemia

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Abstract:

Background: *Cardiovascular disease (CVD) is the leading cause of mortality globally, accounting for 17.9 million deaths annually. In India, it is a significant public health issue, responsible for 28% of total deaths. Anemia, particularly iron deficiency anaemia, often coexists with CVD, exacerbating cardiovascular complications. Serum ferritin levels, which correlate with total body iron stores, are complex in CVD patients due to the dual role of ferritin reflecting iron storage and an acute-phase reactant. This study aims to evaluate serum ferritin levels in CVD patients to better understand the relationship between ferritin, anemia, and inflammation.*

Methodology: *The study used a retrospective cross-sectional research model and convenient sampling methodologies. Participants were divided into a study group (CVD with Anaemia) and a control group (CVD-only). Participants were classified as anaemic based on hemoglobin thresholds. The study was approved by the Institution Committee of Ethics in Human Research and each patient signed a written informed permission form. The study was conducted based on American Heart Association criteria and comprehensive clinical evaluations. The study's ethical standards were upheld by the Indian Council of Medical Research.*

Result: *The prevalence of anaemia was 61.3%, with 89 out of 145 meeting the criteria. Ferritin levels were similar between the two groups, but no significant differences were found in other inflammatory parameters.*

Conclusion: *This study highlights challenges in using serum ferritin alone to assess anemia in cardiovascular disease patients. Ferritin levels were slightly higher in anemic patients but not significantly different, suggesting it may reflect inflammation more than iron levels. Additional markers like transferrin and inflammatory indices should be considered when evaluating anemia in CVD.*

Keywords: *CVD, Iron deficiency anemia, IL-6, NLR ratio, Ferritin, Inflammation*

Background:

Cardiovascular disease (CVD) is the leading cause of mortality globally, accounting for 17.9 million deaths annually, which constitutes 31% of all deaths. In India, CVD has become a significant public health issue, responsible for 28% of total deaths [1]. The prevalence is increasing among younger adults due to factors such as urbanization, lifestyle changes, and rising incidences of diabetes, hypertension, and dyslipidemia [2]. Concurrently, anaemia, particularly iron deficiency anaemia, often coexists with CVD, exacerbating cardiovascular complications by increasing the heart's workload [3]. This can lead to left ventricular hypertrophy, heart failure, and ischemia, further worsening the prognosis of patients with CVD [4].

Ferritin is the primary intracellular iron storage protein and the best single indicator of total body iron stores [5]. Serum ferritin levels correlate with the body's total amount of stored iron, with each microgram per litre (µg/L) of serum ferritin corresponding to approximately 8-10 mg of stored iron [6]. However, its interpretation is complex in

cardiovascular disease patients. In CVD, serum ferritin can be elevated due to inflammation, even when iron deficiency is present [7]. This is because ferritin reflects iron storage and functions as an acute-phase reactant, rising in response to inflammation [8]. The dual role of ferritin complicates the evaluation of anaemia in CVD patients, as elevated ferritin may not necessarily indicate sufficient iron stores but could be falsely increased due to the inflammatory processes associated with cardiovascular conditions [9].

This study aims to evaluate serum ferritin levels in CVD patients to distinguish whether elevated ferritin indicates iron deficiency or an inflammatory state. By enhancing our understanding of the relationship between ferritin, anaemia, and inflammation in CVD, this research can help guide more precise clinical strategies for managing iron deficiency and improving cardiovascular outcomes.

Method

Study design:

This study was based on a retrospective cross-sectional research model and convenient sampling methodologies. The data were collected from the general medicine OPD, Sri Ramachandra Institute of Higher Education and Research, Porur, Chennai, Tamil Nadu, India, for all patients who had confirmed CVD with Anemia (study group) and CVD only (control group) between 2021 and 2023 in outpatient communities in chennai, Tamil Nadu. Patient data were collected from both manual and electronic records provided by the Medical Records Department.

Population information

The sample size was calculated based on alpha=0.001, beta=0.1 (power=95%), 10% relative precision.

$$\frac{2S^2 (Z_{1-b} + Z_{1-a/2}) 2}{(m_1 - m_2)^2}$$

Based on the previous study [10], the sample size was calculated to be 221. However, after excluding outliers, the final sample size was adjusted to 145 individuals. Patient data were collected from both manual and electronic records provided by the Medical Records Department at Sri Ramachandra Institute of Higher Education and Research.

Laboratory findings:

Laboratory blood tests were performed for parameters related to complete blood counts (CBCs), lipid profiles, including total cholesterol (TC), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), and triglycerides (TG); and inflammatory markers C-reactive protein (CRP), IL-6 and NLR ratio, Iron deficiency profile, including serum iron, ferritin, total iron binding capacity (TIBC).

Participant groups and criteria: Patients were divided into a study group (CVD with Anaemia; CVD-ANA) and a control group (CVD-only) based on the principal indications for CVD. Participants were classified as anaemic based on haemoglobin thresholds consistent with guidelines from the World Health Organization and other established medical authorities [11]. Specifically, men were considered anaemic if their haemoglobin concentration was below 13.0 g/dL, while the threshold for women was set at less than 12.0 g/dL.

Inclusion criteria: subjects in the age group of above 18 years clinically diagnosed with cardiovascular disease (CVD). Diagnosis was established through American heart Association criteria with comprehensive clinical evaluations and diagnostic testing. This included medical record review, physician assessments, imaging studies, blood tests, electrocardiograms, and exercise stress tests [12].

Exclusion criteria: Individuals with chronic inflammatory conditions such as chronic liver disease, chronic kidney disease, arthritis, Irritable bowel disease and active infections. Adults with known history of endocrine disorders cancer, and relevant drug treatment, a history of repeated blood transfusions, genetic causes of iron overload and also adults undergoing treatment for any other co-morbid conditions were excluded.

Ethics:
The protocol was approved by the Institution Committee of Ethics in Human Research, which is a division of the Sri Ramachandra Institute of Higher Education and Research, in accordance with Indian Council of Medical Research regulations (Ref No. CSP/20/NOV/87/181). Each patient who wished to participate in the research signed a written informed permission form after being informed.

Results
This study was conducted with 145 participants aged 19 years and above, from both sexes. Two groups of participants were considered from the sample used in this study: the CVD-ANA study group (n = 89) and the CVD-only control group (n = 56). The CVD-ANA group comprised 39.5% males and 60.5% females, while the CVD - only group comprised 65.5% males and 34.5% females (table.1). The diagnostic criteria for anaemia in this study were aligned with widely recognized clinical standards, primarily determined by measuring blood haemoglobin levels. Participants were classified as anaemic based on haemoglobin thresholds consistent with guidelines from the World Health Organization and other established medical authorities. Specifically, men were considered anaemic if their haemoglobin concentration was below 13.0 g/dl, while the threshold for women was set at less than 12.0 g/dl, based on iron metabolism parameters like serum iron, total iron binding capacity. The prevalence of anaemia in the study population was found to be 61.3%, with 89 out of the 145 participants meeting the criteria for anaemia. The mean ferritin levels were 508.9±25.9 for anaemic participants and 505±21.46 for non-anaemic individuals (table.1). The Mann-Whitney U test did not reveal any statistically significant differences when comparing serum ferritin levels between study group and control group. Similarly, comparing ferritin with other inflammatory parameters like CRP, IL-6 and NLR ratio between anaemic and non-anaemic group showed no significant differences.

Variable	**CVD-only (mean ± SD) (n=56)**	**CVD-ANA (mean ± SD) (n=89)**
Age (years)	51.5 ± 16.05	60.0 ± 16.0
Male	65.5%	39.5%
Female	34.5%	60.5%
HDL(mg/dl)	32.2±1.7	30.9±2.69*[a]
LDL(mg/dl)	177.7±8.6	171.6±10.4*[a]
T. Cholesterol(mg/dl)	247±20.9	236±24.8*[a]
TGL(mg/dl)	239.9±18.7	232.3±20.1*[a]
CRP (mg/L)	5.07±0.77	5.25±0.84*[a]
IL-6 (pg/ml)	33.79±2.58	33.9±3.03*[a]
NLR ratio	4.01±0.38	4.12±0.26*[a]
Hb (gm/dl)	13.6±1.88	9.7±0.5**[a]
S. Iron (mcg/dl)	84±17	32.06±3.4 **[a]
TIBC(mcg/dl)	330.5±36.8	523.4±34.9**[a]
S. Ferritin (ng/ml)	505±21.46	508.9±25.9[NA]

Table 1 Comparison of demographic data and laboratory findings between CVD-ANA patients and CVD-only patients

The normal ranges of each parameter were as follows: T. Cholesterol: <200 mg/dl, Triglycerides: <200 mg/dl, HDL: >40 mg/dl, and LDL: <130 mg/dl. CRP :< 0.5 mg/dl, NLR ratio: 0.78 – 3.53, Hb: > 13gm/dl, IL-6: < 5 pg/l, S. Iron: 35-145mcg/dl, TIBC: 240-450mcg/dl, Serum Ferritin: 30-400ng/l.In the table, the results represent the arithmetic mean with ± predictable error of subjects. The statistical drift of the study was set at p value less than 0.01* or p value less than 0.001**. ANOVA between the experimental groups was performed and mentioned as a symbol of **a**-compared with the control group mean.

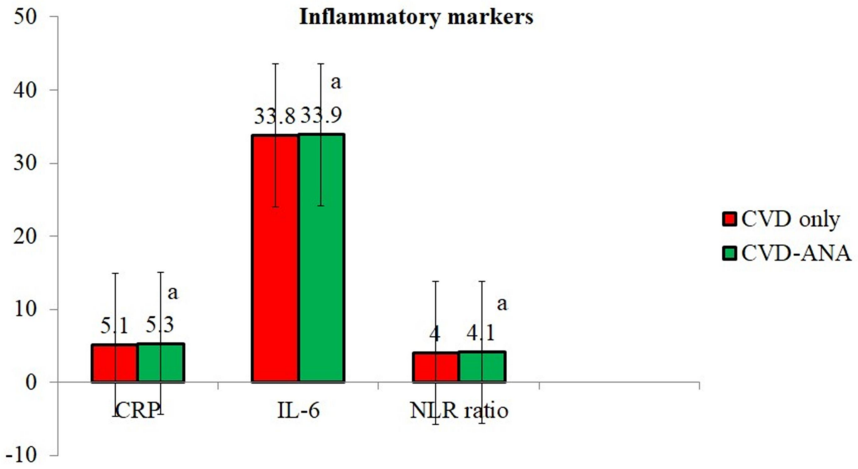

Figure 2. A comparison of inflammatory markers between cardiovascular patients in the study groups.

The results represent the arithmetic mean with ± predictable error of thirty experimental subjects. The statistical difference between the study groups was not observed. ANOVA between the experimental groups was performed and mentioned in the bar diagram as a symbol of **a**-compared with the control group mean; and **b**-compared with the mean of CVD with anemic group.

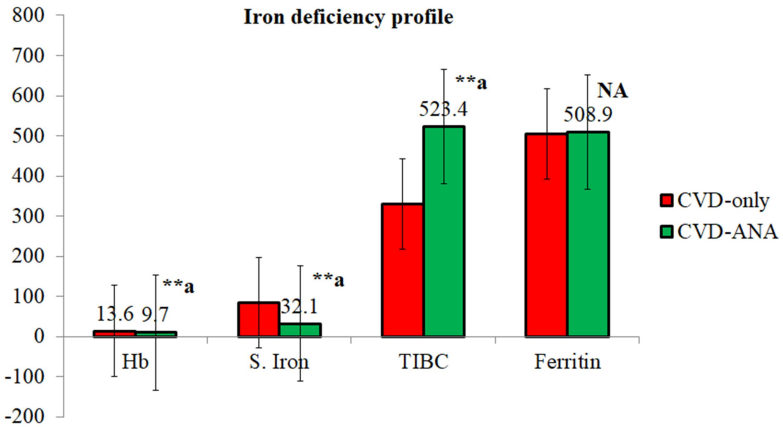

Figure. 3. A comparison of Iron deficiency anemia profile between cardiovascular patients in the study groups.
The results represent the arithmetic mean with ± predictable error of thirty experimental subjects. The statistical drift of the study was set at p value less than 0.01* or p value less than 0.001**, NA- no statistical difference. ANOVA between the experimental groups was performed and mentioned in the bar diagram as a symbol of **a**-compared with the control group mean; and **b**-compared with the mean of CVD with anemic group.

Discussion

The findings of this study highlight the complex interplay between serum ferritin, anaemia, and inflammation in the context of cardiovascular disease. The study found no significant association between elevated ferritin levels and anaemic status in the overall CVD population. This suggests that ferritin may not be a reliable indicator of iron deficiency anaemia in CVD patients, as it can be influenced by the inflammatory processes associated with cardiovascular conditions [13]. Though lacking in statistical significance for many comparisons with serum iron, TIBC, the findings provide essential insights into the complex interplay between ferritin, haemoglobin, and inflammation, which are all relevant to the pathophysiology of anaemia and cardiovascular diseases. Furthermore, by comparing the study's findings with existing research, we can better understand ferritin's role in iron metabolism, its response to inflammation, and its implications for cardiovascular health [14].

However, the study did uncover a significant inverse relationship between ferritin and haemoglobin in the iron deficiency anaemic group of participants with accompanying inflammation. This finding aligns with the notion that in the presence of inflammation, ferritin levels can rise even when iron stores are depleted, complicating the assessment of anaemic status [15]. This draws attention to the need to consider inflammation as a potential confounding factor when interpreting ferritin levels in CVD patients.

Ferritin is a critical intracellular protein that facilitates the storage and sequestration of excess iron within the body [16]. Composed of 24 subunits, ferritin is primarily found in the liver, spleen, and bone marrow, serving as a buffer against iron deficiency and overload. Serum ferritin, a commonly measured clinical parameter, is generally regarded to represent the body's iron reserves under normal physiological conditions [17]. Each ferritin molecule can accommodate approximately 4,500 iron atoms, making it a pivotal regulator of iron homeostasis within the organism [18]. However, ferritin also serves a dual function as an acute-phase reactant, meaning its levels can rise in response to inflammation, infection, or tissue damage, independent of iron status [19]. Inflammatory cytokines, particularly interleukin-6 (IL-6), can stimulate ferritin synthesis, causing its serum levels to increase during inflammatory states such as cardiovascular disease (CVD) [20]. This ability of ferritin to act as an acute-phase protein complicates its role as a marker of iron stores, particularly in patients with chronic inflammation, such as those with CVD. In such cases, elevated serum ferritin levels may not indicate sufficient iron reserves but instead reflect the body's inflammatory response [21].

The findings of this study are consistent with this understanding, as the inverse relationship between ferritin and haemoglobin, serum iron and TIBC was observed in the study group of participants with accompanying inflammation. This suggests that ferritin may not be a reliable indicator of anaemic status in CVD patients in the presence of inflammation. Other markers, such as soluble transferrin receptor, may be necessary to more accurately assess iron deficiency [22].

The study found a significant inverse correlation between serum ferritin and haemoglobin levels in participants with concomitant inflammation. This suggests that higher ferritin concentrations were associated with lower haemoglobin values within this study group. This inverse relationship aligns with the known pathophysiology of anaemia of chronic disease, wherein ferritin levels rise as part of the body's inflammatory response [23]. Inflammatory cytokines, particularly interleukin-6, stimulate the synthesis of ferritin, but this increase does not translate to improved iron availability for erythropoiesis. Instead, iron becomes sequestered within cells, leading to a functional iron deficiency that impairs red blood cell production and contributes to the development of anaemia [25]. This inflammation-driven anaemia occurs in the context of chronic conditions like cardiovascular disease, where pro-inflammatory cytokines, such as IL-6, stimulate hepcidin production, inhibiting iron absorption and trapping iron within macrophages [26]. This reduces iron availability for erythropoiesis, causing anaemia despite normal or elevated ferritin levels, complicating diagnosis and management [27].

This inverse correlation between ferritin and haemoglobin is consistent with the findings of other studies in patients with chronic diseases, including CVD. Elevated ferritin levels were associated with poor outcomes and were indicative of an inflammatory state rather than iron sufficiency [28]. Therefore, the interpretation of ferritin levels in

the context of CVD must take into account the presence of inflammation, as ferritin can act as a marker of disease severity and inflammation rather than as an accurate indicator of iron stores.

Another finding of this study was the correlation between serum ferritin and lipid profiles, including total cholesterol and LDL-cholesterol levels. This result suggests that ferritin may play a direct role in lipid metabolism in this particular population, although previous research has been mixed. Ferritin's involvement in oxidative stress has led some researchers to hypothesize a link between ferritin and dyslipidemia. Iron deficiency can contribute to the generation of reactive oxygen species (ROS), which in turn can oxidize LDL particles and promote atherosclerosis [29].

The present study found significant correlation between serum ferritin levels and lipid profiles, suggesting that ferritin's involvement in cardiovascular disease may be more closely linked to its role as an inflammatory marker rather than a direct influence on lipid metabolism. This contrasts with some previous research, such as the work by Ellidag et al., which has indicated a potential association between ferritin and lipid peroxidation in specific contexts. However, the relationship does not appear consistent across all populations, as evidenced by the lack of a significant association in this study [30].

The findings of this study have important clinical implications, especially for the management of anaemia in patients with CVD. Elevated ferritin levels, especially in the context of inflammation, may not reflect factual iron sufficiency. Instead, they may be a marker of inflammation, indicating a need for a more nuanced approach to diagnosing and treating anaemia in CVD patients. The use of additional markers, such as transferrin saturation (TSAT), C-reactive protein (CRP), and soluble transferrin receptor (sTfR), can provide a clearer picture of a patient's iron status and inflammatory burden [31].

Managing anaemia in CVD is essential, as untreated anaemia can worsen cardiovascular outcomes by increasing cardiac workload, reducing oxygen delivery to tissues, and exacerbating heart failure symptoms. Iron supplementation, particularly intravenous iron, has been shown to improve outcomes in heart failure patients with iron deficiency, even in the absence of overt anaemia. Given the complexity of interpreting ferritin levels, clinicians should consider inflammation and functional iron deficiency when evaluating iron status and determining the appropriate treatment.

Conclusion

This study underscores the challenges in utilizing serum ferritin as the sole indicator of anemic status in patients with cardiovascular disease. While ferritin levels were marginally higher in anemic individuals, the difference lacked statistical significance, reflecting the multifactorial nature of anaemia in CVD. The role of ferritin as an acute-phase reactant suggests its elevation may be more representative of inflammation rather than true iron adequacy. These findings emphasize the importance of considering additional markers, such as transferrin saturation and inflammatory indices, when assessing iron status and anaemia in CVD patients.

The study highlights the complexity of interpreting serum ferritin levels in the context of cardiovascular disease, particularly in the presence of inflammation. While ferritin is a crucial indicator of iron stores under normal conditions, its function as an acute-phase reactant complicates its use in diagnosing iron deficiency in CVD patients. The significant inverse relationship between ferritin and haemoglobin in participants with inflammation underscores the significance of considering inflammatory status when evaluating ferritin levels. These findings support the growing evidence that ferritin can be a marker of inflammation rather than iron sufficiency in chronic disease states like CVD. Further research is warranted to explore ferritin's role in the pathophysiology of CVD and its potential utility as a biomarker for guiding anaemia management in this population.

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