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# Interrelationship of Serum Ferritin and High-Sensitivity C-Reactive Protein in Ischemic Heart Disease: A Cross-Sectional Study from Northeast India

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## ABSTRACT

**Background**: Ischemic heart disease (IHD) remains the leading global cause of mortality, with inflammation and iron metabolism playing crucial roles in its pathophysiology. High-sensitivity C-reactive protein (hs-CRP) is a marker of systemic inflammation, while ferritin reflects both iron storage and acute-phase response.
**Objective**: To investigate the correlation between serum ferritin and hs-CRP levels in patients with IHD.
**Methods**: A cross-sectional study was conducted involving 104 patients from January 2024 to June 2025. Serum ferritin levels were determined by enzyme-linked immunosorbent assay (ELISA), while hs-CRP levels were assessed using immunoturbidimetric assays. Clinical profiles, electrocardiogram changes, and comorbidities were systematically documented. Multiple regression analysis identified independent predictors of serum ferritin and hs-CRP levels within this cohort.
**Results**: Seventy-five percent of patients had high ferritin levels (>300 ng/mL), with an average of 373.6 ± 100.5 ng/mL. The average hs-CRP was 3.04 ± 1.36 mg/L, and 44.2% of patients had hs-CRP levels above 3 mg/L, which is linked to higher cardiovascular risk. Patients with unstable angina showed the highest ferritin (405.1 ± 107.4 ng/mL) and hs-CRP (3.9 ± 1.3 mg/L) levels. The correlation between ferritin and hs-CRP was weak and not significant (p=0.306).
**Conclusion**: Both ferritin and hs-CRP levels were higher in patients with IHD, suggesting distinct pathophysiological mechanisms—specifically, dysregulated iron metabolism and systemic inflammation. Because the link between these markers was weak, it is important to use a combination of biomarkers rather than a single biomarker for risk assessment.

***Keywords***: *Ischemic heart disease, hs-CRP, serum ferritin, inflammation, biomarkers, cardiovascular risk.*

## INTRODUCTION

Ischemic heart disease (IHD) continues to be the leading cause of death worldwide, accounting for nearly 9 million deaths annually according to the Global Burden of Disease study.[1] The condition arises primarily from atherosclerosis, a chronic inflammatory process characterised by lipid deposition, endothelial dysfunction, and plaque formation within coronary arteries.[2] Although diagnostic and therapeutic strategies have advanced, the identification of reliable biomarkers reflecting both iron metabolism and systemic inflammation remains essential for effective risk stratification and prognosis in patients with IHD.

### Inflammation and Cardiovascular Risk

Inflammation plays a pivotal role in the initiation, progression, and destabilisation of atherosclerotic plaques. High-sensitivity C-reactive protein (hs-CRP), a refined assay of the classical acute-phase reactant CRP, has emerged as a sensitive marker of systemic inflammation.[3] Elevated hs-CRP levels have been consistently associated with elevated risk of myocardial infarction, stroke, and cardiovascular mortality.[4] Katamine et al. demonstrated that hs-CRP predicts future cardiovascular events independent of traditional lipid parameters, underscoring its utility in clinical practice.[5] Current prevention guidelines recognise hs-CRP≥2 mg/L as a risk-enhancing factor for atherosclerotic cardiovascular disease.[6]

### Serum Ferritin and Iron Metabolism

Ferritin is the primary intracellular protein responsible for iron storage. Serum ferritin levels generally reflect body iron stores, but ferritin also behaves as an acute-phase reactant, rising in response to systemic inflammation.[7] High ferritin has been linked to oxidative stress, endothelial injury, and increased cardiovascular risk.[8] Conversely, iron deficiency, reflected by low ferritin, is associated with poor outcomes in chronic heart failure and IHD patients due to impaired oxygen transport and myocardial energetics.[9] Thus, ferritin represents a dual-nature biomarker, simultaneously indicating iron status and inflammatory activity.

### Interplay Between Ferritin and hs-CRP

The relationship between ferritin and hs-CRP is particularly relevant in IHD, where both iron metabolism and inflammation are dysregulated. Inflammatory conditions increase both ferritin and CRP levels, making it difficult to interpret ferritin alone as a marker of iron status. Urbanski et al. suggested that the ferritin/CRP ratio may be a more reliable indicator of iron deficiency during systemic inflammation.[10] Assessing ferritin and hs-CRP together may provide clearer insights into the pathophysiology of IHD, especially in differentiating iron overload from inflammation-induced hyperferritinemia.

### Evidence from Clinical Studies

Several studies have explored the prognostic significance of these biomarkers in cardiovascular disease. Aday and Ridker highlighted hs-CRP as a marker of residual inflammatory risk, suggesting that therapies targeting inflammation may reduce cardiovascular events.[11] Henein et al. demonstrated the role of ferritin in oxidative stress and vascular injury, linking elevated levels to adverse outcomes in coronary artery disease.[12] Malthesh et al. reported that elevated hs-CRP and ferritin levels were linked to longer durations of hospitalisation.[13]

### Rationale for the Present Study

Given the dual role of ferritin as both an iron storage protein and an acute-phase reactant, and the established predictive value of hs-CRP in cardiovascular disease, exploring their relationship in IHD patients may provide insights into the interplay between iron metabolism and inflammation. Understanding this association could help refine risk stratification, guide therapeutic interventions, and improve patient outcomes. This study, therefore, aims to investigate the correlation between serum ferritin and hs-CRP levels in patients with IHD, thereby contributing to the growing body of evidence on biomarker-based cardiovascular risk assessment.

## MATERIALS AND METHODS

### Study Design

This investigation was conducted as an observational, cross-sectional study.

### Study Period

The study was carried out over 18 months, from January 2024 to June 2025.

### Study Population

Participants included patients diagnosed with IHD who were admitted to the Department of General Medicine at Agartala Government Medical College and GBP Hospital (AGMC & GBPH).

### Sample Size Determination

The sample size was calculated using the standard formula for cross-sectional studies. The Kish and Leslie (1965) method was applied to obtain the appropriate estimate.

$$n_0 = \frac{Z^2 * p * (1-p)}{e^2}$$

- $n_0$ = Sample size
- $Z^2$ =Standard normal deviate at 5% level of significance is 1.96
- p = Proportion of inpatients diagnosed with IHD at AGMC and GBP Hospital
- $e^2$ = Margin of error at 5%

Ridker PM,[14] reported a prevalence of 7.3%, which was used to determine the sample size of 104 for this study.

**Inclusion Criteria**

Patients aged 18 years and above were eligible for inclusion in the study. The diagnostic criteria applied for myocardial infarction were used to identify and enroll participants.

**ECG Changes:**

**ST-Segment Elevation (STEMI):** Elevation of the J-point observed in two contiguous leads with the following thresholds:

- ≥1 mm (0.1 mV) in all leads other than leads V2-V3.
- ≥2.5 mm (0.25 mV) in men under 40 years, in V2-V3.
- >1.5 mm (0.15 mV) in women in V2-V3.
- ≥2.0 mm (0.20 mV) in men over 40 years in leads V2-V3.[15]

**Pathological Q-Waves:** The appearance of significant Q-waves (≥0.04 seconds in duration or ≥25% of the height of the subsequent R-wave) in two contiguous leads. These Q-waves generally persist for more than 24 hours.[16]

**T-Wave Abnormalities:** Inversion, flattening, or biphasic changes in T-waves occurring in leads corresponding to the ischemic or injured myocardial region.[17]

**ST-Segment Depression:** Notable ST-segment depression (commonly ≥1 mm) across multiple leads, typically indicative of sub-endocardial ischemia.[16]

**Dynamic ECG Changes:** Sequential electrocardiographic alterations demonstrating the progression of ST-segment elevation or depression over time.[18]

**Cardiac Troponin I:** Elevation of cardiac troponin levels above the 99th percentile of a healthy reference population was considered the diagnostic threshold in this study.[19]

- **Stable Angina:** Defined as chest pain or discomfort that is predictable and reproducible, typically triggered by exertion or emotional stress, and relieved within minutes by rest or nitroglycerin. The symptom pattern remains stable in frequency, severity, and duration for at least 6 weeks, highlighting a persistent imbalance between myocardial oxygen supply and demand in the absence of acute plaque rupture.[19]
- **Unstable Angina:** Unstable angina is characterised by chest pain or discomfort arising from inadequate myocardial perfusion. Presentation may include new-onset pain within the preceding two months, a worsening of pre-existing angina, or angina occurring at rest or with minimal exertion. These episodes usually last more than 20 minutes and do not go away with rest or nitroglycerin alone. This condition represents a clinical manifestation of acute coronary syndrome without myocardial infarction and is commonly associated with plaque rupture and thrombosis.[16,20]

**Exclusion Criteria**

The following groups were excluded from participation in the study:

- Individuals younger than 18 years of age.
- Patients diagnosed with iron-deficiency anaemia.

**Data Collection**

Blood samples were obtained from patients diagnosed with IHD. These samples were subsequently analysed to measure serum ferritin and hs-CRP levels.

**Study Tools**

The study utilised the following instruments and procedures:

- **Structured Data Collection Form:** A standardised form was employed to record patient information, including demographic details, medical history, cardiovascular risk factors, clinical findings, and laboratory results.

- **Electrocardiography (ECG):** A conventional 12-lead ECG was performed to assist in diagnosing and classifying ischemic events.
- **Cardiac Troponin I (Trop I):** A semi-quantitative immunochromatographic assay was used to determine cardiac troponin I levels, confirming myocardial injury.
- **Venous Blood Collection Kit:** Under aseptic precautions, 5 mL of venous blood was drawn from each participant using vacutainers for laboratory analysis.
- **Serum Ferritin Measurement:** Serum ferritin concentrations were assessed utilising the enzyme-linked immunosorbent assay (ELISA) method.
- **High-Sensitivity C-Reactive Protein (hs-CRP):** hs-CRP levels were quantified through an immunoturbidimetric assay.

**Data Analysis**

Descriptive statistics (frequencies and percentages) were used to summarise patient characteristics and the distributions of serum ferritin and hs-CRP levels among individuals with IHD. Inferential statistical methods were used to examine correlations between these biomarkers in patients presenting with myocardial infarction. Multiple regression analysis was used to identify independent predictors of serum ferritin and hs-CRP levels in this cohort.

**Ethical Considerations**

The study implemented ethical safeguards by obtaining informed consent from all participants, maintaining the strict confidentiality of patient information, and adhering to established institutional and international ethical guidelines throughout the research process.

**RESULTS**

The study comprised 104 patients diagnosed with IHD, in whom serum ferritin and hs-CRP levels were assessed to explore their interrelationships. The participants had a mean age of 60.6 ± 11.9 years, with ages ranging from 41 to 79 years. The age distribution revealed that 23 individuals (22.1%) were between 40 and 50 years, while 18 cases (17.3%) fell within the 51–60 years group. The largest proportion was observed in the 61–70-year age group, comprising 37 patients (35.6%). Additionally, 26 participants (25.0%) were aged above 70 years (Figure 1).

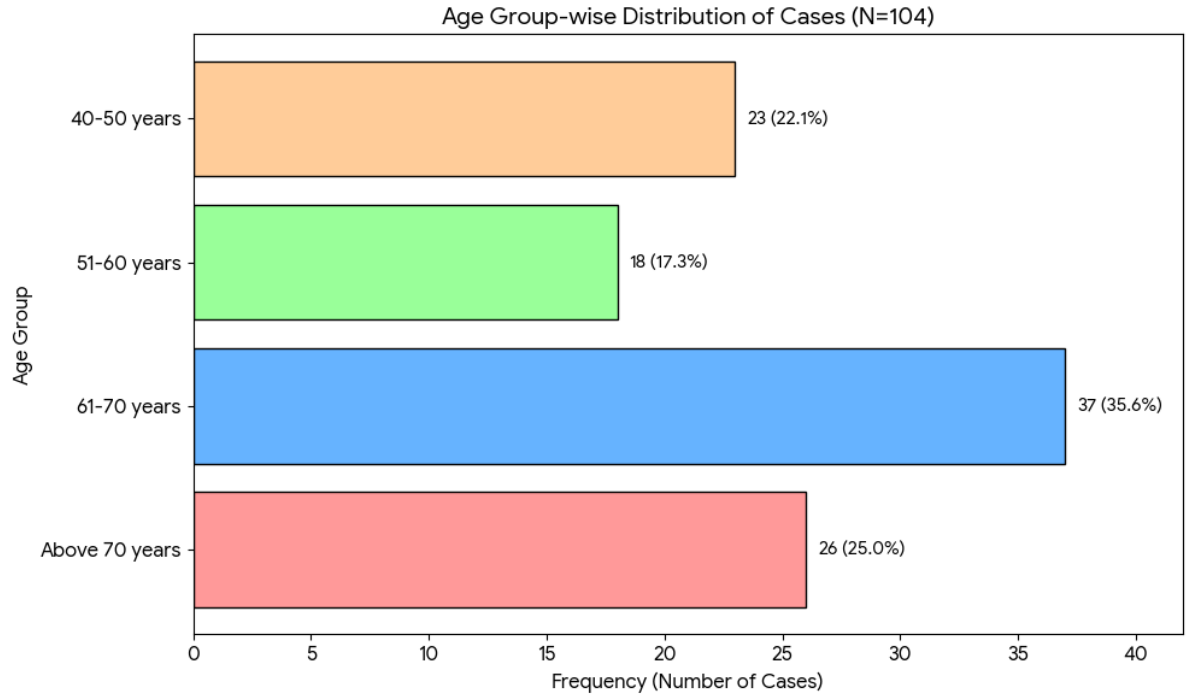


**Figure 1:** Age Group-wise Distribution of the Cases

Figure 2 indicates that, in terms of gender distribution, 76 were male (73.1%), and 28 were female (26.9%).

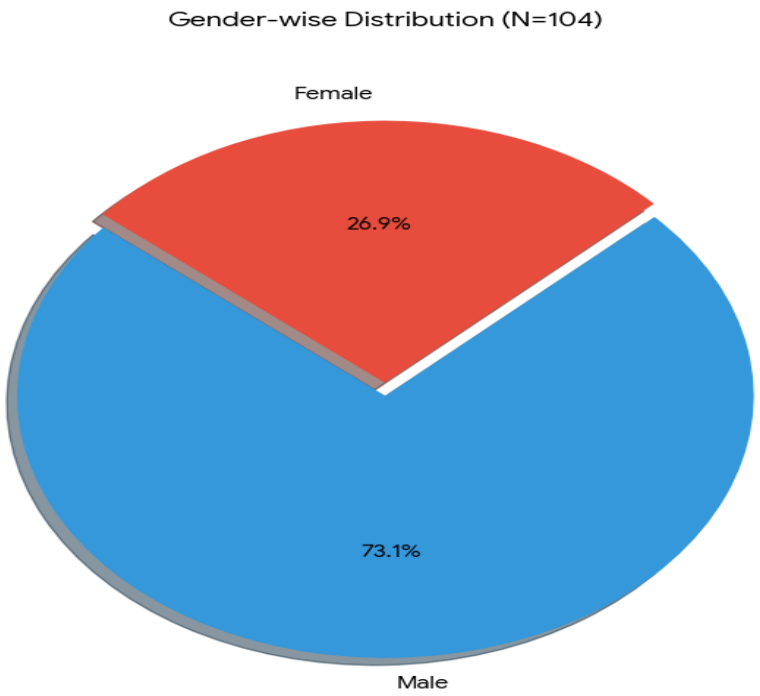


**Figure 2:** Gender-wise Distribution of the Cases

When the age distribution was examined by gender, clear differences emerged. Among females, the majority of cases were concentrated in the 61–70-year group (46.4%), followed by those aged 70 and above (32.1%). Only a small proportion of women fell into the younger age brackets of 40–50 years (14.3%) and 51–60 years (7.1%). In contrast, male cases were more evenly distributed across age groups: 25.0% were between 40–50 years, 21.1% were in the 51–60 years range, 31.6% were in the 61–70 years group, and 22.4% were above 70 years (Figure 3).

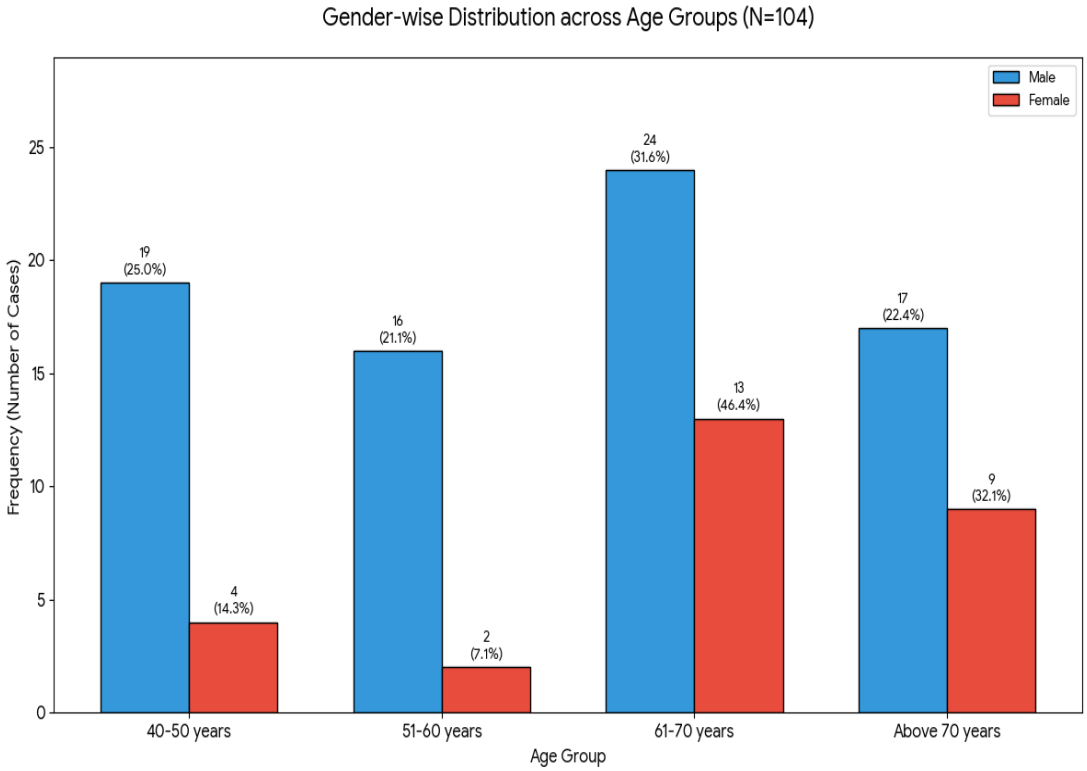


**Figure 3:** Gender-wise Distribution concerning Age Group

The socio-demographic characteristics of the study participants are summarised in Table 1, which presents details on religion, educational attainment, occupation, and marital status.

**Table 1**: Socio-demographic profile of the cases (N=104)

| Socio-demographic profile | Frequency | Percent |
|---|---|---|
| **Religion:** | | |
| **Hindu** | 46 | 44.2 |
| **Muslim** | 50 | 48.1 |
| **Christian** | 8 | 7.7 |
| **Educational level:** | | |
| **Graduate & above** | 58 | 55.7 |
| **Upto secondary** | 18 | 17.3 |
| **Upto primary level** | 28 | 26.9 |
| **Occupation:** | | |
| **Employed** | 26 | 25.0 |
| **Self employed** | 17 | 16.3 |
| **Housewife** | 17 | 16.3 |
| **Un-employed** | 44 | 42.3 |
| **Marital status:** | | |
| **Married** | 81 | 77.9 |
| **Widowed** | 23 | 22.1 |

More than half of the patients (52.9%) presented with ST-elevation myocardial infarction (STEMI), making it the most common clinical type observed. Non-STEMI accounted for 22.1% of cases, while unstable angina was reported in 16.3%. A smaller proportion (8.7%) had stable angina (Figure 4).

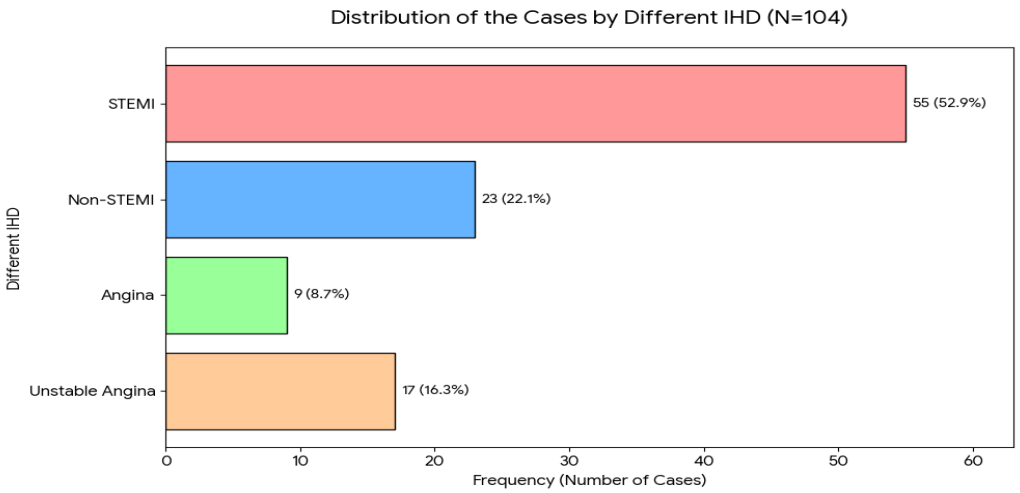


**Figure 4:** Distribution of Cases by IHD Type

More than half of the patients (51.0%) reported symptoms for only 1 day, while 38.4% experienced illness for 2 to 5 days. A smaller proportion (10.6%) had a duration of more than 5 days (Figure 5).

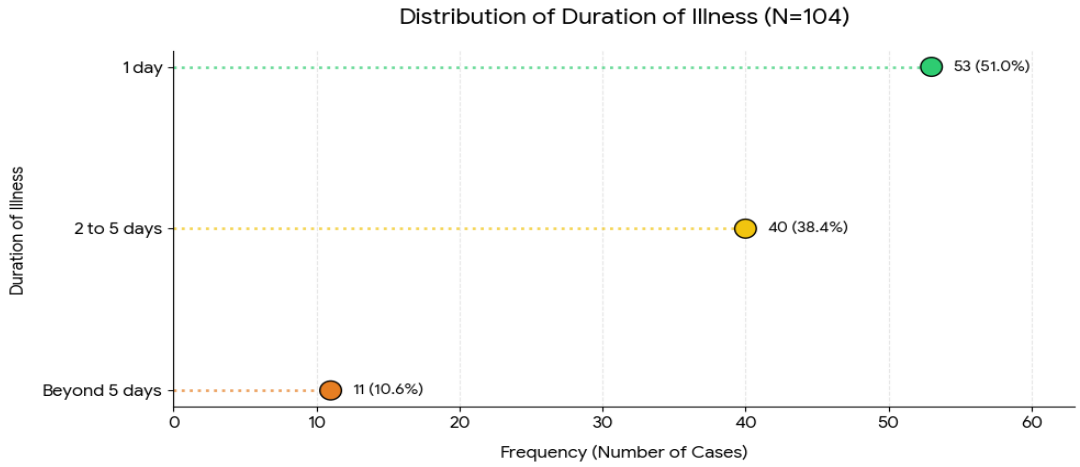


**Figure 5:** Distribution of Duration of Illness

The presence of major co-morbidities is summarised in Table 2. Hypertension was the most prevalent, affecting 79.8% of patients, followed by diabetes in 48.1%. Chronic kidney disease was reported in 14.4% of cases, while chronic obstructive pulmonary disease (COPD) was present in 7.7%.

**Table 2:** Distribution of cases by presence of major co-morbidities (N=104)

| Major co-morbidities | Frequency | Percent |
|---|---|---|
| Hypertension | 83 | 79.8 |
| Diabetes | 50 | 48.1 |
| Chronic kidney disease | 15 | 14.4 |
| COPD | 8 | 7.7 |

The distribution of lifestyle risk factors reveals a concerning pattern of unhealthy behaviours. More than half of the participants (55.8%) reported smoking, while nearly one-third (28.8%) consumed alcohol. The most striking finding, however, is the predominance of sedentary behaviour, with 77.9% of individuals reporting no physical activity. Only a small proportion were physically active (16.3%) or moderately active (5.8%), underscoring very limited engagement in health-promoting practices (Table 3).

**Table 3**: Distribution of cases by lifestyle risk factors (N=104)

| Risk lifestyles | Frequency | Percent |
|---|---|---|
| Smoking | 58 | 55.8 |
| Alcohol | 30 | 28.8 |
| Physical activity:<br>Active<br>Moderate<br>Sedentary | <br>17<br>6<br>81 | <br>16.3<br>5.8<br>77.9 |

The analysis of vital parameters shows that the mean heart rate was 75.5 ± 11.4 beats per minute, with a median of 75 and a range of 60-99. Systolic blood pressure (SBP) averaged 140.3 ± 16.1 mmHg, with a median of 139 mmHg and a range of 110–170 mmHg. Similarly, diastolic blood pressure (DBP) averaged 83.5 ± 10.1 mmHg, with a median of 86 mmHg and a range of 70–100 mmHg. The mean body mass index (BMI) was 24.1 ± 3.1 kg/m², with a median of 23.2 and a range of 18.8–29.9 (Table 4).

**Table 4**: Distribution of the cases' vital parameters (N=104)

| Vital parameters | Mean, SD | Median | Ranges |
|---|---|---|---|
| Heart rate /min | 75.5 ± 11.4 | 75 | 60-99 |
| SBP (mmHg) | 140.3 ± 16.1 | 139 | 110-170 |
| DBP (mmHg) | 83.5 ± 10.1 | 86 | 70-100 |
| BMI (kg/m2) | 24.1 ± 3.1 | 23.2 | 18.8-29.9 |

Table 5 compares serum ferritin and hs-CRP levels across different forms of IHD, revealing important differences in inflammatory and iron-storage markers. Patients with unstable angina showed the highest mean ferritin levels (405.1 ± 107.4 ng/mL) and elevated hs-CRP (3.9 ± 1.3 mg/L). Non-STEMI and STEMI cases had comparable ferritin values (379.4 ± 127.6 ng/mL and 370.3 ± 65.1 ng/mL, respectively) and similar hs-CRP levels (3.09 ± 1.37 mg/L and 3.02 ± 1.25 mg/L, respectively). In contrast, stable angina patients had lower ferritin (318.8 ± 136.4 ng/mL) and markedly reduced hs-CRP (1.4 mg/L).

**Table 5**: Comparison of mean serum ferritin and hs-CRP across IHD subtypes (N=104)

| Different IHD | Serum ferritin (Mean, SD/median) | Serum hs-CRP (Mean, SD/median) |
|---|---|---|
| Non-STEMI | 379.4 ± 127.6 | 3.09 ± 1.37 |
| STEMI | 370.3 ± 65.1 | 3.02 ± 1.25 |
| Angina | 318.8 ± 136.4 | 1.4 |
| Unstable Angina | 405.1 ± 107.4 | 3.9 ± 1.3 |

In Figure 6, the correlation analysis between serum ferritin and hs-CRP indicates a weak, statistically nonsignificant association. The Pearson correlation coefficient was 0.101, indicating only a very slight positive association between ferritin and hs-CRP levels (p=0.306).

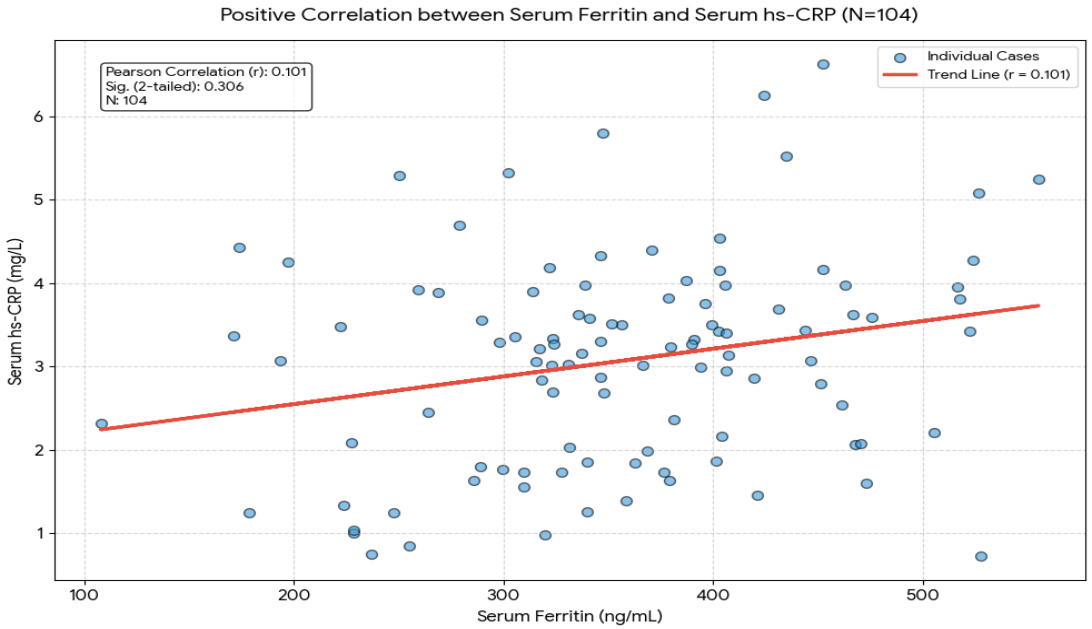


**Figure 6:** Scatter Plot: Correlation between Serum Ferritin and Serum hs-CRP (r=0.101, p=0.306)

Serum ferritin and hs-CRP levels demonstrated notable elevations, underscoring the role of iron metabolism and inflammation in disease progression. Elevated ferritin (>300 ng/mL) was observed in 78 patients (75%), with a mean value of 373.6 ± 100.5 ng/mL and a median of 366 ng/mL, ranging from 159.5 to 659.5 ng/mL. In parallel, serum hs-CRP levels averaged 3.04 ± 1.36 mg/L, with a median of 2.92 mg/L and a range of 0.64 to 6.68 mg/L. Notably, 46 patients (44.2%) had hs-CRP values above 3 mg/L (Table 6).

**Table 6**: Distribution of the IHD cases by serum ferritin and serum hs-CRP (N=104)

| Biochemical parameters | Mean, SD/ frequency | Median / percent | Ranges |
|---|---|---|---|
| **Serum ferritin**<br>**High ferritin (>300ng/mL)** | 373.6 ± 100.5<br>78 | 366<br>75% | 159.5 to 659.5 |
| **Serum hs-CRP**<br>**Above 3 level** | 3.04 ± 1.36<br>46 | 2.92<br>44.2% | 0.64 to 6.68 |

## DISCUSSION

Recent evidence confirms that both serum ferritin and hs-CRP are elevated in patients with IHD, but they reflect distinct mechanisms—iron metabolism dysregulation and systemic inflammation, respectively. The present study's findings of high ferritin and hs-CRP levels, particularly in unstable angina, align with recent global research linking these biomarkers to poor prognosis and prolonged hospitalisation.

### Elevated Ferritin and Cardiovascular Risk

Ferritin was elevated in 75% of patients in this study, consistent with recent reports. Liu et al. (2023) found that high ferritin levels in IHD patients admitted to intensive care units were independently associated with poor prognosis and increased mortality, suggesting ferritin as a prognostic biomarker in acute settings.[21] Similarly, Aisikeer et al. (2025) demonstrated that elevated ferritin in coronary artery disease patients with heart failure predicted adverse outcomes, including rehospitalisation and cardiovascular death.[22] These findings support the current study's observation that elevated ferritin levels are common in IHD and may contribute to oxidative stress and endothelial dysfunction, thereby accelerating atherosclerosis.

Ferritin presents interpretive challenges due to its dual role as both an iron-storage protein and an acute-phase reactant. Hyperferritinaemia may reflect true iron overload or may be driven by systemic inflammation, independent of iron status. Huang et al. (2025) showed that ferritin is most useful as part of a group of biomarkers for precise cardiovascular diagnosis, especially when considered with other inflammatory markers.[23] Therefore, evaluating ferritin in combination with hs-CRP is essential, rather than considering ferritin in isolation.

### hs-CRP as a Marker of Residual Inflammatory Risk

The study found that 44.2% of patients had hs-CRP levels above 3 mg/L, a threshold associated with heightened cardiovascular risk. This finding is consistent with Ridker's research, which established that hs-CRP levels above 3 mg/L are associated with a significantly increased risk of future cardiovascular events, thereby validating the threshold observed in the present study.[24] Earlier studies, including the CANTOS trial by Ridker et al. (2017), have confirmed hs-CRP as a

key biomarker for cardiovascular risk prediction, highlighting its role in residual inflammatory risk even after lipid control.[25] Elevated hsCRP in unstable angina patients in the present study mirrors earlier findings, such as those by Liuzzo et al. (1994), who demonstrated that higher hsCRP levels in acute coronary syndromes were associated with plaque instability and worse clinical outcomes.[26]

Inflammation-driven plaque destabilisation has become an important therapeutic target. Katamine et al. (2025) demonstrated that elevated hs-CRP levels are closely associated with plaque vulnerability and adverse events in patients with stable coronary disease, supporting the use of hs-CRP in guiding precision therapies to reduce inflammatory burden in IHD.[27] Therefore, hs-CRP remains a reliable marker for identifying patients at risk of recurrent events, even when lipid levels are optimally managed.

**Weak Correlation Between Ferritin and hs-CRP**

The present study observed only a weak, nonsignificant correlation between ferritin and hs-CRP (p=0.306). This suggests that while both markers are elevated, they capture different aspects of IHD pathophysiology. Urbanski et al. (2024) proposed the ferritin/CRP ratio as a more reliable indicator of iron deficiency in inflammatory states, highlighting the limitations of interpreting ferritin alone.[10] The lack of correlation here supports the idea that ferritin elevation may reflect oxidative stress and iron metabolism, whereas hs-CRP is a direct marker of systemic inflammation.

Inflammation-driven plaque destabilisation has gained recognition as a significant therapeutic target. Libby (2021) emphasized that hs-CRP, together with other inflammatory biomarkers, is insufficient when used in isolation. Instead, a comprehensive panel of markers is required to adequately reflect the complexity of plaque progression and systemic cardiovascular risk.[28]

**Implications for Clinical Practice**

- Elevated ferritin levels are indicative of oxidative stress and iron dysregulation, both of which may contribute to vascular impairment.
- Increased hs-CRP levels reflect systemic inflammation and plaque instability, serving as predictors of acute coronary events.
- Combined assessment of ferritin and hs-CRP provides a more comprehensive risk profile, particularly for distinguishing between stable and unstable forms of ischemic heart disease.
- Recent studies (2023–2025) confirm the prognostic significance of both biomarkers. However, the weak correlation between ferritin and hs-CRP underscores the importance of utilising integrated biomarker panels rather than relying on single parameters.

**Limitations**

- Single-centre study, limiting generalizability.
- Cross-sectional design precludes causal inference.
- Relatively small sample size.
- Lack of longitudinal follow-up to assess outcomes such as mortality or recurrent events.
- Potential confounding from comorbidities (e.g., diabetes, CKD) not fully adjusted in analysis.

## CONCLUSION

The findings indicate that ferritin functions as both an iron-storage protein and an acute-phase reactant. Additionally, hs-CRP serves as a marker of systemic inflammation. Elevated concentrations of both biomarkers were frequently observed in patients with IHD, especially those with unstable angina. However, the weak correlation between these biomarkers suggests that they reflect different aspects of disease pathophysiology. Integrating ferritin and hs-CRP into biomarker panels may enhance cardiovascular risk stratification and guide precision therapies targeting both iron metabolism and inflammation. Future multicentre, longitudinal studies are warranted to validate these findings and explore their prognostic utility in diverse populations.

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