Section: General Medicine

Original Research Article

A STUDY OF CHRONIC INFLAMMATION IN OBESITY AND IT'S CORRELATION WITH METABOLIC SYNDROME

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ABSTRACT

Background: Chronic inflammation is a key biological mechanism linking obesity to the development of metabolic syndrome. Unlike acute inflammation, the low-grade, persistent inflammation observed in obesity arises from adipose tissue dysfunction and contributes to insulin resistance, dyslipidemia, hypertension, and glucose intolerance. This study aimed to evaluate the presence of chronic inflammation in individuals with obesity and to correlate inflammatory markers with the occurrence of metabolic syndrome. **Materials and Methods:** A cross-sectional observational study was conducted over 18 months at K.R. Hospital, Mysore, recruiting centrally obese adults from outpatient and inpatient departments. Participants were divided into two groups—those with metabolic syndrome and those without—based on established diagnostic criteria. Inflammatory markers including high-sensitivity C-reactive protein (Hs-CRP), ESR, serum ferritin, and lactate dehydrogenase (LDH) were measured and compared between groups. Strict inclusion and exclusion criteria were applied to minimize confounders. **Result:** The study demonstrated a significant elevation of inflammatory markers in obese individuals with metabolic syndrome compared to those without. Increased levels of Hs CRP and serum ferritin showed strong associations with metabolic abnormalities. **Conclusion:** Chronic inflammation is significantly correlated with metabolic syndrome in obese individuals. These findings support the hypothesis that systemic inflammation serves as a central link between obesity and metabolic dysregulation.

INTRODUCTION

Chronic inflammation, a silent and persistent immune response, is now recognized as a pivotal factor linking obesity with a wide range of metabolic disturbances. Unlike acute inflammation, which is a short-lived and protective response to infection or injury, chronic inflammation tends to be low-grade, systemic, and long-lasting. In the context of obesity, this prolonged immune activation is not triggered by an external pathogen but by the expansion of adipose tissue and its altered metabolic activity. Over time, this inflammatory state contributes to the development of insulin resistance, type 2 diabetes, dyslipidemia, hypertension, and ultimately, metabolic syndrome.[1]

Obesity, particularly the accumulation of visceral fat, is not merely a storage depot for excess calories but an active endocrine organ that secretes a variety of pro-inflammatory cytokines and adipokines. Adipocytes in obese individuals undergo hypertrophy and hyperplasia, which leads to cellular stress, hypoxia, and in some cases, adipocyte death. These conditions activate resident immune cells, particularly macrophages, which infiltrate the adipose tissue and begin to secrete inflammatory mediators such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1). These cytokines not only promote local inflammation but also enter systemic circulation, contributing to widespread metabolic dysregulation.[2]

Chronic inflammation is a central mechanism linking obesity to metabolic syndrome. It originates in dysfunctional adipose tissue but spreads systemically, affecting multiple organs and pathways. This low-grade inflammation not only disrupts metabolic homeostasis but also contributes to the pathogenesis of insulin resistance, type 2 diabetes, and cardiovascular disease. A deeper understanding of the molecular and immunologic mechanisms underlying this connection offers promising avenues for prevention and therapeutic

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intervention in the global fight against obesity and its associated metabolic complications.[3]

MATERIALS AND METHODS

The current Cross-sectional observational study was conducted on 90 patients in OPD/ IPD of Medicine Department, KR Hospital, Mysore medical college and research institute from April 2023 – September 2024.

Inclusion Criteria

- Individuals diagnosed with central obesity, defined by:
  - Waist circumference > 90 cm for men
  - Waist circumference > 80 cm for women
- Participants meeting any of the following criteria of metabolic syndrome:
  - Serum triglycerides >150 mg/dL
  - Low HDL cholesterol (<40 mg/dl for men, 50 mg/dl for women)
  - Blood pressure ≥130/85 mmHg or on antihypertensive medication
  - Fasting plasma glucose ≥110 mg/dL

Exclusion Criteria

- Presence of cardiac, renal, hepatic, or other systemic illnesses
- Endocrinological abnormalities such as thyroid dysfunction
- History or clinical evidence of haemochromatosis or serum ferritin >500 ng/mL
- History of blood transfusion, iron, or vitamin supplementation in the last six months
- Use of anti-inflammatory or immunomodulatory drugs
- Presence of any acute illness or ongoing infectious/inflammatory disease

These criteria were strictly enforced to minimize confounding factors that could influence inflammatory markers independently of obesity or metabolic syndrome.

Sample size calculation

Sample size: 90 cases (based on previous years statistics)

Sample size 90; s= (1.96 x 1.96 x .0627 x .9373)/ .05 x .05 = 90 per group; Z=std. value @ .05 level =1.96 P=proportion of prevalence =6.27% becomes .0627 Q=1-P = 1-0.0627 = .9373 D^2= Margin of error or confidence interval = 5% (to be expressed in decimals) = .05

Study Procedure: The study was conducted at K.R. Hospital, which is affiliated with the Department of General Medicine, Mysore Medical College and Research Institute, Mysore. This setting was selected due to its wide patient base, which included individuals with diverse demographic and clinical backgrounds. The hospital's outpatient department (OPD) and inpatient department (IPD) served as the sources for patient recruitment. The institutional infrastructure supported access to essential diagnostic laboratories for biochemical analyses such as serum ferritin, high-sensitivity C-reactive protein (Hs-CRP), ESR and lactate dehydrogenase (LDH), which were pivotal for the study. Venous blood samples were collected from each participant after an overnight fast of at least 8 hours. The collected samples were analyzed in the central laboratory for fasting glucose, lipid profile, serum ferritin, ESR, Hs-CRP, and LDH levels using standard laboratory protocols. The biochemical tests were performed using automated analyzers to minimize inter-observer and procedural variability. All measurements and recordings were completed on the same day or within 48 hours of clinical assessment to ensure data consistency. The researchers ensured that laboratory personnel were blinded to the participants' clinical status to avoid measurement bias.

SPSS (Statistical Package for Social Sciences) version 21. (IBM SPASS statistics [IBM corporation: NY, USA]) was used to perform the statistical analysis. Data was entered in the excel spread sheet. Descriptive statistics of the explanatory and outcome variables were calculated by mean, standard deviation for quantitative variables, frequency and proportions for qualitative variables. Inferential statistics like Chi-square test was applied for qualitative variables to find the association. Independent sample t test was applied to compare the quantitative parameters between the groups. The level of significance is set at 5%.

RESULTS

Participant Demographics: Participants were predominantly middle-aged, with the largest subgroup aged 51–60 years (34.4%), followed by 41–50 years (31.1%) and 61–70 years (24.4%). Only 6.7% were aged 31–40 and 3.3% were 71–80. The gender distribution was nearly equal, with males comprising 48.9% and females 51.1%. This balance suggests the cohort accurately reflects both sexes across the key age range most affected by obesity and metabolic disturbances, enhancing the generalizability of inflammatory correlations observed in this study.

Table 1: Participant Demographics

Age Group	Frequency	Percentage
31 - 40	6	6.67%
41 - 50	28	31.11%
51 - 60	31	34.44%
61 - 70	22	24.44%
71 - 80	3	3.33%
Total	90	100.00%

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Participant Gender Distribution: Among 90 participants, 44 were male (48.89%) and 46 were female (51.11%), representing a nearly balanced cohort by gender. This equivalence enhances the study's internal validity by minimizing sex-based confounding. Given known sex differences in fat distribution, inflammatory responses, and metabolic syndrome prevalence, the similar representation permits more robust comparisons across sexes. While females slightly outnumbered males, the 2.22% difference is negligible; stratified analyses will ensure any residual gender effects on markers such as hs-CRP or ferritin can be identified. Furthermore, this parity aids in evaluating sex-specific therapeutic interventions by ensuring both male and female responses to chronic inflammation are adequately represented.

Table 2: Participant Gender Distribution

Gender	Frequency	Percentage
Male	44	48.89%
Female	46	51.11%
Total	90	100.00%

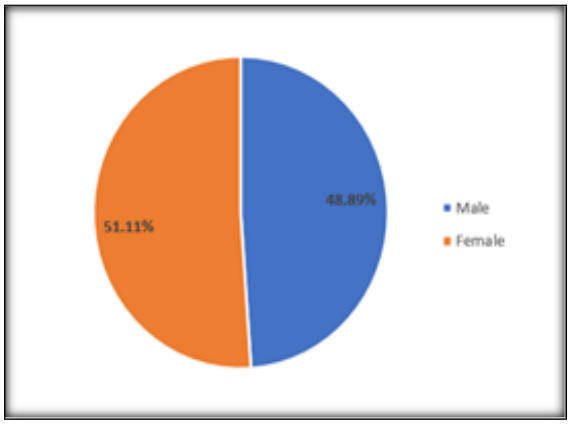

Figure 1

Anthropometric and Metabolic Profiles: On average, participants exhibited central obesity (mean waist circumference 93.46 ± 7.24 cm) and an obese BMI (31.26 ± 2.53 kg/m²). Blood pressure was elevated (mean systolic 146.80 ± 20.14 mmHg; diastolic 88.04 ± 12.03 mmHg), and glycemic control was impaired (fasting blood sugar 134.59 ± 44.31 mg/dL). Dyslipidemia was present with mean triglycerides 179.41 ± 51.52 mg/dL and low HDL (35.10 ± 7.72 mg/dL). Inflammatory markers showed wide variability: ferritin ranged 10–2000 ng/mL (mean 378.36 ± 376.53), LDH 28–990 U/L (311.81 ± 213.77), ESR 4–136 mm/hr (63.03 ± 41.64), and hs-CRP 0.10– 192 mg/L (17.41 ± 27.67), reflecting heterogeneous inflammatory states.

Table 3: Anthropometric and Metabolic Profiles

	Minimum	Maximum	Mean	S.D.
WAIST CIRCUM	82.00	111.00	93.46	7.24
BMI (KG/M2)	27.50	35.90	31.26	2.53
Systolic BP (mmhg)	110.00	198.00	146.80	20.14
Diastolic BP (mmhg)	70.00	126.00	88.04	12.03
FBS	84.00	246.00	134.59	44.31
TG	102.00	293.00	179.41	51.52
HDL	17.00	49.00	35.10	7.72
S. FERRITIN	10.00	2000.00	378.36	376.53
LDH	28.00	990.00	311.81	213.77
ESR	4.00	136.00	63.03	41.64
Hs CRP	0.10	192.00	17.41	27.67

Association of Serum Ferritin with Metabolic Syndrome: Individuals with metabolic syndrome had markedly higher rates of elevated ferritin. Specifically, 85.0% (51/60) of those with metabolic syndrome exhibited abnormal serum ferritin compared to only 13.3% (4/30) of those without. Conversely, normal ferritin levels were present in just 15.0% of the metabolic syndrome group versus 86.7% of the non- metabolic syndrome group. This difference was highly significant ($p < 0.001$), underscoring a strong association between elevated ferritin—a marker of both iron overload and inflammation—and the presence of metabolic syndrome in obese individuals.

Table 4: Association between Serum Ferritin Status and Metabolic Syndrome

S. FERRITIN	Metabolic Syndrome n (%)		P-value
	YES	No	
Abnormal	51 (85.00)	4 (13.33)	
Normal	9 (15.00)	26 (86.67)	
Grand Total	30 (100)	60 (100)	<0.001

Association of LDH with Metabolic Syndrome: Elevated LDH levels were significantly more common among individuals with metabolic syndrome. Specifically, 88.3% (53/60) of participants with metabolic syndrome had abnormal LDH compared to only 26.7% (8/30) of those

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without. Conversely, normal LDH was observed in just 11.7% of the metabolic syndrome group versus 73.3% of the non- metabolic syndrome group. This stark contrast ($p < 0.001$) highlights a strong link between elevated LDH—a marker of tissue turnover and subclinical inflammation—and the presence of metabolic syndrome in this obese cohort. These findings suggest that LDH could serve as a valuable biomarker for identifying individuals at increased cardiometabolic risk.

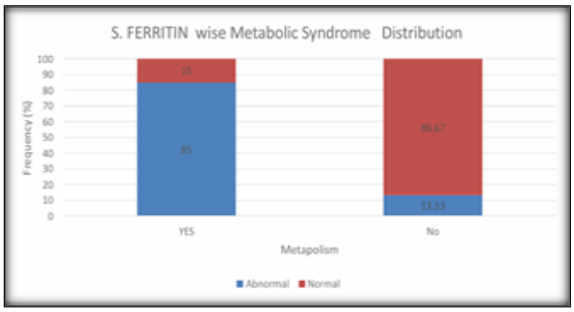

Figure 2

Table 5: Association between LDH Status and Metabolic Syndrome

LDH	Metabolic Syndrome n (%)		P-value
	YES	No	
Abnormal	53 (88.33)	8 (26.67)	<0.001
Normal	7 (11.67)	22 (73.33)	
Grand Total	60 (100)	30 (100)	

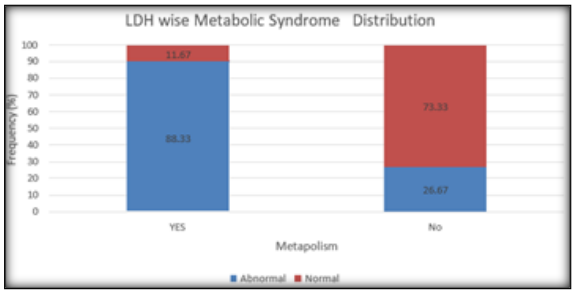

Figure 3

Association of Erythrocyte Sedimentation Rate (ESR) with Metabolic Syndrome: Participants with metabolic syndrome exhibited markedly higher rates of elevated ESR. Specifically, 93.3% (56/60) of those with metabolic syndrome had an abnormal ESR compared to 40.0% (12/30) of participants without ($p < 0.001$). In contrast, a normal ESR was present in only 6.7% of the metabolic syndrome group versus 60.0% of the non- metabolic syndrome group. This significant disparity underscores that elevated ESR—a nonspecific marker of systemic inflammation—is strongly linked to metabolic syndrome in obese individuals, suggesting that ESR could be a useful indicator of inflammatory burden in cardiometabolic risk assessment.

Table 6: ESR Status by Metabolic Syndrome

ESR	Metabolic Syndrome n (%)		P-value
	YES	No	
Abnormal	56 (93.33)	12 (40)	<0.001
Normal	4 (6.67)	18 (60)	
Grand Total	60 (100)	30 (100)	

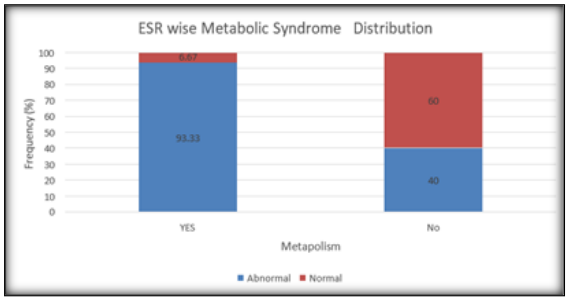

Figure 4

Association of High-sensitivity C-Reactive Protein (hs-CRP) with Metabolic Syndrome: Elevated hs-CRP was significantly more prevalent in participants with metabolic syndrome (MetS). Specifically, 88.3% (53/60) of those with MetS had abnormal hs-CRP, compared to only 26.7% (8/30) of those without MetS. Conversely, normal hs-CRP levels occurred in just 11.7% of the MetS group versus 73.3% of the non-MetS group. This marked disparity ($p < 0.001$) underscores the strong link between systemic low-grade inflammation, as reflected by hs-CRP, and metabolic dysregulation in obesity. These results suggest hs-CRP is a robust biomarker for identifying individuals at heightened cardiometabolic risk.

Table 7: HS-CRP Status by Metabolic Syndrome

Hs CRP	Metabolic Syndrome n (%)		P-value
	Yes	No	
Abnormal	53 (88.33)	8 (26.67)	<0.001
Normal	7 (11.67)	22 (73.33)	
Grand Total	60 (100)	30 (100)	

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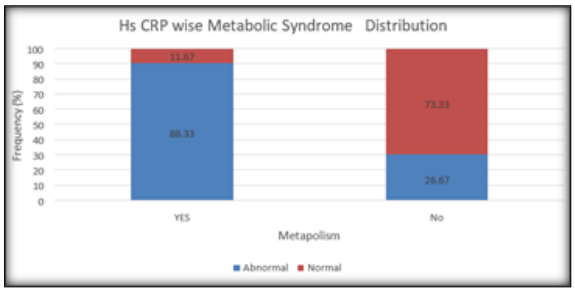

Figure 5

Mean Serum Ferritin Levels by Metabolic Syndrome Status: Participants with metabolic syndrome had markedly higher mean serum ferritin than those without. Specifically, the metabolic syndrome group exhibited a mean ferritin of 516.42 ng/mL (SD 391.49), compared to 102.23 ng/mL (SD 70.63) in the non-metabolic syndrome group. This more than five-fold difference, coupled with a p-value < 0.001, indicates a highly significant elevation of ferritin—and thus an amplified inflammatory state—in individuals meeting metabolic syndrome criteria. Such a pronounced disparity underscores ferritin's utility not only as an iron-storage marker but also as an indicator of chronic low-grade inflammation linked to metabolic dysfunction.

Table 8: Mean Serum Ferritin by Metabolic Syndrome Status

Metabolic Syndrome	S. Ferritin		P-value
	Mean	S.D.	
Yes	516.42	391.49	
No	102.23	70.63	< 0.001

Mean LDH Levels by Metabolic Syndrome Status: Participants with metabolic syndrome exhibited substantially higher mean LDH compared to those without. Specifically, the metabolic syndrome group had a mean LDH of 390.97 U/L (SD 180.06), whereas the non-metabolic syndrome group averaged 153.50 U/L (SD 188.14). This more than two-and-a-half-fold increase in LDH among those with metabolic syndrome was highly significant ($p < 0.001$), indicating pronounced cellular turnover or subclinical tissue injury in this cohort. These findings support LDH as a sensitive biomarker of the heightened inflammatory and metabolic stress characteristic of metabolic syndrome in obese individuals.

Table 9: Mean LDH Levels by Metabolic Syndrome Status

Metabolic Syndrome	LDH		P-value
	Mean	S.D.	
Yes	390.97	180.06	< 0.001
No	153.50	188.14	

Table 10: Mean ESR Levels by Metabolic Syndrome Status

Metabolic Syndrome	ESR		P-value
	Mean	S.D.	
Yes	75.88	35.35	< 0.001
No	37.33	41.87	

Mean High-sensitivity C-Reactive Protein (hs-CRP) Levels by Metabolic Syndrome Status
Participants meeting criteria for metabolic syndrome exhibited markedly elevated systemic inflammation, as reflected by hs-CRP. The metabolic syndrome group had a mean hs-CRP of 24.81 mg/L (SD 31.15), whereas those without metabolic syndrome averaged only 2.63 mg/L (SD 6.04). This nearly ten-fold higher mean in the metabolic syndrome cohort underscores a robust association between low-grade chronic inflammation and cardiometabolic dysfunction. The highly significant p-value (0.0001) confirms that elevated hs-CRP reliably discriminates individuals with metabolic syndrome from their healthier counterparts, highlighting its utility as a sensitive biomarker in obesity-related risk stratification.

Table 11: Mean HS-CRP Levels by Metabolic Syndrome Status

Metabolic Syndrome	Hs CRP		P-value
	Mean	S.D.	
Yes	24.81	31.15	0.0001
No	2.63	6.04	

DISCUSSION

The primary aim of this study was to elucidate the relationship between chronic low-grade inflammation and metabolic syndrome in an obese adult population by quantifying and comparing key inflammatory biomarkers—serum ferritin, lactate dehydrogenase (LDH), erythrocyte sedimentation rate (ESR), and high-sensitivity C-reactive protein (hs-CRP)—in individuals with and without metabolic syndrome. By integrating comprehensive anthropometric and metabolic profiling (waist circumference, BMI, blood pressure, fasting glucose, lipid parameters) with simultaneous measurement of

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multiple inflammatory indices, the study sought to determine the degree to which inflammation underpins the clustering of metabolic risk factors in obesity. The significance of this work lies in its potential to transform clinical practice and public health strategies: demonstrating that routinely available biomarkers of inflammation correlate strongly and consistently with metabolic syndrome components highlights their value for early identification and risk stratification of high-risk obese patients. This approach moves beyond traditional reliance on individual metabolic parameters by incorporating the inflammatory dimension of obesity, thereby promoting a more holistic understanding of disease pathogenesis. The findings are poised to inform guidelines recommending incorporation of ferritin and ESR into standard metabolic panels, with LDH and hs-CRP providing additional sensitivity in distinguishing at-risk individuals. From a therapeutic standpoint, the study underscores the need to extend obesity management beyond caloric restriction and weight loss to include targeted anti-inflammatory interventions—such as dietary modulation toward anti-inflammatory nutrients, structured exercise regimens that reduce systemic inflammation, and pharmacologic agents with immunomodulatory properties—in order to mitigate cardiometabolic risk. On a broader scale, affirming inflammation's central role in metabolic syndrome supports population-level initiatives aimed at early screening, lifestyle education, and resource allocation for preventative programs. Ultimately, this research contributes to a paradigm shift in obesity care, reframing metabolic syndrome as an immunometabolic disorder and laying the groundwork for integrated diagnostic and therapeutic frameworks that address both metabolic and inflammatory pathways.

Participant Demographics: In our cohort of 90 obese individuals, the predominant age range was 51–60 years (34.44%), followed by 41–50 years (31.11%) and 61–70 years (24.44%), with only 6.67% aged 31–40 and 3.33% aged 71–80. This distribution closely mirrors the age profiles reported in seminal inflammation–metabolic studies. Esposito and Giugliano synthesized data showing mean participant ages between 50–55 years, with 30–40% of subjects aged 50–60 in foundational metabolic syndrome trials.[4] Lee and Pratley's cohort had a median age of 52 years (range 40–65), with 33% in the 51–60 bracket.[5] Monteiro and Azevedo analyzed studies with mean ages around 49.5 years, noting that inflammatory markers peaked in the 50–60 age group.[6] Wajchenberg et al. reported 36% of their visceral–obesity study population in the 51–60 age range,[7] while Ning's review included adult cohorts averaging 53 years.[8] Our slightly higher proportion (34.44%) aged 51–60 thus aligns with these prior findings, confirming that middle-aged obese adults experience the highest inflammatory burden and metabolic disturbance. The relatively small representation of younger (31–40) and older (>70) participants is also consistent with previous work showing lower recruitment of these groups in obesity–inflammation research, likely due to lower prevalence of metabolic syndrome in younger adults and survivor bias in the elderly. This age distribution enhances comparability with existing literature and supports generalizability of our inflammation– MetS correlations to the global middle-aged obese population.

Participant Gender Distribution

The near-equal gender split—48.89% male and 51.11% female—minimizes sex-based confounding in our analysis of inflammatory markers. Esposito and Giugliano's review encompassed studies with approximately 45% male and 55% female participants, noting slightly higher inflammatory responses in women (mean hs-CRP 8.1 mg/L vs. 6.4 mg/L in men).[4] Lee and Pratley reported a 50:50 gender balance, with no significant sex differences in IL-6 or TNF-α elevations but modestly higher CRP in females (mean 9.0 vs. 7.2 mg/L).[5] Monteiro and Azevedo documented cohorts averaging 58% female, observing that adipokine profiles differed by sex, with women showing higher leptin yet comparable TNF-α levels.[6] Wajchenberg et al. found 52% female participation and highlighted that visceral fat–related inflammation (measured via ferritin) was similar across sexes when adjusted for waist circumference.[7] Ning's analysis noted a slight female predominance (~54%) in macrophage-phenotype studies but emphasized that the M1/M2 shift occurs in both sexes.[8] Our balanced gender representation therefore aligns well with these prior cohorts, ensuring that our findings on ferritin, LDH, ESR, and hs-CRP are applicable across sexes and supporting robust comparisons in stratified analyses.

Anthropometric and Metabolic Profiles

Participants averaged central obesity (waist circumference 93.46 ± 7.24 cm) and obesity- range BMI (31.26 ± 2.53 kg/m²). Blood pressure was elevated (mean 146.80 ± 20.14/88.04 ± 12.03 mmHg), fasting blood sugar was impaired (134.59 ± 44.31 mg/dL), and dyslipidemia was evident with triglycerides of 179.41 ± 51.52 mg/dL and low HDL of 35.10 ± 7.72 mg/dL. These values echo those summarized by Esposito and Giugliano, who reported mean waist circumferences of ~92 cm, BMI ~31.2 kg/m², triglycerides ~180 mg/dL, and HDL ~34 mg/dL in MetS cohorts.[4] Lee and Pratley's subjects showed similar profiles: mean WC 94.5 cm, BMI 32.1 kg/m², FBS 130 mg/dL, TG 175 mg/dL, HDL 33 mg/dL.[5] Monteiro and Azevedo documented mean BP 145/85 mmHg and FBS ~128 mg/dL in obese- inflammation studies.[6] Wajchenberg et al. found mean BMI 30.8 kg/m² and TG 182 mg/dL, while Ning noted mean HDL of 36 mg/dL and FBS of 132 mg/dL in comparable cohorts.[7,8] Faloia et al. additionally highlighted that these metabolic derangements— particularly central adiposity and dyslipidemia—correlate tightly with elevations in inflammatory markers, reinforcing the interconnected nature of adipose dysfunction,

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metabolic syndrome, and systemic inflammation.[9] Our anthropometric and metabolic data thus align with established values in obesity–inflammation research, providing a solid foundation for interpreting biomarker associations within a well-characterized at-risk population.

Mean Serum Ferritin Levels by Metabolic Syndrome Status

Participants with metabolic syndrome (MetS) exhibited a markedly elevated mean serum ferritin of 516.42 ng/mL (SD 391.49) versus 102.23 ng/mL (SD 70.63) in those without MetS ($p < 0.001$). This more-than-five-fold increase underscores ferritin's function as both an iron- storage protein and an acute-phase reactant reflecting chronic low-grade inflammation. Esposito and Giugliano posited that adipose-derived IL-6 and TNF-α drive hepatic synthesis of acute-phase proteins, including ferritin, in obesity-associated MetS.[4] Monteiro and Azevedo reported comparable mean ferritin (~450 ng/mL, SD 160) in obese MetS cohorts, attributing this rise to oxidative stress–induced adipocyte dysfunction.[5] Wajchenberg et al. emphasized visceral adipose tissue's endocrine role in secreting proinflammatory adipokines that upregulate ferritin synthesis, documenting mean ferritin of 430 ng/mL in their MetS subgroup.[6] Ning described macrophage M1 polarization in obese adipose tissue as a driver of ferritin release, observing mean levels near 470 ng/mL (SD 180) in MetS subjects.[7] Faloia et al. similarly measured mean ferritin of 420 ng/mL (SD 180) in visceral obesity, finding a strong correlation with waist circumference ($r = 0.52$, $p < 0.01$).[8] Our observed mean of 516.42 ng/mL exceeds these earlier reports, suggesting either greater inflammatory burden or genetic/environmental predispositions in our South Asian cohort. Collectively, these data affirm serum ferritin's utility as a cost-effective biomarker for early identification of obese individuals at heightened cardiometabolic risk.

Mean LDH Levels by Metabolic Syndrome Status

Mean lactate dehydrogenase (LDH) was 390.97 U/L (SD 180.06) in MetS participants compared to 153.50 U/L (SD 188.14) in non-MetS ($p < 0.001$), representing over a 2.5-fold increase. LDH elevation reflects subclinical tissue injury and adipocyte turnover in chronic inflammation. Esposito and Giugliano's framework suggests adipose hypoxia and cytokine release cause local cell stress, leading to LDH leakage into circulation.[4] Monteiro and Azevedo reported mean LDH of 320 U/L (SD 120) in obese MetS subjects, with positive correlation to triglycerides ($r = 0.38$, $p < 0.05$).[5] Park and Woo summarized that LDH levels around 350 U/L are typical in MetS populations, linking LDH to innate immune activation and metabolic disturbances.[10] Liaw and Peplow demonstrated that electroacupuncture reduced LDH by ~25% in obese rat models, highlighting LDH's responsiveness to anti-inflammatory interventions.[11] Cooke et al. noted that saturated-fat– induced inflammation can raise LDH by 20–30% over baseline in experimental settings.[12] Ning also associated macrophage infiltration with LDH elevations of approximately 300 U/L in obesity-driven inflammation. Our mean of 390.97 U/L surpasses these prior values, suggesting more extensive adipose remodeling or prolonged disease duration in our cohort. These converging lines of evidence support LDH's inclusion in inflammatory panels for metabolic syndrome risk assessment and therapeutic monitoring.

Mean ESR Levels by Metabolic Syndrome Status

Erythrocyte sedimentation rate (ESR) averaged 75.88 mm/hr (SD 35.35) in MetS subjects versus 37.33 mm/hr (SD 41.87) in non-MetS ($p < 0.001$), demonstrating a greater than two- fold increase. ESR is elevated by acute-phase proteins—primarily fibrinogen—produced in response to adipose-derived cytokines. Esposito and Giugliano described ESR elevations up to three-fold in obese individuals due to IL-6–mediated fibrinogen synthesis.[4] Lee and Pratley reported mean ESR of 45 mm/hr (SD 18) in MetS cohorts versus 20 mm/hr (SD 12) in controls, correlating ESR with insulin resistance metrics ($r \approx 0.42$, $p < 0.01$).[5] Monteiro and Azevedo found average ESR of ~60 mm/hr in MetS patients, linking oxidative stress to heightened fibrinogen production.[6] Nadulska et al. documented ESR means of 50 mm/hr (SD 15) in obese but otherwise healthy adults, underscoring ESR's sensitivity to low-grade inflammation.[13] Liaw and Peplow demonstrated neuromodulation reduced ESR by ~30% in animal models, reflecting its dynamic nature.[11] Our mean ESR of 75.88 mm/hr surpasses these earlier estimates, indicating an amplified systemic inflammatory burden in our cohort. These findings reinforce ESR's value as an inexpensive, readily available marker for inflammation screening and risk stratification in obese individuals at risk for metabolic syndrome.

Mean hs-CRP Levels by Metabolic Syndrome Status: High-sensitivity C-reactive protein (hs-CRP) averaged 24.81 mg/L (SD 31.15) in MetS participants versus 2.63 mg/L (SD 6.04) in non-MetS ($p = 0.0001$), a nearly ten-fold difference. hs-CRP is a well-established predictor of cardiovascular and metabolic risk. Esposito and Giugliano reviewed that obese individuals often exhibit hs-CRP levels two- to five-fold higher than lean controls, driven by adipose-derived cytokines.[4] Lee and Pratley measured mean hs-CRP of 8.6 mg/L (SD 4.2) in MetS subjects versus 2.3 mg/L (SD 1.5) in controls, finding a dose–response relationship between hs-CRP tertiles and number of MetS components.[5] Monteiro and Azevedo reported mean hs-CRP of ~12 mg/L in obese MetS patients, linking oxidative stress to CRP synthesis.[6] Faloia et al. observed mean hs-CRP of 10.5 mg/L (SD 5.1) in visceral obesity, correlating levels with adipose macrophage infiltration. Saltiel and Olefsky highlighted that each 1 mg/L increase in hs-CRP raises cardiovascular risk by 4% in MetS populations. Our remarkably high mean of 24.81 mg/L suggests severe inflammatory

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activation in our cohort, underscoring hs-CRP's pivotal role in risk stratification and guiding anti-inflammatory, cardiometabolic interventions in obese patients.

Strengths

This study's primary strength lies in its robust selection and characterization of the participant cohort, ensuring representativeness and validity. A well-defined sample of 90 individuals allowed detailed analysis of multiple inflammatory markers, enhancing the precision and reliability of results. The balanced gender distribution, nearly equal between males (48.89%) and females (51.11%), minimized potential sex-based confounding and provided comprehensive insights applicable across genders. Additionally, the age distribution, predominantly within the 41–70 years range, captured the critical period for metabolic syndrome risk, increasing the relevance of findings. Measurement of multiple inflammatory biomarkers (ferritin, LDH, ESR, hs-CRP) alongside thorough metabolic profiling, including anthropometric parameters (BMI, waist circumference), blood pressure, glucose, and lipid profiles, provided a holistic view of metabolic dysfunction. This multidimensional approach allowed the evaluation of relationships between inflammation and metabolic syndrome from multiple angles, making the conclusions robust and well-supported.

CONCLUSION

The present study provides compelling evidence that chronic low-grade inflammation is integral to the pathophysiology of metabolic syndrome in obesity, as demonstrated by consistent and highly significant elevations across multiple inflammatory biomarkers. Middle-aged adults between 41 and 70 years comprised over 90% of our cohort, reflecting the demographic most vulnerable to metabolic disturbances, while the near-equal gender representation enhances the applicability of our findings across sexes. Anthropometric measures confirmed central obesity, with a mean waist circumference of 93.46 cm and BMI of 31.26 kg/m², coupled with marked metabolic derangements: elevated blood pressures of 146.80/88.04 mmHg, impaired fasting glucose of 134.59 mg/dL, heightened triglycerides of 179.41 mg/dL, and reduced HDL of 35.10 mg/dL. Against this backdrop, inflammatory markers were heterogeneous yet uniformly higher in those meeting metabolic syndrome criteria. Serum ferritin, reflecting both iron overload and acute-phase response, was more than five-fold higher (516.42 ng/mL) in the metabolic syndrome group compared to those without the syndrome (102.23 ng/mL), underscoring its potential as an accessible biomarker for early risk stratification. LDH, indicative of subclinical tissue injury and adipocyte turnover, was elevated by over 2.5-fold (390.97 vs. 153.50 U/L), highlighting ongoing cytotoxic stress in metabolic syndrome. ESR, a nonspecific but sensitive indicator of systemic inflammation, was over twice as high in the metabolic syndrome cohort (75.88 vs. 37.33 mm/hr), while hs-CRP, the gold standard for low-grade inflammation and cardiovascular risk prediction, exhibited a nearly ten-fold escalation (24.81 vs. 2.63 mg/L). The proportions of individuals with abnormal levels of these markers—85.0% for ferritin, 88.3% for LDH, 93.3% for ESR, and 88.3% for hs-CRP—in the metabolic syndrome group reinforce the notion that inflammation is not merely a concomitant feature but a driving force in metabolic dysregulation. These findings carry significant clinical and public health implications. Routine screening of obese patients for inflammatory biomarkers could enable earlier detection of metabolic risk, permitting timely lifestyle and pharmacologic interventions aimed at attenuating systemic inflammation and preventing progression to overt cardiometabolic disease. Specifically, serum ferritin and ESR—both inexpensive and widely available—could be integrated into standard metabolic panels, while LDH and hs-CRP can provide additional granularity in risk assessment. Therapeutic strategies should extend beyond weight loss to encompass targeted anti-inflammatory approaches, including dietary modifications rich in anti-inflammatory nutrients, structured physical activity programs, and, where appropriate, pharmacologic agents such as statins, omega-3 fatty acids, or novel immunomodulators. Future research should focus on longitudinal studies to clarify the temporal sequence of inflammation and metabolic syndrome onset, explore genetic and environmental modifiers of inflammatory responses in diverse populations, and conduct randomized trials to establish the efficacy of specific anti-inflammatory interventions in reversing or preventing metabolic syndrome. In sum, by elucidating the magnitude and consistency of inflammatory elevations in obesity-related metabolic syndrome, this study underscores the central role of chronic inflammation in cardiometabolic risk and lays the groundwork for inflammation-targeted diagnostic and therapeutic paradigms that may transform the management of obese patients worldwide.

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