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Comparative Evaluation of Lipid Profiles and Cardiac Risk Markers in Obese and Non-Obese Patients Attending a Cardiology Clinic

Maryam Ashraf [1], Muhammad Zahid Ali Raza [2], Faiza Altaf [3], Maida Anwar[1], Arsalan Aslam Chaudhary [4], Aamir Siddique [5]*

1- Department of Cardiology, Sharif Medical City Hospital, Lahore, Pakistan
2- Department of Cardiology, Nawaz Sharif Medical College, Gujrat, Pakistan
3- Department of Paediatric Medicine, Imran Idrees Teaching Hospital, Sialkot, Pakistan
4- Department of Cardiology, King Edward Medical University, Lahore, Pakistan
5- Department of Cardiology, Chaudhary Pervaiz Ilahi Institute of Cardiology, Wazirabad, Pakistan

***Corresponding Author:** Maryam Ashraf **Email:** mrymthaheem@yahoo.com

ABSTRACT

Background: Obesity is highly prevalent in Pakistan and contributes to cardiovascular risk by promoting dyslipidemia and low-grade inflammation.

Objectives: To compare fasting lipid profiles and advanced cardiac risk markers—high-sensitivity C-reactive protein (hs-CRP), apolipoprotein B/A-I ratio, and atherogenic index of plasma (AIP)—between obese and non-obese adults attending a cardiology clinic.

Methods: In this comparative cross-sectional study at Sharif Medical City Hospital, Lahore (1st, January 2024 – 31st, December 2025), 35 obese (BMI ≥ 30 kg/m²) and 35 non-obese (BMI < 25 kg/m²) outpatients (30–60 years) without diabetes, hypertension, thyroid dysfunction, recent acute coronary syndrome, chronic inflammation, pregnancy, or lipid-lowering therapy were consecutively enrolled. After a 10–12 h fast, serum total cholesterol, triglycerides, HDL-C, and LDL-C (enzymatic assays); apolipoproteins A-I and B (immunoturbidimetry); and hs-CRP (high-sensitivity immunoturbidimetry) were measured. VLDL-C was estimated as TG/5 and AIP as $\log_{10}$(TG/HDL-C). Independent-samples t-tests and Pearson's correlations were performed (SPSS v.26; $p < 0.05$).

Results: Compared with non-obese subjects, the obese group had significantly higher triglycerides (192 ± 48 vs. 144 ± 35 mg/dL), LDL-C (138 ± 30 vs. 119 ± 26 mg/dL), VLDL-C (38 ± 10 vs. 29 ± 7 mg/dL), and total cholesterol (214 ± 36 vs. 196 ± 32 mg/dL; all $p \leq 0.02$), and lower HDL-C (37 ± 9 vs. 52 ± 11 mg/dL; $p < 0.001$). Hs-CRP (4.0 ± 1.4 vs. 1.9 ± 0.8 mg/L), ApoB/A-I ratio (0.96 ± 0.22 vs. 0.64 ± 0.17), and AIP (0.69 ± 0.20 vs. 0.46 ± 0.15) were markedly elevated in the obese cohort (all $p < 0.001$).

Conclusion: In this Pakistani cardiology population, obesity is associated with an atherogenic lipid profile, heightened systemic inflammation, and unfavourable lipoprotein ratios. Incorporation of these advanced markers into routine risk assessment may improve early identification of high-risk patients.

Keywords: Obesity, Dyslipidemias, Lipoproteins, Triglycerides, Cholesterol, LDL, HDL, Apolipoproteins A-I, Risk Assessment

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INTRODUCTION

Obesity has become a worldwide health disaster of gargantuan proportions that has nearly tripled in the last four decades. Back when this was a problem relegated to high-income countries, it has now completely taken hold in low and middle-income countries and changed the world landscape of chronic disease risk [1]. Rapid urbanization, calorie-dense diets, and increasingly sedentary lifestyles have led to a marked rise in obesity rates in Pakistan. This rise is of obvious importance for cardiovascular health, for excess adiposity does not simply represent an inert energy reserve, but rather an active endocrine organ that deranges metabolic and inflammatory homeostasis [2].

Expansions in adipose tissue result in cascades of biochemical alterations. Hepatic lipid processing pathways are overwhelmed by greater quantities of free fatty acids released by enlarged fat cells that are dysregulated. The enzyme pathway that leads to HDL formation and reverse cholesterol transport is down-regulated, while the liver synthesizes and secretes higher levels of triglyceride-rich very–low-density lipoproteins. As a result, obese individuals usually have the classic atherogenic dyslipidemia triad of elevated triglycerides, decreased HDL cholesterol, and an excess of small, dense LDL particles, all of which are linked to endothelial dysfunction and plaque formation [3].

In addition to lipid derangements, obesity is a state of chronic low-grade inflammation. Expanded fat depots are infiltrated by adipocytes and pro-inflammatory cytokines and acute phase reactants from infiltrating immune cells, which perpetuate vascular injury and accelerate atherosclerotic processes. These biomarkers capture subclinical inflammation (high sensitivity C-reactive protein) that gives clinicians an early warning signal of heightened cardiovascular risk even before overt symptoms or structural changes in the heart and vessels develop [4].

Traditional lipid panels—while indispensable—offer only a partial view of an individual's cardiovascular risk profile. Parameters that better represent the underlying pathobiology have been introduced through innovations in lipoprotein and inflammatory marker assessment [5]. For example, the ratio of apolipoprotein B to apolipoprotein A-I quantifies the balance between atherogenic particles carrying apolipoprotein B and protective HDL particles carrying apolipoprotein A-I. The atherogenic index of plasma (as the logarithm of the triglyceride to HDL cholesterol ratio) is a surrogate for LDL particle size and density, which are related to plaque vulnerability. Taken together, these advanced measures offer a more mechanistic, richer picture of cardiovascular risk in metabolically challenged populations [6].

In Pakistan's tertiary cardiology clinics, patients frequently present with established heart disease or multiple risk While these advanced lipid and inflammatory markers are factors, systematic evaluation of these markers is uncommon. For example, total cholesterol and LDL cholesterol are commonly used as standard practice, but may miss subtler yet equally important messages of cardiometabolic dysfunction. In this setting, clinicians can perform a direct comparison between obese and non obese patients, and thereby identify the biochemical signatures that most appropriately require tailored prevention and treatment strategies [7].

Further supporting the need for localized data are the specific context of Pakistani dietary patterns, genetic predispositions, and constraints to healthcare resource availability. Lipid metabolism and inflammatory response differences across ethnic groups have been documented, and risk thresholds developed in Western cohorts may

not generalize fully to South Asian populations. Additionally, rising healthcare costs and lack of access to cutting-edge therapies in most parts of Pakistan make it even more urgent to start early and correctly risk-stratify to target the interventions most likely to yield the most benefit [8].

As such, this current study aimed to fill a major gap by assessing the fasting lipid profile in comparison to selected cardiac risk markers—high sensitivity C-reactive protein, apolipoprotein B/A1 ratio, and atherogenic index of plasma—in obese versus non obese adults attending a cardiology clinic. In this analysis, current study seek to shed light on metabolic and inflammatory burdens that are unique to obesity in our patient population, to help inform more nuanced clinical decision-making, and contribute to more effective cardiovascular disease prevention strategies in Pakistan [9].

MATERIALS AND METHODS

The current study was designed as a comparative cross-sectional study and was conducted in the cardiology department of Sharif Medical City Hospital, Lahore, Pakistan, between 1st January 2024 and 31st December 2025. Seventy adults aged 30–60 years were enrolled consecutively and stratified by body mass index into two equal groups: 35 obese (BMI $\geq$ 30 kg/m²) and 35 non-obese (BMI < 25 kg/m²). Individuals with diabetes mellitus, hypertension, thyroid dysfunction, acute coronary syndrome within the preceding three months, chronic inflammatory or autoimmune disease, pregnancy, or current lipid-lowering therapy were excluded to minimize confounding.

Anthropometric measurements were obtained using a calibrated digital scale and stadiometer, with BMI calculated as weight (kg) divided by height squared (m²). After a 10–12-hour overnight fast, trained phlebotomists drew 5 mL of venous blood from each participant. Samples were allowed to clot at room temperature, centrifuged at 3,000 rpm for 10 minutes, and serum aliquots stored at –20 °C until batch analysis.

Biochemical assays were performed in the hospital's central laboratory under strict internal quality-control protocols. Serum total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol were measured enzymatically on a Roche Cobas c311 analyzer, with very-low-density lipoprotein cholesterol estimated as one-fifth of the triglyceride concentration. High-sensitivity C-reactive protein was measured using an immunoturbidimetric assay on a Beckman Coulter AU5800 system, and apolipoproteins A-I and B were quantified using immunoturbidimetry on the same platform. The $\log_{10}$ (triglycerides/HDL-C) was used to compute the atherogenic index of plasma.

SPSS version 26 was used to enter and analyse the data; Pearson's correlation coefficients were computed to look at relationships between lipid and inflammatory markers, the Shapiro-Wilk test was used to determine normality, the Shapiro-Wilk test was used to evaluate between-group differences, and the continuous variables were presented as mean ± SD. When a two-tailed $p < 0.05$, statistical significance was established. Written informed permission was given by each participant, and the Institutional Review Board granted ethical approval. In addition to being advised of their right to withdraw at any time without affecting their clinical care, participants were given unique research codes to preserve confidentiality.

RESULTS

Seventy patients were enrolled and stratified into obese (n = 35) and non obese (n = 35) groups equally. Age distribution, sex ratio, marital status, most socioeconomic and lifestyle

characteristics, were similar in both cohorts, thus creating a well-balanced comparison (Table 1). Although participants with obesity had significantly elevated measures of adiposity (all $p < 0.001$, BMI, waist circumference, waist–hip ratio, and body fat percentage), a number of these measures were not significantly elevated among overweight participants (BMI, waist circumference, waist–hip ratio, and body fat percentage). Obese patients also had higher systolic and diastolic blood pressures and modestly elevated fasting glucose levels ($p \leq 0.04$), which are suggestive of early cardiometabolic perturbations clinically.

In Table 1, the demographic, socioeconomic, anthropometric, clinical, and lifestyle characteristics of the study population are described. Mean age (47.9 ± 7.2 vs. 46.8 ± 8.1 years; $p = 0.52$) and sex distribution (54.3% vs. 51.4% male; $p = 0.74$) were comparable between the obese and non-obese groups, however, substantial differences were observed in measures of adiposity and related clinical parameters. The mean BMI of the obese subjects was 33.1 ± 2.9 kg/m², while the non-obese subjects' mean BMI was 23.0 ± 1.6 kg/m² ($p < 0.001$) (Table 1). Similarly, waist circumference and waist–hip ratio (proxies for central adiposity) were higher in the obese group (102 ± 8 cm vs. 82 ± 6 cm, $p < 0.001$, 0.94 ± 0.05 vs. 0.86 ± 0.04, $p < 0.001$), indicating higher visceral fat accumulation. The amount of adipose tissue expansion was remarkably high, as it was markedly higher among obese individuals (36.5 ± 5.0% vs. 25.2 ± 4.8%; $p < 0.001$). In the clinical setting, obese patients had mild but significant increases in systolic (128 ± 10 vs. 122 ± 8 mmHg; $p = 0.01$) and diastolic (82 ± 8 vs. 78 ± 6 mmHg; $p = 0.04$) blood pressure, fasting blood glucose (98 ± 10 vs. 92 ± 8 mg/dL; $p = 0.03$), and early cardiometabolic perturbations. These findings were also paralleled by lifestyle factors, with a higher percentage of obese subjects reporting sedentary behavior (62.9% vs 42.9%; $p = 0.08$) and less vigorous physical activity, but no significant difference in family history of diabetes or hypertension.

Table-1: Demographic, Socioeconomic, Anthropometric, Clinical, and Lifestyle Characteristics

Characteristic	Obese (n = 35)	Non-Obese (n = 35)	p-Value
Age, years (mean ± SD)	47.9 ± 7.2	46.8 ± 8.1	0.52
Age < 40, n (%)	10 (28.6%)	12 (34.3%)	0.60
Age 40–50, n (%)	15 (42.9%)	13 (37.1%)	0.58
Age > 50, n (%)	10 (28.6%)	10 (28.6%)	0.61
Male, n (%)	19 (54.3%)	18 (51.4%)	0.74
Female, n (%)	16 (45.7%)	17 (48.6%)	—
Married, n (%)	30 (85.7%)	28 (80.0%)	0.53
Single, n (%)	5 (14.3%)	7 (20.0%)	—
No formal education, n (%)	4 (11.4%)	3 (8.6%)	0.69
Primary education, n (%)	8 (22.9%)	7 (20.0%)	0.77
Secondary education, n (%)	12 (34.3%)	14 (40.0%)	0.61
Tertiary education, n (%)	11 (31.4%)	11 (31.4%)	1.00

Employed, n (%)	20 (57.1%)	22 (62.9%)	0.61
Unemployed, n (%)	15 (42.9%)	13 (37.1%)	—
Urban residence, n (%)	28 (80.0%)	25 (71.4%)	0.41
Rural residence, n (%)	7 (20.0%)	10 (28.6%)	—
Income < 50,000 PKR, n (%)	10 (28.6%)	12 (34.3%)	0.60
Income 50,000–100,000 PKR, n (%)	15 (42.9%)	14 (40.0%)	0.81
Income > 100 000 PKR, n (%)	10 (28.6%)	9 (25.7%)	0.80
Never smoker, n (%)	25 (71.4%)	28 (80.0%)	0.32
Former smoker, n (%)	5 (14.3%)	4 (11.4%)	0.45
Current smoker, n (%)	5 (14.3%)	3 (8.6%)	0.25
Vegetarian diet, n (%)	4 (11.4%)	3 (8.6%)	—
Mixed diet, n (%)	31 (88.6%)	32 (91.4%)	—
Sedentary activity, n (%)	22 (62.9%)	15 (42.9%)	0.08
Moderate activity, n (%)	10 (28.6%)	14 (40.0%)	—
Vigorous activity, n (%)	3 (8.6%)	6 (17.1%)	—
Family history of diabetes, n (%)	18 (51.4%)	12 (34.3%)	0.12
Family history of hypertension, n (%)	20 (57.1%)	15 (42.9%)	0.20
Weight, kg (mean ± SD)	92 ± 12	68 ± 10	< 0.001
Height, cm (mean ± SD)	167 ± 8	171 ± 7	0.03
BMI, kg/m² (mean ± SD)	33.1 ± 2.9	23.0 ± 1.6	< 0.001
Waist circumference, cm (mean ± SD)	102 ± 8	82 ± 6	< 0.001
Waist–hip ratio (mean ± SD)	0.94 ± 0.05	0.86 ± 0.04	< 0.001
Body fat, % (mean ± SD)	36.5 ± 5.0	25.2 ± 4.8	< 0.001
Systolic BP, mmHg (mean ± SD)	128 ± 10	122 ± 8	0.01
Diastolic BP, mmHg (mean ± SD)	82 ± 8	78 ± 6	0.04
Fasting glucose, mg/dL (mean ± SD)	98 ± 10	92 ± 8	0.03

The fasting lipid profiles (Table 2) were highly atherogenic in obese patients. Obese group had total cholesterol moderately elevated (214 ± 36 mg/dL vs 196 ± 32 mg/dL, $p = 0.02$), but the most striking difference occurred with triglycerides (192 ± 48 mg/dL vs 144 ± 35 mg/dL, $p < 0.001$) and HDL-C (37 ± 9 mg/dL vs 52 ± 11 mg/dL, $p < 0.001$). Obesity related dyslipidemia is reflected by hypertriglyceridemia and reduced HDL-C, both of which represent impaired lipolytic clearance and diminished reverse cholesterol transport. The sum of the lipid burden was also greater in obese patients (138 ± 30 mg/dL vs. 119 ± 26 mg/dL; $p < 0.001$), and LDL-C levels were significantly higher in obese patients (138 ± 30 mg/dL vs. 119 ± 26 mg/dL; $p < 0.001$). The obese cohort had elevated VLDL-C estimated

as one-fifth of triglycerides: 38 ± 10 mg/dL versus 29 ± 7 mg/dL ($p < 0.001$), which further underscores hepatic overproduction of TG-rich particles. Together, these alterations increase the risk of LDL particle oxidation and atheroma formation.

Table-2: Fasting Lipid Profiles

Parameter	Obese (n = 35)	Non-Obese (n = 35)	p-Value
Total Cholesterol (mg/dL)	214 ± 36	196 ± 32	0.02
Triglycerides (mg/dL)	192 ± 48	144 ± 35	< 0.001
HDL-C (mg/dL)	37 ± 9	52 ± 11	< 0.001
LDL-C (mg/dL)	138 ± 30	119 ± 26	< 0.001
VLDL (mg/dL)	38 ± 10	29 ± 7	< 0.001

Table 3 summarizes advanced cardiac risk markers that obesity is associated with, both increased inflammation and a more atherogenic lipoprotein profile. Consequently, mean hs-CRP levels in the obese group were more than twice those in non-obese individuals (4.0 ± 1.4 mg/L vs. 1.9 ± 0.8 mg/L; $p < 0.001$), and this state of chronic low-grade inflammation, favoring endothelial dysfunction, was present. Obese patients had significantly raised apolipoprotein B/A-I ratio (0.96 ± 0.22 vs. 0.64 ± 0.17, $p < 0.001$), indicating an increased number of ApoB-containing lipoproteins compared to protective ApoA-I-rich HDL. In the obese cohort, $\log_{10}$[TG/HDL-C] was 0.69 ± 0.20 vs 0.46 ± 0.15 in non-obese subjects ($p < 0.001$), and this narrowed the atherogenic index of plasma toward small, dense LDL particles with greater atherogenic potential. Together, these results reflect the consistent pattern in which obesity is associated not only with traditional lipid abnormalities, but also pro-inflammatory and lipoprotein particle–based risk markers. Elevated adiposity measures, dyslipidemia, and inflammation converge in obese patients and support the integration of these advanced biomarkers into clinical risk stratification.

Table-3: Cardiac Risk Markers

Marker	Obese (n = 35)	Non-Obese (n = 35)	p-Value
hs-CRP (mg/L)	4.0 ± 1.4	1.9 ± 0.8	< 0.001
ApoB/ApoA-I Ratio	0.96 ± 0.22	0.64 ± 0.17	< 0.001
Atherogenic Index of Plasma	0.69 ± 0.20	0.46 ± 0.15	< 0.001

In comparison to non-obese patients, obese patients had significantly greater adiposity, higher blood pressure, and impaired glycemic control. Lipid profiles of the former were markedly atherogenic with elevated triglycerides, LDL-C, and VLDL-C and decreased HDL-C, with pronounced systemic inflammation (hs-CRP) and unfavorable lipoprotein ratios (ApoB/ApoA-I, AIP). The convergent risk factors point to the multitude of cardiovascular burdens that obesity entails and underscore the importance of comprehensive risk stratification and targeted intervention in this high-risk population.

DISCUSSION

In this comparative analysis of obese and non-obese patients attending a cardiology clinic, we found a clear clustering of cardiometabolic risk factors in the obese cohort. Early perturbations in metabolic homeostasis were apparent in obese individuals with significantly higher measures of central adiposity (body mass index [BMI], waist circumference, waist–hip ratio, and body fat percentage) as well as modest elevation in blood pressure and fasting glucose [10]. Taken together, these findings highlight the interdependence of obesity and cardiometabolic derangements, where adipose tissue serves both as a storage depot for excess calories and as an active endocrine organ sequestering adipokines and free fatty acids that promote insulin resistance and endothelial dysfunction [11].

The dyslipidemic profile of obese patients (Table 2) consisted of severe hypertriglyceridemia, high LDL-C and VLDL-C, and low HDL-C. The constellation of these abnormalities is associated with the overproduction of triglyceride-rich lipoproteins by the liver in obesity and impaired lipoprotein lipase activity — all factors that support the development of small, dense LDL particles that are highly susceptible to oxidation. The resultant lipid milieu accelerates the atherogenic process through the promotion of subendothelial cholesterol deposition and proinflammatory signaling in the arterial wall [12, 13].

Obese subjects had significantly elevated high-sensitivity C-reactive protein (hs CRP), reflecting a state of chronic low-grade inflammation (Table 3). Macrophage infiltration and upregulation of pro-inflammatory cytokines (interleukin 6 and tumor necrosis factor alpha) are enhanced due to adipose tissue expansion and drive hepatic CRP synthesis. In addition to being a marker of systemic inflammation, hs-CRP also directly participates in atherogenesis by impairing nitric oxide–mediated vasodilation and promoting endothelial adhesion molecule expression [14].

Also of note were the disproportionate burden of atherogenic particles in obesity demonstrated by the apolipoprotein B/A-I ratio and the atherogenic index of plasma (AIP). The ApoB/A-I ratio is a measure of the pro-atherogenic lipoproteins to the anti-atherogenic HDL particles, and AIP is related to LDL particle size and density [15]. Table 3 shows that both indices were significantly higher in the obese group as well, further supporting the concept that simple cholesterol measurements may underestimate the residual risk conferred by lipoprotein particle number and quality [16].

These convergent risk factors of adiposity, dyslipidemia, inflammation, and unfavorable lipoprotein ratios clinically combine to increase cardiovascular event risk in obese individuals. In addition to standard lipid panels, routine assessment of advanced markers like hs-CRP, apolipoprotein ratios, and AIP can help refine risk stratification and identify patients who may benefit from early, intensive interventions[17]. Pharmacologic therapies, including statins, fibrates, or novel agents that alter triglyceride metabolism and inflammation, may be indicated in high-risk obese patients but remain foundational with lifestyle modification targeting weight loss and physical activity [18].

This study has limitations of a cross-sectional design, i.e., it cannot establish causal relations, and a single center setting with a small sample size of 70 participants, which might limit generalizability. Exclusion of patients on lipid-lowering therapy also limits the applicability of these findings to patients with untreated lipid profiles, and their effects on these advanced markers of atherosclerosis should be examined further [19].

Longitudinal follow-up to determine how changes in adiposity and metabolic parameters affect cardiovascular outcomes over

time and trials of targeted therapies to determine how they affect both traditional as well as advanced risk markers in obese populations are needed in future research [20].

CONCLUSION

The present study shows that obesity in a Pakistani cardiology clinic population is associated with an unfavourable constellation of cardiometabolic risk factors, including atherogenic dyslipidemia, systemic inflammation, and unfavourable lipoprotein particle profiles. The combination of traditional and advanced lipid and inflammatory markers into routine clinical evaluation may help to optimize the early identification of high-risk patients and define personalized preventive and therapeutic strategies to reduce cardiovascular morbidity and mortality in obese patients.

Conflict of Interest:

The authors declare that no conflicts of interest exist.

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Authors' Contributions:

All authors contributed equally to this work.

Data Availability:

De-identified data are available from the corresponding author upon reasonable request.

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