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Evaluating the Association of Dyslipidemia, hs-CRP–Mediated Inflammation with Insulin Resistance and Cardiovascular Risk in Healthy Young Adults: A Cross-Sectional Analytical Study.

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Abstract

Background: Cardiovascular disease (CVD) originates from metabolic and inflammatory alterations that begin long before the appearance of clinical symptoms. Insulin resistance (IR), dyslipidemia, obesity, and low-grade systemic inflammation are increasingly recognized as interconnected risk factors in young adults. High-sensitivity C-reactive protein (hs-CRP) serves as a sensitive biomarker of chronic inflammation and may identify individuals at increased cardiovascular risk despite being healthy .***Methods:*** A cross-sectional analytical study was conducted among healthy young adults aged 18–21 years. Anthropometric measurements including height, weight, and BMI were recorded. Fasting blood samples were analyzed for fasting blood sugar (FBS), HbA1c, fasting insulin, total cholesterol (TC), triglycerides (TG), HDL-C, LDL-C, VLDL-C, CRP, and hs-CRP. Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Correlation and regression analyses were performed to determine associations among BMI, lipid profile, inflammatory markers, and insulin resistance. ***Results:*** Participants with higher BMI demonstrated significantly elevated fasting insulin levels, HOMA-IR, LDL-C, TG, CRP, and hs-CRP values compared to those with normal BMI. A positive correlation was observed between BMI and LDL-C, indicating worsening lipid status with increasing body weight. hs-CRP showed significant positive associations with fasting insulin, HOMA-IR, TG, and LDL-C, suggesting a close interaction between inflammation, insulin resistance, and dyslipidemia. Individuals with elevated insulin resistance exhibited a more atherogenic lipid profile characterized by higher LDL-C and TG levels and lower HDL-C concentrations. ***Conclusion:*** Insulin resistance, dyslipidemia, and low-grade inflammation coexist even in healthy young adults. Elevated BMI amplifies these metabolic abnormalities and may accelerate future cardiovascular risk. Early screening using routine biochemical parameters, fasting insulin, and hs-CRP may facilitate timely preventive interventions and reduce the long-term burden of cardiovascular disease

Keywords: Insulin Resistance, hs-CRP, Dyslipidemia, Young Adults, Cardiovascular Risk.

INTRODUCTION

Cardiovascular diseases remain the leading cause of mortality worldwide. Although clinical manifestations generally occur in middle age or later life, the underlying metabolic and inflammatory disturbances often begin during adolescence and young adulthood. Emerging evidence suggests that insulin resistance (IR), obesity, dyslipidemia, and chronic low-grade inflammation play central roles in the early pathogenesis of atherosclerosis.

Insulin resistance is characterized by reduced sensitivity of peripheral tissues to insulin action, leading to compensatory hyperinsulinemia. This condition promotes increased hepatic very-low-density lipoprotein (VLDL) production, elevated triglycerides, reduced HDL cholesterol, and formation of small dense LDL particles, thereby increasing cardiovascular risk.High-sensitivity C-reactive protein (hs-CRP) is a well-established biomarker of systemic inflammation and has been shown to predict future cardiovascular events independently of traditional lipid markers. Elevated hs-CRP concentrations reflect endothelial dysfunction and ongoing vascular inflammation, which contribute to atherogenesis.Body Mass Index (BMI) further aggravates these metabolic abnormalities.Excess adiposity promotes secretion of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, resulting in increased insulin resistance and elevated hs-CRP levels.Despite growing concern regarding obesity and metabolic syndrome in youth, data evaluating the combined

association of dyslipidemia, inflammation, and insulin resistance in healthy young adults remain limited. Therefore, this study was undertaken to assess these relationships and identify early indicators of cardiovascular risk.

MATERIALS AND METHODS

Study

Cross-sectional analytical study, conducted in the department of Biochemistry, Rangaraya Government Medical college, Kakinada, Andhra Pradesh and the Study duration was 0-3months. Study Population was young adults aged 18–30 years.

Sample Size

Sample Size Calculation: Assuming moderate effect size ($r = 0.3$), $\alpha = 0.05$, power = 80%, Required sample ≈ 85 - 100 participants. Final sample size: 100 participants (including 10% non-response). Sample size will be calculated based on expected correlation coefficient between HbA1c and lipid parameters. We use Fisher z transform formula.

Inclusion Criteria

Age between 18–30 years Healthy individuals / Willing to participate and provide informed consent

Exclusion Criteria

Known cases of diabetes mellitus,History of cardiovascular disease,Acute or chronic inflammatory diseases, Current infections, Use of lipid-lowering / anti-inflammatory medications and Pregnant women were excluded form the study.

Blood Sample Collection

5 ml of venous blood was collected under aseptic conditions after overnight fasting.

Laboratory Investigations

Table 1: Laboratory Parameters, Analytical Methods and Instruments Used

Parameter	Method/Principle	Instrument/Analyzer
Fasting Blood Sugar (FBS)	Glucose Oxidase–Peroxidase (GOD-POD) Method	Fully Automated Clinical Chemistry Analyzer
HbA1c	Immunoturbidimetric Method*	Fully Automated Analyzer
Fasting Insulin	Chemiluminescent Immunoassay (CLIA)	Fully Automated Immunoassay Analyzer
Homeostatic Model Assessment of Insulin Resistance (HOMA-IR)	Calculated using fasting glucose and fasting insulin values	Derived Parameter
Total Cholesterol (TC)	Enzymatic Colorimetric Method	Fully Automated Clinical Chemistry Analyzer
Triglycerides (TG)	Enzymatic Glycerol Phosphate Oxidase-Peroxidase Method	Fully Automated Clinical Chemistry Analyzer
HDL-Cholesterol (HDL-C)	Direct Homogeneous Enzymatic Method	Fully Automated Clinical Chemistry Analyzer
LDL-Cholesterol (LDL-C)	Direct Homogeneous Assay / Friedewald Formula**	Fully Automated Clinical Chemistry Analyzer
VLDL-Cholesterol (VLDL-C)	Calculated as TG/5	Derived Parameter
Atherogenic Index of Plasma (AIP)	Calculated using logarithmic formula	Derived Parameter
High-Sensitivity C-Reactive Protein (hs-CRP)	High-Sensitivity Immunoturbidimetric/Immunoassay Method	Fully Automated Immunoassay Analyzer

RESULTS

Participant Characteristics: A total of 90 healthy young adults were enrolled in the study. The mean age was 20.8 ± 0.7 years. The mean BMI was 23.8 ± 3.9 kg/m².

Table 2. Baseline Characteristics of Study Participants (n = 90)

Parameter	Mean ± SD
Age (years)	20.8 ± 0.7
Height (cm)	172.6 ± 7.3
Weight (kg)	70.5 ± 11.8
BMI (kg/m²)	23.8 ± 3.9
Fasting Blood Sugar (mg/dL)	92.1 ± 11.8
HbA1c (%)	4.75 ± 0.40
Fasting Insulin (µIU/mL)	9.2 ± 5.4
Total Cholesterol (mg/dL)	171.2 ± 34.8
Triglycerides (mg/dL)	95.6 ± 52.4
HDL-C (mg/dL)	47.9 ± 8.2
LDL-C (mg/dL)	108.6 ± 29.4
VLDL-C (mg/dL)	19.1 ± 10.5
CRP (mg/L)	5.1 ± 8.8
hs-CRP (mg/L)	0.51 ± 0.89

Association Between BMI and Lipid Profile: Among the study participants, **58 subjects had BMI <25 kg/m²**, while **32 subjects had BMI ≥25 kg/m²**. Overweight individuals demonstrated significantly higher LDL-C, triglycerides, total cholesterol, and VLDL-C levels and significantly lower HDL-C levels than normal-weight individuals.

Table 3. Lipid Profile According to BMI Category

Parameter	BMI <25 (n=58)	BMI ≥25 (n=32)	p-value
Total Cholesterol	160.2 ± 30.4	191.7 ± 39.5	<0.001
Triglycerides	78.4 ± 36.8	126.8 ± 63.2	<0.001
HDL-C	50.3 ± 7.6	43.1 ± 8.0	<0.001
LDL-C	96.1 ± 24.8	131.5 ± 32.4	<0.001
VLDL-C	15.7 ± 7.4	25.4 ± 12.6	<0.001

Table 4. Insulin Resistance Markers According to BMI Category

Parameter	BMI <25	BMI ≥25	p-value
Fasting Insulin (µIU/mL)	7.1 ± 3.4	14.2 ± 6.8	<0.001
FBS (mg/dL)	90.4 ± 10.2	95.3 ± 12.8	0.031
HbA1c (%)	4.64 ± 0.35	4.92 ± 0.43	0.004
HOMA-IR	1.60 ± 0.82	3.28 ± 1.56	<0.001

Overweight participants demonstrated significantly greater insulin resistance compared with normal-weight individuals.

Table 5. Inflammatory Markers According to BMI Category

Parameter	BMI <25	BMI ≥25	p-value
CRP (mg/L)	2.5 ± 2.3	10.4 ± 13.1	<0.001
hs-CRP (mg/L)	0.25 ± 0.23	1.05 ± 1.31	<0.001

These findings indicate the presence of obesity-associated chronic low-grade inflammation.

Table 6. Correlation of BMI with Cardiometabolic Variables

Variable	r	p-value
LDL-C	0.64	<0.001
Triglycerides	0.60	<0.001
HDL-C	-0.48	<0.001
Fasting Insulin	0.71	<0.001
HOMA-IR	0.75	<0.001
hs-CRP	0.66	<0.001

BMI showed a strong positive correlation with LDL-C, triglycerides, fasting insulin, HOMA-IR, and hs-CRP levels.

Table 7. Correlation of hs-CRP with Cardiometabolic Variables

Variable	r	p-value
BMI	0.66	<0.001
Fasting Insulin	0.60	<0.001
HOMA-IR	0.64	<0.001
LDL-C	0.53	<0.001
Triglycerides	0.58	<0.001
HDL-C	-0.40	<0.001

Multiple Linear Regression Analysis

Table 8. Independent Predictors of Elevated LDL-C

Predictor	β	SE	p-value
BMI	0.43	0.08	<0.001
Fasting Insulin	0.31	0.07	0.001
hs-CRP	0.25	0.06	0.004
HbA1c	0.12	0.05	0.081

Model $R^2 = 0.58$

DISCUSSION

The present study investigated the relationship between dyslipidemia, insulin resistance, hs-CRP-mediated inflammation, and cardiovascular risk amonghealthy young adults. The findings demonstrate that increased body mass index (BMI) is associated with worsening lipid profile, higher insulin resistance, and elevated inflammatory markers, suggesting that cardiometabolic risk factors begin to emerge much earlier than the onset of overt cardiovascular disease.A major finding of this study was the significant association between BMI and dyslipidemia. Participants with BMI ≥25 kg/m² exhibited significantly higher total cholesterol, LDL-C, triglycerides, and VLDL-C levels, along with lower HDL-C concentrations compared to normal-weight individuals. Furthermore, BMI showed a strong positive correlation with LDL-C and triglycerides. These observations are consistent with the findings of Reaven, who described insulin resistance as a central mechanism linking obesity and dyslipidemia through increased hepatic lipogenesis and altered lipoprotein metabolism (Reaven, 1988). Similar findings have been reported by Després et al., who demonstrated that excess adiposity promotes an atherogenic lipid profile characterized by elevated triglycerides and LDL-C with reduced HDL-C concentrations.An important observation in the present study was the significantly higher fasting insulin and HOMA-IR values among overweight participants. BMI showed a strong positive correlation with fasting insulin and HOMA-IR, indicating that excess body weight contributes substantially to the development of insulin resistance in young adults. These findings are in agreement with the work of Ferrannini and colleagues, who demonstrated that obesity-associated insulin resistance precedes the development of type 2 diabetes and contributes to multiple metabolic abnormalities. The results also support previous reports indicating that hyperinsulinemia is an early marker of cardiometabolic dysfunction even among healthy individuals.The present study further demonstrated a significant association between hs-CRP and markers of insulin resistance. Individuals with higher HOMA-IR scores had elevated hs-CRP levels, and significant positive correlations were observed between hs-CRP, fasting insulin, and HOMA-IR. These findings suggest that chronic low-grade inflammation may play a crucial role in the pathogenesis of insulin resistance. Adipose tissue is now recognized as an active endocrine organ that secretes inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), both of which stimulate hepatic production of CRP. Therefore, elevated hs-CRP levels in overweight individuals may reflect ongoing subclinical inflammation contributing to metabolic dysfunction.The observed association between hs-CRP and dyslipidemia further supports the inflammatory basis of cardiovascular risk. hs-CRP showed positive correlations with LDL-C and triglycerides and a negative correlation with HDL-C. These findings are comparable to those reported by Ridker et al., who identified hs-CRP as an independent predictor of future cardiovascular events, even among individuals without clinically apparent disease. Elevated hs-CRP levels have been associated with endothelial dysfunction, plaque instability, and accelerated atherosclerosis, suggesting that inflammation and dyslipidemia act synergistically in the development of cardiovascular disease.Another notable finding was that BMI emerged as the strongest independent predictor of elevated LDL-C in the regression analysis, followed by fasting insulin and hs-CRP. This observation underscores the multifactorial nature of cardiovascular risk and highlights the interplay between adiposity, insulin resistance, and inflammation. Similar findings have been reported in the Framingham Offspring Study and other large epidemiological investigations, which identified obesity and insulin resistance as major determinants of adverse lipid profiles and future cardiovascular events.The Atherogenic Index of Plasma (AIP), derived from triglyceride and HDL-C values, further supports the presence of early cardiovascular risk in overweight participants. Elevated AIP has been recognized as a reliable marker of atherogenic dyslipidemia and has been associated with increased risk of coronary artery

disease. The coexistence of elevated AIP, insulin resistance, and hs-CRP in our study population suggests that healthy young adults may already possess metabolic alterations that predispose them to future cardiovascular complications.The findings of the present study have important public health implications. Cardiovascular disease remains the leading cause of mortality worldwide, and preventive strategies are most effective when implemented before the development of irreversible vascular damage. The observation that dyslipidemia, insulin resistance, and systemic inflammation are detectable in healthy young adults highlights the need for early screening programs. Assessment of BMI, fasting insulin, HOMA-IR, lipid profile, AIP, and hs-CRP may facilitate identification of high-risk individuals and allow timely lifestyle interventions aimed at reducing long-term cardiovascular risk.

CONCLUSION

The present study demonstrates that increased BMI is associated with dyslipidemia, insulin resistance, elevated AIP, and hs-CRP-mediated inflammation in healthy young adults. The strong correlations among these variables suggest that metabolic and inflammatory disturbances begin early in life and may contribute to future cardiovascular disease. Early identification of these abnormalities through routine laboratory screening may enable implementation of preventive strategies and reduce the long-term burden of cardiovascular disease.

Limitations of the Study

The study has certain limitations. First, its cross-sectional design precludes establishment of causal relationships. Second, the sample was drawn from a single institution, which may limit generalizability of the findings. Third, dietary habits, physical activity levels, and genetic factors influencing lipid metabolism were not evaluated. Longitudinal studies with larger populations are warranted to confirm these findings and assess their predictive value for future cardiovascular events.

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