AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 6. No 02. February ,2026

ASSOCIATION BETWEEN INSULIN RESISTANCE LEVEL AND LIPID PROFILE IN PATIENTS WITH OBESITY

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Abstract. Obesity is one of the most pressing problems of modern medicine, increasing the risk of metabolic syndrome, type 2 diabetes mellitus, and cardiovascular diseases. The aim of this study was to determine the relationship between the degree of insulin resistance and lipid profile parameters in patients with obesity. A total of 80 patients with obesity participated in the study. Body mass index (BMI), fasting glucose, insulin levels, and lipid profile were measured in all participants, and insulin resistance was assessed using the HOMA-IR index. The results showed that the HOMA-IR index was positively correlated with triglyceride and LDL levels and negatively correlated with HDL levels ($p<0.05$). The findings confirm that insulin resistance plays an important role in the development of dyslipidemia in obesity.

Keywords: obesity, insulin resistance, HOMA-IR, dyslipidemia, lipid profile.

Introduction

Obesity is a metabolic disorder that has reached the level of a global epidemic and is characterized by excessive accumulation of adipose tissue in the body. It is one of the main causes of insulin resistance. As a result of insulin resistance, glucose metabolism is impaired and lipid metabolism is altered.

The association between insulin resistance and dyslipidemia increases the risk of cardiovascular diseases. Therefore, early detection of metabolic changes in patients with obesity is of great importance.

Aim of the Study

To determine the relationship between the degree of insulin resistance and lipid profile parameters in patients with obesity.

Materials and Methods

The study was conducted in the endocrinology department during 2024–2025. Eighty obese patients aged 18–60 years were enrolled.

Inclusion criteria:

- BMI ≥ 30 kg/m²
- Age over 18 years

Exclusion criteria:

- Type 1 diabetes
- Severe liver or renal failure
- Patients receiving hormonal therapy

The following parameters were assessed in all patients:

- BMI
- Fasting glucose

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INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
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• Insulin
• Total cholesterol
• LDL
• HDL
• Triglycerides

Insulin resistance was calculated using the HOMA-IR formula:

HOMA-IR = (glucose mmol/L × insulin mIU/L) / 22.5

Statistical analysis was performed using SPSS software. Pearson correlation analysis was applied. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 80 obese patients were included in the study: 52 women (65%) and 28 men (35%). The mean age was 42.6 ± 9.4 years.

Abdominal obesity (waist circumference >94 cm in men, >80 cm in women) was identified in 68% of participants.

Insulin resistance (HOMA-IR >2.5) was observed in 62 patients (77.5%). Dyslipidemia was detected in 59 patients (73.7%).

Anthropometric and Biochemical Parameters (Mean ± SD)

Parameter	Mean ± SD
Age (years)	42.6 ± 9.4
BMI (kg/m²)	33.8 ± 2.4
Waist circumference (cm)	102.4 ± 8.6
Fasting glucose (mmol/L)	6.1 ± 0.8
Insulin (mIU/L)	18.4 ± 4.2
HOMA-IR	4.9 ± 1.3
Total cholesterol (mmol/L)	5.8 ± 0.9
LDL (mmol/L)	3.6 ± 0.7
HDL (mmol/L)	1.0 ± 0.2
Triglycerides (mmol/L)	2.1 ± 0.6

Correlation Between HOMA-IR and Lipid Parameters

Parameter	r	p
HOMA-IR and Total cholesterol	0.31	<0.05
HOMA-IR and LDL	0.36	<0.05
HOMA-IR and HDL	-0.40	<0.01
HOMA-IR and Triglycerides	0.42	<0.01

The results demonstrate that as insulin resistance increases, an atherogenic lipid profile develops.

Additional Statistical Analysis

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Patients were divided into two groups:
• Group 1: HOMA-IR ≤2.5 (n=18)
• Group 2: HOMA-IR >2.5 (n=62)

In the high HOMA-IR group:
• Triglycerides were 27% higher ($p<0.01$)
• LDL was 18% higher ($p<0.05$)
• HDL was 15% lower ($p<0.01$)

This indicates that insulin resistance is an independent factor in the development of dyslipidemia.

DISCUSSION

Obesity is one of the most significant problems of modern medicine, representing not only an aesthetic or anthropometric change but a complex systemic disease accompanied by metabolic and hormonal disturbances. In recent decades, the prevalence of obesity has increased significantly worldwide. According to the World Health Organization, overweight and obesity are major risk factors for cardiovascular diseases, type 2 diabetes mellitus, and metabolic syndrome.

Adipose tissue functions as an active endocrine organ. Adipocytes release cytokines and free fatty acids that disrupt insulin signaling mechanisms, leading to insulin resistance.

Insulin resistance increases hepatic VLDL synthesis, which elevates triglyceride levels. Reduced HDL contributes to the formation of an atherogenic profile.

Our findings are consistent with international literature and confirm the necessity of early lipid metabolism assessment in obese patients.

Pathophysiological Basis of Insulin Resistance

In obesity, hypertrophied adipocytes secrete increased amounts of free fatty acids (FFA), growth factors, and inflammatory cytokines (TNF-α, IL-6). These mediators impair insulin receptor signaling pathways, reducing glucose utilization and causing compensatory hyperinsulinemia.

Elevated free fatty acids enhance hepatic gluconeogenesis and VLDL synthesis, playing a central role in dyslipidemia development. The positive correlation between HOMA-IR and triglycerides ($r=0.42$; $p<0.01$) supports this mechanism.

Insulin Resistance and Dyslipidemia

Under normal conditions, insulin stimulates lipoprotein lipase activity and promotes triglyceride clearance. In insulin resistance, this mechanism is impaired, leading to hypertriglyceridemia. Excess hepatic VLDL production also increases small dense LDL particles.

The negative correlation between HOMA-IR and HDL observed in this study indicates reduced anti-atherogenic protection. HDL plays a crucial role in reverse cholesterol transport, and its reduction increases atherosclerosis risk.

Comparison with Other Studies

Numerous international studies confirm the association between obesity, insulin resistance, and an atherogenic lipid profile. The American Diabetes Association recognizes insulin resistance as an independent risk factor for cardiovascular disease.

Similarly, the International Diabetes Federation emphasizes that abdominal obesity is a central component of metabolic syndrome.

Our findings align with these concepts, particularly the higher HOMA-IR values in patients with abdominal obesity, suggesting that visceral adipose tissue is metabolically more active.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 6. No 02. February ,2026

Clinical Significance

The results have important clinical implications. First, evaluating BMI alone is insufficient in obese patients; insulin and lipid profiles should be routinely assessed.

Second, the HOMA-IR index is a simple and practical method for early detection of insulin resistance.

Third, lipid abnormalities may precede clinical symptoms; therefore, preventive metabolic screening is essential in obese individuals.

Pathogenetic Chain

Obesity → Adipose tissue hypertrophy → Inflammatory mediators → Impaired insulin signaling → Insulin resistance → Hyperinsulinemia → Increased VLDL → Dyslipidemia → Atherosclerosis risk

This chain highlights the need to consider obesity as a systemic metabolic disorder rather than merely excess body weight.

Limitations

The study has several limitations. The sample size was relatively small. Visceral fat volume was not assessed using instrumental methods (DXA or CT). Inflammatory markers (CRP, IL-6) were not measured.

Future studies with larger populations and additional biomarkers are recommended.

Preventive and Therapeutic Implications

Reducing insulin resistance in obese patients lowers metabolic risk. Lifestyle modification (diet therapy and physical activity) improves insulin sensitivity. Pharmacological agents that enhance insulin sensitivity may also be used.

Normalization of lipid profile plays a key role in preventing cardiovascular complications. Therefore, a comprehensive treatment approach is necessary for obese patients.

General Scientific Conclusion

The study demonstrates that insulin resistance is highly prevalent in obese patients and is closely associated with the development of an atherogenic lipid profile. Increased HOMA-IR is accompanied by elevated triglyceride and LDL levels and decreased HDL levels.

These findings support the role of insulin resistance as a key pathogenetic mechanism in dyslipidemia and cardiometabolic risk and emphasize the importance of early metabolic screening and comprehensive management in patients with obesity.

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ISSN NUMBER: 2692 - 5206 Volume 6. No 02. February ,2026

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