Section: General Medicine

Original Research Article

COMPARATIVE ANALYSIS OF INSULIN RESISTANCE PROFILES IN LEAN AND OBESE INDIVIDUALS WITH TYPE 2 DIABETES MELLITUS: A HOSPITAL BASED CROSS-SECTIONAL STUDY

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Received : 05/09/2023
Received in revised form : 05/10/2023
Accepted : 15/10/2023

Keywords:
Diabetes Mellitus, Insulin resistance, Obesity, BMI, Lipid profile.

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DOI: 10.47009/jamp.2023.5.5.229

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (5); 1165-1170

Abstract

Background: Type 2 diabetes mellitus (T2DM) is a prevalent metabolic disorder characterized by elevated blood glucose levels due to insulin resistance and impaired insulin secretion. Despite its association with obesity, an increasing number of lean individuals are being diagnosed with T2DM, prompting interest in the distinct insulin resistance patterns between lean and obese T2DM patients. This study aims to comprehensively compare insulin resistance profiles in these two groups to unravel the underlying mechanisms contributing to T2DM in each phenotype. Understanding these differences may lead to tailored treatment strategies and novel therapeutic targets. **Materials and Methods:** This cross-sectional study, conducted from February 2021 to January 2022 in a tertiary care hospital, enrolled lean and obese adults diagnosed with Type 2 Diabetes Mellitus (T2DM). Approval from the Institutional Review Board and informed consent were obtained, with exclusion criteria covering specific diabetes types, comorbidities, pregnancy, lactation, and relevant medications. With a calculated sample size of 184, participants were categorized into two groups: Group A (n=46, lean) and Group B (n=138, obese). Comprehensive clinical assessments, including anthropometric measurements and fasting blood sample analyses, were performed. Insulin resistance was evaluated using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), and statistical comparisons were made between the lean and obese groups ($p<0.05$ considered significant). **Result:** In this cross-sectional study of adults with Type 2 Diabetes Mellitus (T2DM), lean individuals (n=46) demonstrated significant differences compared to obese counterparts (n=138). Lean participants exhibited lower BMI (22.82 ± 1.54 vs. 32.53 ± 2.85, $p<0.0001$) and waist circumference (85.43 ± 5.21 vs. 101.24 ± 7.68, $p<0.0001$). Lean individuals had higher fasting plasma glucose (FPG) levels (216.48 ± 32.33 mg/dL vs. 188.27 ± 23.42 mg/dL, $p<0.0001$) and lower fasting serum insulin levels (8.21 ± 3.54 mU/L vs. 20.65 ± 2.82 mU/L, $p<0.0001$) compared to their obese counterparts. Systolic blood pressure was also lower in lean individuals (131 ± 10 mmHg vs. 142 ± 12 mmHg, $p<0.0001$). These results reveal significant anthropometric and metabolic distinctions between lean and obese T2DM patients. **Conclusion:** Clinically, these insights may inform tailored management strategies for individuals with T2DM, emphasizing the importance of addressing obesity and individualized treatment approaches.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by elevated blood glucose levels resulting from insulin resistance and impaired insulin secretion.[1] This global epidemic poses a significant public health challenge, with its prevalence steadily rising over the past few decades (1.6 to 26%).[2-4] Traditionally, T2DM has been associated with obesity, particularly central adiposity, as a major risk factor.[5] However, emerging evidence suggests that a substantial number of individuals with T2DM are lean, lacking the typical excess adipose tissue commonly

ISSN (O): 2687-5365; ISSN (P): 2753-6556

observed in this population.[6,7] This intriguing phenomenon has sparked interest in understanding the pathophysiological differences between lean and obese individuals with T2DM, particularly in the context of insulin resistance.[8]

Insulin resistance, a key feature of T2DM, refers to the diminished ability of insulin to promote glucose uptake into target tissues, such as skeletal muscle, liver, and adipose tissue.[8,9] It plays a central role in the development and progression of the disease, contributing to hyperglycaemia, dyslipidaemia, and increased cardiovascular risk. While obesity is a well-established contributor to insulin resistance, recent studies have illuminated the existence of non-obese individuals who also exhibit significant insulin resistance and, consequently, T2DM.[10-12]

The etiological and mechanistic distinctions between insulin resistance in lean and obese individuals with T2DM remain poorly understood.[10,11] These differences have important implications for clinical management, as treatment strategies may need to be tailored to address the unique underlying factors in each subgroup.[12] Furthermore, the identification of specific markers or pathways associated with insulin resistance in lean T2DM individuals may yield novel therapeutic targets.[12-14]

In light of these considerations, this cross-sectional study aimed to comprehensively compare insulin resistance profiles between lean and obese adults diagnosed with T2DM. By examining a diverse population, we seek to elucidate the underlying factors contributing to insulin resistance in these two distinct phenotypes and provide valuable insights into the pathophysiology of T2DM. This investigation holds the potential to not only enhance our understanding of the heterogeneity within the T2DM population but also inform the development of more precise and effective interventions for individuals with this debilitating condition. As we delve into the intricate interplay of genetics, lifestyle, and metabolic factors, we aspire to shed light on the multifaceted nature of insulin resistance in lean and obese adults with T2DM.

MATERIALS AND METHODS

Study Design and Participants

This cross-sectional study was conducted among lean and obese adults diagnosed with Type 2 Diabetes Mellitus (T2DM) visiting OPD or admitted in ward of the department of General Medicine of tertiary care hospital for a period of one year during February 2021 to January 2022. The study protocol received approval from the Institutional Review Board and adhered to the principles of the Declaration of Helsinki. Informed consent was obtained from all participants. Exclusion criteria included Gestational diabetes or other forms of diabetes, History of significant comorbidities (e.g., cardiovascular disease, chronic kidney disease), Pregnancy or lactation, and on medications that could significantly affect insulin sensitivity. The sample size was calculated as 184 based on a power analysis with a significance level of 0.05 and a power of 0.80, taking into account the expected effect size of 0.20 from previous literature on insulin resistance in lean and obese individuals with T2DM. The participants enrolled into two groups in the ratio of 1:3; Group A with n=46 [lean (BMI < 25 kg/m²)] and Group B with n=138 [obese (BMI ≥ 30 kg/m²)].

Data Collection

All participants underwent a comprehensive clinical evaluation. Demographic information, medical history, and medication use were recorded. Height, weight, waist circumference, and blood pressure were measured using standardized protocols. BMI was calculated as weight in kilograms divided by the square of height in meters.

Fasting blood samples were collected from all participants after an overnight fast of at least 8 hours. The following laboratory parameters were analysed: Fasting plasma glucose (FPG) using a standard glucose oxidase method; Haemoglobin A1c (HbA1c) levels; Fasting serum insulin levels using an enzyme-linked immunosorbent assay (ELISA) (normal range: 3 to 25 mU/L); C-peptide using chemilumiscence (normal range: 0.81 to 3.85 ng/mL), and Lipid profile, including total cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglycerides.

Insulin resistance (IR) was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) formula: HOMA−IR = (FPG × Fasting insulin/22.5). Higher HOMA-IR values indicate greater insulin resistance, and HOMA cut-off point for diagnosis of IR was taken as 2.77.

Statistical Analysis

Statistical analysis was performed using SPSS version 20.0. Descriptive statistics were used to summarize participant characteristics, and results were expressed as means ± standard deviations for continuous variables and as percentages for categorical variables. Differences between lean and obese groups were analysed using independent t-tests for continuous variables and chi-squared tests for categorical variables. A p-value of <0.05 was considered statistically significant.

RESULTS

The lean group (n=46) had a significantly lower mean age of 50.23 years (±6.36) compared to the obese group (n=138), which had a mean age of 55.71 years (±7.18) ($p < 0.0001$). In terms of gender distribution, the lean group had 19 (43.5%) females and 27 (56.5%) males, while the obese group had 74 (54.3%) females and 64 (45.7%) males, with no statistically significant difference observed ($p = 0.147$). Notably, the two groups exhibited significant differences in BMI and waist

ISSN (O): 2687-5365; ISSN (P): 2753-6556

circumference. The lean group had a significantly lower mean BMI of 22.82 kg/m² (±1.54) compared to the obese group's mean BMI of 32.53 kg/m² (±2.85) ($p < 0.0001$). Similarly, waist circumference was significantly lower in the lean group, with a mean of 85.43 cm (±5.21), in contrast to the obese group, which had a mean waist circumference of 101.24 cm (±7.68) ($p < 0.0001$). Furthermore, blood pressure measurements revealed significant differences between the two groups. The lean group exhibited lower blood pressure values, with a mean systolic blood pressure of 131 mmHg (±10) and a mean diastolic blood pressure of 82 mmHg (±7), while the obese group had a mean systolic blood pressure of 142 mmHg (±12) and a mean diastolic blood pressure of 85 mmHg (±8). These differences were statistically significant, with a p-value of < 0.0001 for systolic blood pressure and 0.024 for diastolic blood pressure [Table 1].

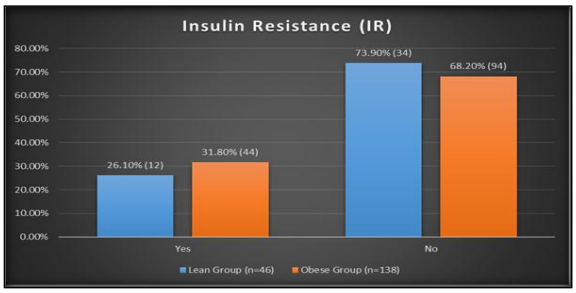

Figure 1: Comparison of Insulin resistance among two groups using HOMA formula (p value = 0.459)

Significant differences were observed in several key markers between the two groups. Fasting Plasma Glucose (FPG) levels were significantly higher in the lean group, with a mean of 206.48 mg/dL (±32.33), in contrast to the obese group, which had a lower mean FPG of 188.27 mg/dL (±23.42) ($p < 0.0001$). Hemoglobin A1c (HbA1c) levels were also markedly different, with the lean group exhibiting a higher mean HbA1c of 8.53% (±0.42) compared to the obese group's mean HbA1c of 7.81% (±0.63) ($p < 0.0001$). Furthermore, fasting serum insulin levels were significantly lower in the lean group, with a mean of 7.21 mU/L (±3.54), while the obese group had a higher mean fasting serum insulin of 8.65 mU/L (±4.82) ($p < 0.0001$). Similarly, C-peptide levels were notably lower in the lean group, with a mean of 2.27 ng/mL (±0.31), in contrast to the obese group's mean C-peptide level of 3.56 ng/mL (±0.49) ($p < 0.0001$). Total cholesterol and LDL-C levels were also significantly different between the two groups. The lean group had a lower mean total cholesterol of 198.61 mg/dL (±15.94) compared to the obese group's mean of 215.48 mg/dL (±21.29) ($p < 0.0001$). Additionally, LDL-C levels were significantly lower in the lean group, with a mean of 111.77 mg/dL (±12.36), while the obese group had a higher mean LDL-C of 142.47 mg/dL (±18.92) ($p < 0.0001$). Although HDL-C levels were also different, the lean group showed higher levels, with a mean of 51.44 mg/dL (±5.25), while the obese group had a lower mean HDL-C of 45.57 mg/dL (±6.33) ($p < 0.0001$). Conversely, triglyceride levels did not exhibit a statistically significant difference between the two groups, with the lean group having a mean triglyceride level of 178.75 mg/dL (±28.63) and the obese group having a mean of 181.29 mg/dL (±25.36) ($p = 0.550$). Regarding insulin resistance, the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) values demonstrated a significant difference. The lean group had a mean HOMA-IR of 3.23 (±0.35), while the obese group exhibited a slightly higher mean HOMA-IR of 3.54 (±0.66) ($p = 0.002$). These findings highlight substantial distinctions in various laboratory parameters related to glucose metabolism, lipids, and insulin sensitivity between the lean and obese groups, underscoring the complex interplay of these factors in individuals with Type 2 Diabetes Mellitus [Table 2].

Among the lean group participants (n=46), 12 individuals (26.1%) were identified as having insulin resistance, while 34 individuals (73.9%) showed no evidence of insulin resistance. In contrast, within the obese group (n=138), 44 individuals (31.8%) were classified as insulin resistant, while 94 individuals (68.2%) were found to be free of insulin resistance. The comparison of insulin resistance prevalence between the two groups revealed no statistically significant difference ($p = 0.459$), indicating that the occurrence of insulin resistance did not significantly vary between the lean and obese groups. These findings underscore the importance of investigating other factors contributing to insulin resistance in the context of type 2 diabetes mellitus in lean and obese individuals, as explored in subsequent sections [Figure 2].

Table 1: Comparison of baseline characteristics among the two groups

Characteristic	Lean Group (n=46)	Obese Group (n=138)	p-value
Age (years)	50.23 ± 6.36	55.71 ± 7.18	< 0.0001
Gender			
Female	19 (43.5%)	74 (54.3%)	0.147
Male	27 (56.5%)	64 (45.7%)	
BMI (kg/m²)	22.82 ± 1.54	32.53 ± 2.85	< 0.0001
Waist Circumference (cm)	85.43 ± 5.21	101.24 ± 7.68	< 0.0001
Blood Pressure (mmHg)			
Systolic BP	131 ± 10	142 ± 12	< 0.0001
Diastolic BP	82 ± 7	85 ± 8	0.024

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Table 2: Comparison of laboratory parameters among the two groups

Laboratory Parameter	Lean Group (n=46)	Obese Group (n=138)	p-value
Fasting Plasma Glucose (mg/dL)	206.48 ± 32.33	188.27 ± 23.42	< 0.0001
Hemoglobin A1c (%)	8.53 ± 0.42	7.81 ± 0.63	< 0.0001
Fasting Serum Insulin (mU/L)	7.21 ± 3.54	8.65 ± 4.82	< 0.0001
C-peptide (ng/mL)	2.27 ± 0.31	3.56 ± 0.49	< 0.0001
Total Cholesterol (mg/dL)	198.61 ± 15.94	215.48 ± 21.29	< 0.0001
HDL-C (mg/dL)	51.44 ± 5.25	45.57 ± 6.33	< 0.0001
Triglycerides (mg/dL)	178.75 ± 28.63	181.29 ± 25.36	0.550
LDL-C (mg/dL)	111.77 ± 12.36	142.47 ± 18.92	< 0.0001
HOMA-IR	3.23 ± 0.35	3.54 ± 0.66	0.002

DISCUSSION

This cross-sectional study aimed to compare insulin resistance and various metabolic parameters between lean and obese adults diagnosed with Type 2 Diabetes Mellitus (T2DM). The findings revealed several significant differences in participant characteristics and laboratory parameters, shedding light on the intricate relationship between obesity, insulin resistance, and metabolic dysregulation in this population.

Our study found that obese individuals with T2DM were, on average, older than their lean counterparts. This observation aligns with the well-established notion that T2DM tends to develop at an older age in lean individuals compared to those with obesity.[1] This age discrepancy may be attributed to diverse pathophysiological mechanisms underlying diabetes in different phenotypes, including differences in beta-cell function, insulin sensitivity, and genetic factors. Scheen et al., Imbeault et al., and Karakelides et al., however, noted that age had no independent effect on insulin resistance and increasing age per se did not influence glucose homeostasis and was not a cause of insulin resistance.[15-17]

In terms of gender distribution, our study did not find a significant difference between the two groups. This observation contrasts with Geer et al, that suggested a higher prevalence of T2DM in obese males as there was an elevated visceral and hepatic adiposity reported in males.[18] However, it is crucial to note that our study focused on individuals with existing T2DM, which may have influenced the gender distribution compared to studies encompassing the entire spectrum of diabetes risk. Also, Geer et al., showed that along with lower adiponectin levels and absence of oestrogen, males are noted to have higher insulin resistance compared to females.[18]

BMI and waist circumference emerged as crucial discriminators between the lean and obese groups. Unsurprisingly, obese individuals with T2DM exhibited significantly higher BMI and waist circumference than their lean counterparts. These findings underscore the central role of adiposity in the pathogenesis of T2DM and are consistent with extensive evidence linking obesity to increased diabetes risk.[3] Additionally, the higher blood pressure observed in the obese group is in line with previous studies highlighting the association between hypertension and obesity in T2DM patients.[4] This was congruent to the study of Shikha et al., that mean systolic blood pressure were significantly higher in obese subjects.[19]

The laboratory parameters further elucidate the metabolic disparities between the two groups. Notably, fasting plasma glucose (FPG) and HbA1c levels were significantly elevated in the lean group compared to the obese group. While counterintuitive at first glance, this finding may reflect differences in disease progression and beta-cell function. Lean individuals with T2DM may experience more rapid beta-cell failure and earlier loss of glycaemic control, leading to higher FPG and HbA1c levels and it was in line with studies done by Das et al., Shrivastav et al., Coleman et al., and Mohan et al., have suggested that lean individuals with T2DM experience more pronounced hyperglycaemia.[3,20-22] Conversely, studies by, Al-Goblan et al., Asegaonkar et al., and Akter et al., that has elucidated the connection between obesity and chronic systemic inflammation.[23-25]

Fasting serum insulin and C-peptide levels were both significantly lower in the lean group, suggesting reduced insulin secretion. This finding is consistent with previous research indicating that lean individuals with T2DM often exhibit a more pronounced impairment in beta-cell function. In contrast, the obese group had higher insulin levels, which may be indicative of insulin resistance. The relationship between obesity and insulin resistance is well-established, and our study reinforces this association in the context of T2DM, and it was in coherence with the studies by Anoop et al., Gonzalez-Cantero et al., and Shaisho et al.[26-28]

Total cholesterol and LDL-C levels were also significantly elevated in the obese group. These lipid abnormalities are common in individuals with obesity and T2DM and are associated with an increased risk of cardiovascular disease. On the other hand, the lean group had higher levels of HDL-C, a protective factor against cardiovascular events. This was in alignment to the data of Lukich et al., Sinharoy et al., Chandra et al., Barma et al., and Das et al., that BMI had a positive relationship with LDL and that lean persons with diabetes had lower incidence of dyslipidaemia with a generally favourable lipid profile.[29-33]

Interestingly, HOMA-IR, a surrogate marker of insulin resistance, was slightly higher in the obese

ISSN (O): 2687-5365; ISSN (P): 2753-6556

group. This finding suggests that despite having higher insulin levels, obese individuals may have decreased insulin sensitivity. The interaction between obesity and insulin resistance is complex, involving factors such as adipokines, inflammation, and ectopic fat deposition.[10] This supported previous studies by Ye et al., Zelada et al., and Chung et al., that insulin resistance was significantly increased in overweight/obese T2DM patients.[34-36] The HOMA-IR was also found to be increased in lean T2DM patients among studies by Tan et al., Petersen et al., Chandalia et al., Mishra et al., and Bhatt et al.[37-41]

Limitations

While our study provides valuable insights into the metabolic differences between lean and obese individuals with T2DM, several limitations should be acknowledged. The cross-sectional design precludes causal inferences, and longitudinal studies are needed to elucidate the progression of insulin resistance in these populations. Additionally, our sample size may limit the generalizability of the findings, and larger cohorts should be considered for future investigations.

CONCLUSION

In conclusion, our study highlights the heterogeneity of T2DM by comparing lean and obese individuals with the condition. The findings underscore the critical influence of adiposity on insulin resistance and metabolic dysregulation. Clinically, these insights may inform tailored management strategies for individuals with T2DM, emphasizing the importance of addressing obesity and individualized treatment approaches. Further research is warranted to explore the underlying mechanisms and therapeutic implications of these observations.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556

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ISSN (O): 2687-5365; ISSN (P): 2753-6556