HOMOCYSTEINE LEVEL, INSULIN RESISTANCE AND B-CELL FUNCTION AMONG PATIENTS WITH TYPE 2 DIABETES AND METABOLIC SYNDROME

Mohannad I. Abbas[1], Salman K. Ajlan[2], Lamees M. Hameed[3]

1. BSc. Department of Biochemistry, College of Medicine, University of Basrah, Basrah, IRAQ.
2. MBChB, MRCP(UK) (Endo. & Diabetes). Professor, Department of Biochemistry, College of Medicine, University of Basrah, Basrah, IRAQ
3. MBChB, PhD. Assistant Professor, Department of Biochemistry, College of Medicine, University of Thi-Qar, Thi-Qar, IRAQ.

Received:14.1.2025 **Accepted:13.12. 2025**

Abstract

Background: Homocysteine (Hcy) is thought to increase the risk of developing cardiovascular disease (CVD). Several data indicate that there may be a relationship between hyperhomocysteinemia (HHcy) and insulin resistance (IR), which is a clinical substitute of metabolic syndrome (MetS).
Objective: To determine plasma Homocysteine level, insulin resistance, and β-cell function among patients with type 2 diabetes (T2D) and metabolic syndrome (MetS).
Methods: The study included 150 patients with T2D (75 patients with T2D and MetS, and 75 patients with T2D without MetS) and 75 apparently healthy control subjects. The diagnosis of MetS was confirmed by the updated ATPIII criteria for the definition of MetS. Blood pressure (BP), Body Mass Index (BMI), and Waist circumference (WC), plasma Homocysteine, fasting blood glucose (FBG), and insulin were determined in all participants. In addition, IR and ß- cell function were determined by Homeostatic model assessment (HOMA-IR and HOMA-B) equations, respectively.
Results: Patients with T2D with and without MetS have significantly higher WC, BMI, and systolic blood pressure (SBP), Hcy, FBG, HOMA-IR, and lower HOMA-B in comparison to the control group ($p < 0.001$). Whereas Patients with T2D and MetS have significantly higher diastolic blood pressure (DBP) and insulin levels than controls, $P<0.001$.
Conclusion: Patients with T2D with and without MetS have significantly higher Hcy levels. This may further increase the risk of cardiovascular disease (CVD) among patients with T2D.
Keywords: Homocysteine, insulin resistance, Hyperhomocysteinemia.

Corresponding author:
Mohannad I. Abbas
Email: mohanad541995@gmail.com

Introduction

During the normal production of methionine and cysteine, an intermediate result is Homocysteine, often known as Hcy, is a sulfur-containing amino acid that is not a protein [1]. It was common in animal protein and may be formed by demethylating the terminal carbon of dietary methionine [2]. Hcy levels in the blood that are more than 15 µmol/L are considered to be a medical condition that is referred to as Hyperhomocysteinemia (HHcy).It has been determined that this is a significant risk factor for the development of a number of diseases, one of which being cardiovascular disease (CVD) [3].

Type 2 diabetes mellitus (T2D) is a prevalent metabolic condition characterised by two factors: decreased insulin secretion by pancreatic β-cells and insulin resistance in sensitive tissues [4].

Diabetes is a metabolic condition that may have many different causes, according to the World Health Organisation (WHO). Diabetes is characterised by chronic hyperglycemia and disturbances in glucose, lipid, and protein metabolism owing to deficits in insulin synthesis, insulin action, or both [5].

Insulin resistance (IR) increases circulating free fatty acids (FFAs), which is thought to cause metabolic syndrome (MetS). Insulin increases muscle and liver glucose

absorption and decreases lipolysis and gluconeogenesis. Adipose tissue insulin resistance affects insulin-mediated lipolysis suppression, increasing circulating free fatty acids and decreasing insulin's antilipolytic action [6]. FFAs block muscle protein kinases, reducing glucose absorption. They increase hepatic protein kinase activity, promoting gluconeogenesis and lipogenesis. The result is hyperinsulinemia to maintain euglycemia. When compensatory mechanisms fail, insulin secretion decreases. FFAs lipotoxically inhibit insulin production from pancreatic beta cells [7].

IR reduces insulin's vasodilation and causes free fatty acid vasoconstriction, causing hypertension [8].

The glomerular filtration rate and the activity of 5, 10-methylTHF reductase are two ways in which the plasma insulin concentration affects Hcy metabolism [9]. The pathogenetic role of insulin resistance (IR) in relation to Hcy levels has been shown by research in rat models, which suggests that insulin changes the activity of metabolic enzymes involved in Hcy turnover [10], Researchers have shown that insulin resistance, as seen in polycystic ovarian syndrome patients and others, is associated with elevated Hcy levels, and that these levels correlate positively with plasma insulin concentration [11]. In Basrah, MetS and its components gained substantial interest, and various research were undertaken in this subject [12-14]. The aim of this study was to determine plasma Hcy level, IR and β-cell function among patients with T2D and MetS.

Patient and method

This is a case-control analytical study conducted in Thi-Qar Specialized center for Diabetes and Endocrinology, in Thi-Qar governorate, Southern Iraq, from December 2023 throughout April 2024. The study included 225 participants, 75 patients with T2D with MetS, they were 35 males and 40 females, 30 -70 years of age. And 75 patients with T2D without MetS, they were 35 males and 40 females, 30 -70 years of age and 75 apparently healthy individuals, matched for both age and sex with patients. T2D patients were diagnosed by specialist the doctor at the center and were divided T2D with and without MetS according to the new Adult Treatment Panel III (ATPIII) criteria for the definition of MetS, which requires the presence of three or more of the following, verified the diagnosis of MetS [15]:

1. Waist circumference (WC) ≥ 102 cm (male) or ≥ 88 cm (female).
2. Triglyceride (TG) ≥ 150 mg/dL (1.7 mmol/L).
3. Male HDL-C levels should be less than 40 mg/dL (1.0 mmol/L) and female levels should be less than 50 mg/dL (1.3 mmol/L).
4. Blood pressure (BP) ≥ 135/85mmHg or medication for hypertension.
5. Fasting plasma glucose (FPG) ≥ 100 mg/dL (5.6 mmol/L) or the presence of diabetes.

Exclusion criteria:: Type 1 Diabetes, Long-term use of folic acid and vitamin B12,Thyroid disease, drug history; oestrogen-containing oral contraceptives, corticosteroids, renal failure, heart and liver failure, Severe infection, pregnancy ,lactation . A comprehensive questionnaire was used to gather information on a variety of sociodemographic factors, such as age, sex, level of education, history of systemic disorders in the family and self, smoking. All participants underwent measurements height, weight, and waist circumference is measured while wearing light clothing and no shoes. The formula used to determine body mass index (BMI) was: (kg/m²). A mercury sphygmomanometer was used to measure systolic blood pressure (SBP) and diastolic blood pressure (DBP) twice, with a minimum 10-minute delay between measures. Determination of Hcy [16] The serum Hcy level is determined using a fully automated abbot system chemiluminescent microparticle immunoassay (CMIA) for the quantitative assessment of total Hcy in human serum or plasma., determination of Fasting blood glucose [17] measured by automated enzymatic reference method using Hexokinase kit provided by Roche Diagnostic Germany by using COBAS INTEGRA system , determination of insulin measured by using fully automated COBAS e411 system Immunoassay [18]. Determination of serum triglyceride [19] COBAS INTEGRA systems in vitro test for quantitative detection of triglycerides levels in human serum and plasma, Serum TG was estimated by enzymatic colorimetric method using Triglyceride's kit (TRIGL), supplied from Roche diagnostics, Germany. Determination of serum HDL-Cholesterol [20] In vitro diagnostic test for the quantitative determination of the HDL-C concentration in human serum and plasma on Roche/Hitachi Cobas Csystems, serum HDL-C was measured by homogeneous enzymatic colorimetric test using HDL-C Gen4 (HDLC4) kit provided by Roche diagnostics, Germany. The following equations were used to estimate β-cell function and insulin resistance (IR). [21]

HOMA-B= 360 × fasting insulin (μIU/ml) / FBG (mg/dL) - 63.

The normal value of HOMA-B is 100%.

HOMA-IR = FBG (mg/dL) x fasting insulin (μIU/ml) / 405.

The normal HOMA-IR reading is ≤2.5. Fasting blood glucose, while IR is insulin resistance. Statistical analysis was carried out using Statistical Package for the Social Sciences (SPSS) version 23. The results were reported as mean ± SD and percentages. P-values < 0.05 were deemed statistically significant.

Results

Table 1 presents the characteristics and anthropometric measurements among males. Patients with T2D with and without MetS and controls. BMI, WC, and SBP were significantly higher among patients with T2D with and without MetS than among controls, (P < 0.001). In addition, DBP was significantly higher among patients with T2D and MetS than among controls, (P < 0.001), while the mean DBP show non-significant difference between patients with T2D without MetS and controls, (P > 0.05). Also, BMI, WC, SBP and DBP among patients with T2D with MetS was significantly higher than the T2D without MetS, (P<0.001). However, there was no significant difference in age, between male patients and male controls (p > 0.05).

The characteristics and anthropometric measurements among females are presented in Table 2. Female patients with T2D with and without MetS and controls. BMI, WC, and SBP were significantly higher among patients with T2D with and without MetS than among controls, (P < 0.001). The mean DBP was significantly higher among patients with T2D and MetS than among controls, (P < 0.001), while the mean DBP show non-significant difference between patients with T2D without MetS and controls, (P > 0.05). In addition, BMI, WC, SBP and DBP among patients with T2D with MetS was significantly higher than the T2D without MetS, (P<0.001). On the other hand, there was no significant difference in age, between female patients and female controls, (p > 0.05).

Table 3 presents Hcy, FBG and insulin levels among male patients and control subjects. Hcy among was significantly higher among male patients with T2D with and without MetS than control subjects, P < 0.001. Also, Hcy level among patients with T2D with MetS was significantly higher than the T2D without MetS, (P < 0.001). Whereas, FBG among patients with T2D with and without MetS were significantly higher than controls, (P < 0.001). On the other hand, FBG show nosignificant difference between patients with T2D with and without MetS, (P > 0.05). Plasma insulin was significantly higher among patients with T2D and MetS than controls, (P < 0.001), whereas plasma insulin show no significant differences between patients T2D with and without MetS and also between patients T2D without MetS and controls, (P > 0.05).

Tables 4 demonstrate Hcy, FBG and insulin levels among females. Hcy among was significantly higher among patients with T2D with and without MetS than control subjects, P < 0.001. Also, Hcy level among patients with T2D with MetS was significantly higher than the T2D without MetS, (P < 0.001). In addition, FBG among patients with T2D with and without MetS were significantly higher than controls, (P < 0.001). On the other hand, FBG show non-significant difference between patients with T2D with and without MetS, (P > 0.05). Plasma insulin was significantly higher among patients with T2D and MetS than both patients without T2D, (P < 0.01) as well as controls, (P < 0.001). On the other hand, plasma insulin show no significant differences between female patients T2D without MetS and female controls, (P > 0.05).

HOMA-IR and HOMA-B levels among male patients with T2D with and without MetS and controls are demonstrated in Table 5. Patients with T2D with and without MetS have significantly higher HOMA- IR and significantly lower HOMA-B values among than controls, (P < 0.001). In addition, HOMA-B was significantly higher among male patients T2D and MetS than male patients with T2D without MetS, (P < 0.001). On the other hand, HOMA- IR values among patients showed no significant differences between patients with T2D with and without MetS, (P > 0.05).

Table 6 presents HOMA-IR and HOMA-B values among females. Patients with T2D with and without MetS have significantly higher HOMA- IR and significantly lower HOMA-B values among in comparison to controls, (P < 0.001). HOMA-B was significantly higher among female patients T2D and MetS compared to female patients with T2D without MetS, (P < 0.001). On the other hand, there were no significant differences between patients with T2D with and without MetS with regard to HOMA-IR, (P > 0.05).

Discussion

The present study revealed no significant differences among patients with T2D and the control group concerning sex and age. The results in this present study are consistent with other studies that have investigated the link between anthropometric measurements and Hcy level. These workers concluded that Hcy may contribute to the elevation of blood pressure. Atif et al. [22] noted an elevation of plasma Hcy levels in the majority of patients with hypertension. The study revealed that 80% of hypertensive subjects exhibited HHcy. Nabipour et al. [23] In their study

of the link between metabolic syndrome and homocysteine levels, they found no significant correlation between homocysteine levels and body mass index. However, Vayá et al. [24] identified in four studies that higher Hcy levels were mostly related with abdominal obesity. Sanlier and Yabanci [25] identified a correlation between increased body weight and HHcy, with no observed sex differences Findings from the present the study found a significant increase in mean Hcy levels in T2D subjects compared to controls, implying that high Hcy is associated with T2D. Hcy, In this study, Hcy levels were observed to be higher in males than in females and to increase with age, consistent with findings from previous studies [26]. The results in this study are consistent with other studies that have investigated the link between Hcy level and T2D [27]. Hoogeveen et al. [28] indicated the elevated serum Hcy represent a 1.6-fold greater cardiovascular disease risk factor in patients with type 2 diabetes compared to folks who are not diabetic.IR, as measured by HOMA-IR, was higher in T2D patients with hyperhomocysteinemia than in those with normal homocysteine levels. This is a feature of T2D as well as the associated MetS [29]. In this study, the likelihood of having HHcy increases significantly with the severity of IR in T2D patients. Giltay et al. [30] found the prevalence of HHcy in type 2 DM the patients are highly correlated for an intensity of IR. By using the hyperisulinemic euglycemic clamp on 24 healthy, non-obese individuals. Emoto et al. [31] similarly found that fasting insulin was an independent predictor of plasma Hcy levels in patients with T2D. However, the primary method for influencing plasma Hcy levels in patients with T2D might be described as follows: For starters, the IR is critical in the pathophysiology of type 2 diabetes. Previous research found that blood Hcy levels were higher in IR patients [32]. Additionally, in T2D patients, these studies demonstrated that IR was an independent predictor of blood Hcy levels. [33]. Regarding HOMA-B, patients with T2D demonstrated significantly low HOMA-B levels than among controls. In addition, The mean HOMA-B values among patients was distinctly lower than normal value.Other investigations found a substantial independent connection between insulin releasing function of pancreatic islet β-cells and blood Hcy levels. This correlation suggests a connection between the pathophysiology of T2D and HHcy. [34]. Pancreatic β-cell activity diminishes in both types of diabetes, leading to a worsening of glycaemic control. In T2D, impaired β-cell activity is primarily linked to IR, which is commonly accompanied by hyperlipidaemia and obesity. T2D is characterised by loss of β-cell function, which can occur progressively prior to diagnosis. Obese people with impaired fasting glucose show a 40% drop in β-cell mass, whereas those with overt T2D have a 60% loss [35].

In conclusion, patients with T2D with and without MetS have significantly higher Hcy levels compared to controls. Also, patients with T2D have significantly higher HOMA-IR and significantly lower HOMA-B than control subjects. These findings considerably increase the risk of CVD and CV events among patients with T2D.

This study had some limitations, we did not provide the plasma folic acid and vitamin B12 levels in the patients and the sample size is not large. The large-scale sample size could make the conclusions more credible.

References

1. HERMANN, Anton; SITDIKOVA, Guzel. Homocysteine: biochemistry, molecular biology and role in disease. Biomolecules, 2021, 11.5: 737.
2. D'SOUZA, Stephen W.; GLAZIER, Jocelyn D. Homocysteine metabolism in pregnancy and developmental impacts. Frontiers in cell and developmental biology, 2022, 10: 802285.
3. SON, P.; LEWIS, L. Hyperhomocysteinemia.[Updated 2020 May 21]. *StatPearls [Internet]; StatPearls Publishing: Treasure Island, FL, USA*, 2022.
4. ZHAO, Xuefei, et al. The crucial role and mechanism of insulin resistance in metabolic disease. *Frontiers in endocrinology*, 2023, 14: 1149239.
5. DILWORTH, Lowell; FACEY, Aldeam; OMORUYI, Felix. Diabetes mellitus and its metabolic complications: the role of adipose tissues. *International journal of molecular sciences*, 2021, 22.14: 7644.
6. ZHAO, Xuefei, et al. The crucial role and mechanism of insulin resistance in metabolic disease. *Frontiers in endocrinology*, 2023, 14: 1149239.
7. SRIRAMAN, Rajagopalan; TOOKE, John E. Endothelial dysfunction and insulin resistance. *Metabolic Syndrome and Related Disorders*, 2004, 2.2: 129-136.
8. SAKR, Hussein F., et al. Insulin resistance and hypertension: mechanisms involved and modifying factors for effective glucose control. *Biomedicines*, 2023, 11.8: 2271.
9. TOLEDO, Mateus Zucato. *Feeding Rumen-Protected Methionine During Pre-and Postpartum Period in Dairy Cows: Impacts on Plasma Amino Acids,*

Production, Reproduction, and Health. The University of Wisconsin-Madison, 2021.

10. Zhao X, An X, Yang C, Sun W, Ji H, Lian F. The crucial role and mechanism of insulin resistance in metabolic disease. Frontiers in endocrinology. 2023 Mar 28; 14:1149239.
11. BHUSHAN, Rashmi; SINHA, Parul. Correlation of serum homocysteine levels and hyperinsulinaemia with body mass index in polycystic ovarian syndrome. *Journal of human reproductive sciences*, 2022, 15.1: 34-41.
12. BADER, Khamail Ali; MAATOOK, Majid A.; ZABOON, Ibrahim A. Metabolic syndrome distribution based on diagnostic criteria and family history among adults in Al-Basra, Iraq. *Journal of Public Health in Africa*, 2023, 14.8: 2766.
13. HAMMODI, Mariam Atif; ABD HAZZA, Mazzin; AJLAN, Salman. Prevalence of metabolic syndrome among patients with coronary artery disease in Basrah, Iraq. *Anaesthesia, Pain & Intensive Care*, 2024, 28.2: 353-357.
14. MANSOUR, Abbas Ali, et al. Prevalence of diagnosed and undiagnosed diabetes mellitus in adults aged 19 years and older in Basrah, Iraq. *Diabetes, metabolic syndrome and obesity: targets and therapy*, 2014, 139-144.
15. MORAES, Milena Lima de; URREGO, Iván Darío Castañeda; ROMERO, Gil Robert. Waist circumference cut-off points for the definition of metabolic syndrome in older adults: SABE Colombia study. *Ciência & Saúde Coletiva*, 2025, 30: e06692023.
16. LIU, Yankui, et al. Development of a chemiluminescent immunoassay based on magnetic solid phase for quantification of homocysteine in human serum. *BMC biotechnology*, 2024, 24.1: 77.
17. ADELHELM, Josefine BH, et al. Validation of cobas® pulse point-of-care testing device for blood glucose monitoring. *Scandinavian Journal of Clinical and Laboratory Investigation*, 2025, 1-6.
18. SIERSBÆK, Julie, et al. A sensitive plasma insulin immunoassay to establish the diagnosis of congenital hyperinsulinism. *Frontiers in endocrinology*, 2021, 11: 614993.
19. NGUYEN, Thuan Thi Minh, et al. Evaluation of triglycerides, total cholesterol and high density lipoprotein-cholesterol stability in human whole blood and plasma samples. *MedPharmRes*, 2024, 8.4: 274-282.
20. KAYAMORI, Yuzo, et al. Comparison of the Japan Society of Clinical Chemistry reference method and CDC method for HDL and LDL cholesterol measurements using fresh sera. *Practical Laboratory Medicine*, 2021, 25: e00228.
21. JOG, Komal S., et al. Comparison of novel biomarkers of insulin resistance with homeostasis model assessment of insulin resistance, its correlation to metabolic syndrome in south Indian population and proposition of population specific cutoffs for these indices. *Cureus*, 2023, 15.1.
22. ATIF, Alina, et al. Serum homocysteine concentrations in patients with hypertension. *Pakistan Journal of Physiology*, 2008, 4.1: 21-22.
23. NABIPOUR, I., et al. The metabolic syndrome is not associated with homocysteinemia: the Persian Gulf Healthy Heart Study. *Journal of endocrinological investigation*, 2009, 32.5: 406-410.
24. VAYÁ, Amparo, et al. Homocysteine levels and the metabolic syndrome in a Mediterranean population: a case-control study. *Clinical hemorheology and microcirculation*, 2011, 47.1: 59-66.
25. SANLIER, Nevin; YABANCI, Nurcan. Relationship between body mass index, lipids and homocysteine levels in university students. *JPMA. The Journal of the Pakistan Medical Association*, 2007, 57.10: 491.
26. FENG, Hongru, et al. Study on the relationship between homocysteine and general metabolic indexes in healthy population in Hebei Province, China. *Frontiers in Endocrinology*, 2025, 16: 1523157.
27. KHAN, Gazala Afreen, et al. Genotype and Allele Frequency of Methylenetetrahydrofolate Reductase 677CTmutation in Female Arabs residing in the United Arab Emirates. *Journal of Medicine*, 2024, 25.2: 141-148.
28. SAENZ-PIPAON, Goren, et al. The role of circulating biomarkers in peripheral arterial disease. *International Journal of Molecular Sciences*, 2021, 22.7: 3601.
29. YANG, Xiaoyue, et al. Associations between serum folate level and HOMA-IR in Chinese patients with type 2 diabetes mellitus. *Diabetes, Metabolic Syndrome and Obesity*, 2023, 1481-1491.
30. ZHAO, Wenyan, et al. Association of homocysteine and insulin resistance with increased risk of mortality in a nondiabetic population: third national health and nutrition examination survey. *Metabolic Syndrome and Related Disorders*, 2022, 20.5: 255-263.

31. FU, Dejian, et al. Homocysteine levels are associated with diabetes mellitus in Chinese with H-type hypertension. *Nutrition Research and Practice*, 2024, 18.4: 511-522.
32. LI, Yang, et al. Hyperhomocysteinemia promotes insulin resistance by inducing endoplasmic reticulum stress in adipose tissue. *Journal of Biological Chemistry*, 2013, 288.14: 9583-9592.
33. SÖBÜ, Elif, et al. The association between vitamin B12, folate, homocysteine levels, and carotid intima-media thickness in children with obesity: a cross-sectional study. *Journal of Pediatric Endocrinology and Metabolism*, 2022, 35.8: 1051-1058.
34. YUAN, Xinlu, et al. Association between plasma homocysteine levels and pancreatic islet beta-cell function in the patients with type 2 diabetes mellitus: a cross-sectional study from China. *Annals of Palliative Medicine*, 2021, 10.7: 8169179-8168179.
35. SULEIMAN, Mara, et al. The role of beta cell recovery in type 2 diabetes remission. International journal of molecular sciences, 2022, 23.13: 7435.

Table 1. Characteristics and anthropometric measurements among males

Characteristic	T2D with MetS (n=35)	T2D without MetS (n=35)	Control group (n=35)
Age (year)	50.12 ± 7.65	49.38 ± 9.84	48.97 ± 12.07
BMI (kg/m^2)	32.15 ± 4.46$^{\#*}$	27.29 ± 3.09$^{\$}$	24.61 ± 1.44
WC (cm)	101.40 ± 10.26$^{\#*}$	94.26 ± 7.83$^{\$}$	85.37 ± 5.73
SBP (mmHg)	139.46 ± 10.65$^{\#*}$	129.86 ± 14.60$^{\$}$	121.43 ± 2.41
DBP (mmHg)	85.06 ± 8.51$^{\#*}$	81.26 ± 6.45	80.29 ± 1.36

Data presented as mean ±SD

#: P< 0.001 (patients with T2D and Mets vs. controls)

$: P< 0.001 (patients with T2D without Mets vs. controls).

*: P< 0.001 (Patients with T2D &Mets vs. patients T2D without MetS)

Table 2. Characteristics and anthropometric measurements among females

Characteristic	T2D with MetS (n=40)	T2D without MetS (n=40)	Control group (n=40)
Age (year)	50.78 ± 11.38	48.30 ± 10.28	45.85 ± 11.66
BMI (kg/m^2)	32.15 ± 5.26$^{\#*}$	27.72 ± 3.70$^{\$}$	23.02 ± 1.96
WC (cm)	92.03 ± 9.74$^{\#*}$	87.48 ± 6.33$^{\$}$	78.83 ± 5.42
SBP (mmHg)	136.60 ± 11.64$^{\#*}$	130.20 ± 10.66$^{\$}$	121.30 ± 2.60
DBP (mmHg)	87.10 ± 8.60$^{\#*}$	81.08 ± 6.05	80.25 ± 1.44

#: P< 0.001 (patients with T2D and Mets vs. controls)

$: P< 0.001 (patients with T2D without Mets vs. controls).

*: P< 0.001 (Patients with T2D &Mets vs. patients T2D without MetS)

Table 3. Hcy, FBG and insulin levels among males

Parameters	T2D with MetS N=35	T2D without MetS N=35	Control group N=35
Hcy (µmol/L)	13.12±3.50#*	10.37±2.09 $	5.35±1.35
FBG (mg/dL)	207.17±68.68#	202.37±50.81$	86.19±6.09
Insulin (µU/mL)	10.80±5.20#	8.72±4.12	8.30±1.66

Data presented as mean ±SD

#: P< 0.001 (patients with T2D and Mets vs. controls)

$: P< 0.001 (patients with T2D without Mets vs. controls).

*: P< 0.001 (Patients with T2D &Mets vs. patients T2D without MetS)

Table 4. Hcy, FBG and insulin levels among females

Parameters	T2D with MetS N=40	T2D without MetS N=40	Control group N=40
Hcy (µmol/L)	12.37± 2.57#*	9.99±1.86 $	5.10± 1.55
FBG (mg/dL)	217.02±63.43#	206.85±62.86$	87.59±6.16
Insulin (µU/mL)	11.17±4.16#*	9.37±3.78	8.09±1.84

Data expressed as mean ± SD
#: P< 0.001 (patients with T2D and Mets vs. controls)

$: P< 0.001 (patients with T2D without Mets vs. controls).

*: P< 0. 01 (Patients with T2D &Mets vs. patients T2D without MetS)

Table 5. HOMA-IR and HOMA-B among males

Parameter	T2D with MetS N=35	T2D without MetS N=35	Control group N=35
HOMA- IR	5.61±3.46[#]	4.29±2.19[$]	1.77±0.38
HOMA-B %	33.61 ± 22.98[#*]	26.42 ± 18.71[$]	136.24 ± 40.86

Data expressed as mean ± SD

#: P< 0.001 (patients with T2D and Mets vs. controls)

$: P< 0.001 (patients with T2D without Mets vs. controls).

*: P< 0.001 (Patients with T2D &Mets vs. patients T2D without MetS)

Table 6. HOMA-IR and HOMA-B among females

Parameter	T2D with MetS N=40	T2D without MetS N=40	Control group N=40
HOMA- IR	5.88±2.60 [#]	4.87±2.62 [$]	1.75±0.41
HOMA-B %	33.47 ± 23.78[#*]	28.99 ± 21.50[$]	126.26 ± 43.61

Data expressed as mean ± SD

#: P< 0.001 (patients with T2D and Mets vs. controls)

$: P< 0.001 (patients with T2D without Mets vs. controls).

*: P< 0.001 (Patients with T2D &Mets vs. patients T2D without MetS)