Estimation of lipid profile and some inflammatory biomarkers in patients with diabetes mellitus type 2 linked to hypertension

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Abstract

Diabetes Mellitus (DM) and hypertension are two common diseases that impact a significant section of the population . Pro-inflammatory mediators such as Interferon Gamma (IFN-γ) and C-Reactive Protein (CRP) levels are proposed to be linked to these diseases. The present study was aimed to explore the link between IFN- γ, CRP and lipid profile levels In type 2 diabetes mellitus (T2DM) connected to hypertension. The current study included 78 patients who had T2DM and 40 samples were collected from healthy people, both aged from 35 to 60 years. The total number of T2DM patients group were divided into two groups: 41 patients with Hypertension (HP) and 37 patients without HP, and according to their Body Mass Index (BMI). In addition, the relationship between gender and these diseases was also investigated. The results showed that the levels of Total Cholesterol (TC), Triglycerides (TG), Low-Density Lipoproteins (LDL), and Very Low-Density Lipoproteins (VLDL) were significantly increased in T2DM patients in both women and men. The results also showed a significant increase in the levels of TC, TG, LDL and VLDL in overweighted T2DM patients with HP. However, the levels of High-Density Lipoprotein (HDL) were significantly decreased in all T2DM patients. Moreover, the levels of CRP were significantly increased in T2DM female and male patients. However, the levels of IFN-γ significantly decreased in T2DM female and male patients. Furthermore, body fat percentage and waist/hip ratio significantly increased and were associated with Fasting Blood Sugar (FBS), TC, and CRP in T2DM patients. The findings confirm that lipid profile and pro-inflammatory parameters, such as CRP and IFN-γ, could have a significant impact on T2DM burden that is connected to the presence of HP. Therefore, regulating of lipid profile and pro-inflammatory parameters (CRP and IFN-γ) could protect against development and progression of T2DM accompanied by HP.

Introduction

The Metabolic Syndrome (MS) is a collection of metabolic abnormalities that are characterized by a number of cardiovascular risk factors that are typically linked to the buildup of fat and insulin resistance.[1] Diabetes Mellitus (DM) and Hypertension (HP) have become the foremost common worldwide diseases in the modernized and industrialized civilization. It is expected that the incidence of Type 2 Diabetes Mellitus (T2DM) will rise up to 642 million by 2040 worldwide and 140.2 million of the affected people living in Asia.[2] The incidence of diabetes has been shown to be accompanied by HP.[3] For instance, it was found that up to 25% of population with diabetes are suffering from HP. This issue

indeed becomes the threat to world health care services.[4,5] It is believed that these two diseases have always been accompanied by each other, which can result in serious health problems. Furthermore, it was demonstrated that HP in diabetic individuals increases the risk of vascular disease, nephropathy, retinopathy, cardiac disease, and stroke.[6,7] Moreover, it has been shown that diabetes-associated diseases, such as nephropathy, can also result in hypertension, particularly in Type 1 Diabetes Mellitus (T1DM) patients[8]. There is indeed a variety of risk factors causing the incidence of T2DM. Obesity has been demonstrated to be an essential risk factor that is involved in the development of T2DM.[2,9] The Quetlet's Index, or Body Mass Index (BMI), is one of the most common indicators used for total body adiposity, as in epidemiological studies it has been shown to be linked to T2DM.[10] It is well known that obesity can be greatly involved in T2DM development. On the other side, insulin deficiency or resistance has been shown to cause lipid abnormalities via impacting enzymes and lipid metabolism pathways.[11] It is well known that dyslipidemia involves abnormal concentrations of Total Cholesterol (TC), Trglycerides (TG), Low-Density Lipoproteins (LDL), and Very Low Density Lipoproteins (VLDL) in the blood.[12] The alteration of lipid profile has been observed to be implicated in T2DM occurrence that is linked to HP.[13]

It is believed that the reason behind the contribution of obesity in the incidence of T2DM is that the adipose tissues in obese patients highly secrete pro-inflammatory cytokines such as Interleukin-1β (IL-1β), Tumor Necrosis Factor-α (TNF-α) and Interleukin-6 (IL-6). Moreover, the inflammatory components can greatly affect the pancreas gland[14] which in turn affects negatively the insulin secretion. In fact, the main source for the pro-inflammatory cytokines and chemokines is the resident macrophages in the adipose tissues.[15] Previous study has demonstrated that Interferon Gamma (IFN-γ) is mainly involved in DM pathogenesis via increase the Major Histocompatibility Complex (MHC) class I and II expression on different cells such as β cells in the pancreas.[16] Thus, in the present study we highlighted the correlation of lipid profile alteration and inflammation with the presence of HP in T2DM patients according to their BMI.

Materials and Methods

Design of study

The current study was performed at the National Diabetes Center (Al Mustansiriya University) in Baghdad province. The study population involved 78 T2DM patients (34 women and 44 men), 41 with HP and 37 without HP, while 40 healthy people served as control. Both groups ranged in age from 35 to 60 years. The mean±Standard Deviation (SD) age of the two groups was 47.89 ±7.04 for women and 49.82±6.10 for men. The following were the study's inclusion criteria: i) patients with T2DM; ii) patients with poorly controlled diabetes, having Hemoglobin A1c (HbA1c) levels 8–10% upon hospital admission, after at least three months of continuous antidiabetic medication; iii) patients who provided signed informed consent to take part in the research. However, the exclusion criteria were the following: i) Patients with T1DM; ii) Women who are pregnant or lactating; iii) Patients who did not provide signed informed consent to take part in the research. The statistical method of sample size estimation considered the variations between the two groups in order to know the effect size, the standard error of the mean (SEM) of population, and the levels of statistical significance.

Calculation of BMI

The Body Mass Index (BMI) was calculated with the formulas described before (the weight in kilograms divided by the square of the height in meters - kg/m^2).[17] The two groups of the present study were further divided based on their BMI as following: the first group was with BMI 18-25 kg/m^2 (normal weight), the second group 25-30 kg/m^2 (overweight), and the third group (over 30 kg/m^2) were the obese.

Hypertension physical examinations

T2DM patients underwent to hypertension examinations. In the present study, the definition of HP was as previously described by Zhang *et al*.[18]

Blood sampling

Venous blood was collected from fasting individuals (8 to 12 hours before the test) between 8-10 AM. The blood samples were divided into two aliquots. One aliquot was dispensed in a tube containing Ethylenediaminetetraacetic Acid (EDTA) for Fasting Blood Sugar (FBS) determination, while the other aliquot was transferred into a 5 mL tube and then the samples were centrifuged (3000 rpm) at room temperature for 5 min. Then, serum samples were kept at -20°C until use for lipid profiles as well as C-reactive protein (CRP) and IFN-γ.

Biochemical measurements

Blood samples from T2DM and healthy patients were collected in Greiner Vacuette tubes (#456071, Greiner Bio-one, Kremsmunster, Austria) and then centrifuged for 5 min at room temperature. The levels of FBS and lipid profiles, such as TC, TG, HDL, LDL, and VLDL were quantified spectrophotometrically by catalytic action of peroxidase of H_2O_2 using commercial kits (87356, LP80619, 90406, 90816, BIOLABO SAS, Maizy, France) and based on the manufacturer's instructions. Moreover, using fluorescence immunoassay technology, the HbA1C rapid quantitative test (Finecare™, Guangzhou, P.R. China) was utilized to determine the HbA1C values. Briefly, identification (ID) Chip was inserted into the instrument, and 10 µL of blood was added into the buffer tube. The specimens were mixed well with buffer for 1 min by tapping the tube. Next, 75 µL of mixture was added into well of the test cartridge. The test cartridge was inserted in Finecare™ Fluorescence Immunochromatographic Analysing (FIA) meter and run to read the sample. The results were displayed after 5 min and presented as a percentage.

Serological measurements

Blood samples of healthy and T2DM patients were collected in Greiner Vacuette tubes (#456071, Greiner Bio-one, Kremsmunster, Austria) and centrifuged for 5 min at room temperature. The serum levels of CRP were measured using a sandwich ELISA kit (KHA0031, Invitrogen, Thermo Fisher, Vienna, Austria) with analytical sensitivity less than 10 pg/mL and variation range 18.75-1200 pg/mL. The levels of IFN-γ were also measured using a sandwich ELISA kit (Elabscience Biotechnology, Wuhan, China) with analytical sensitivity less than 9.38 pg/mL and variation range 15.63-1000 pg/mL. The levels of IFN-γ lower than the control group are considered low, and the levels of IFN-γ higher than the control group are considered high. Both proteins were measured based on the manufacturer's instructions.

Ethical approval

The authorization of the present study were done by taking the consent of all participants (patients and controls) in agreement with the authorized committee of the National cCenter for Diabetic Treatment in Baghdad province (reference number:134/212/2021).

Statistical analysis

The analysis of the present data was performed by applying the statistical package for the social sciences (SPSS Version 23). All data in the present study are presented as mean ± standard error of mean (mean ± SEM). Statistical analyses were done using Kruskal-Wallis one-way ANOVA test. Linear Regression analysis was used to examine the relationships between all the investigated variables. The SEM was used to provide a measure of the distance between the population mean and the mean of the sampling distribution of a sample mean. P<0.05 was deemed statistically significant.

Results

Levels of FBS, HbA1C and lipid profile in T2DM female patients with and without HP according to BMI (18-24, 25-29 and Over 30 kg/m^2)

We demonstrated that the levels of FBS were significantly elevated in women with T2DM with and without HP as compared with control group (p= 0.0001 and p= 0.0001, respectively; Figure

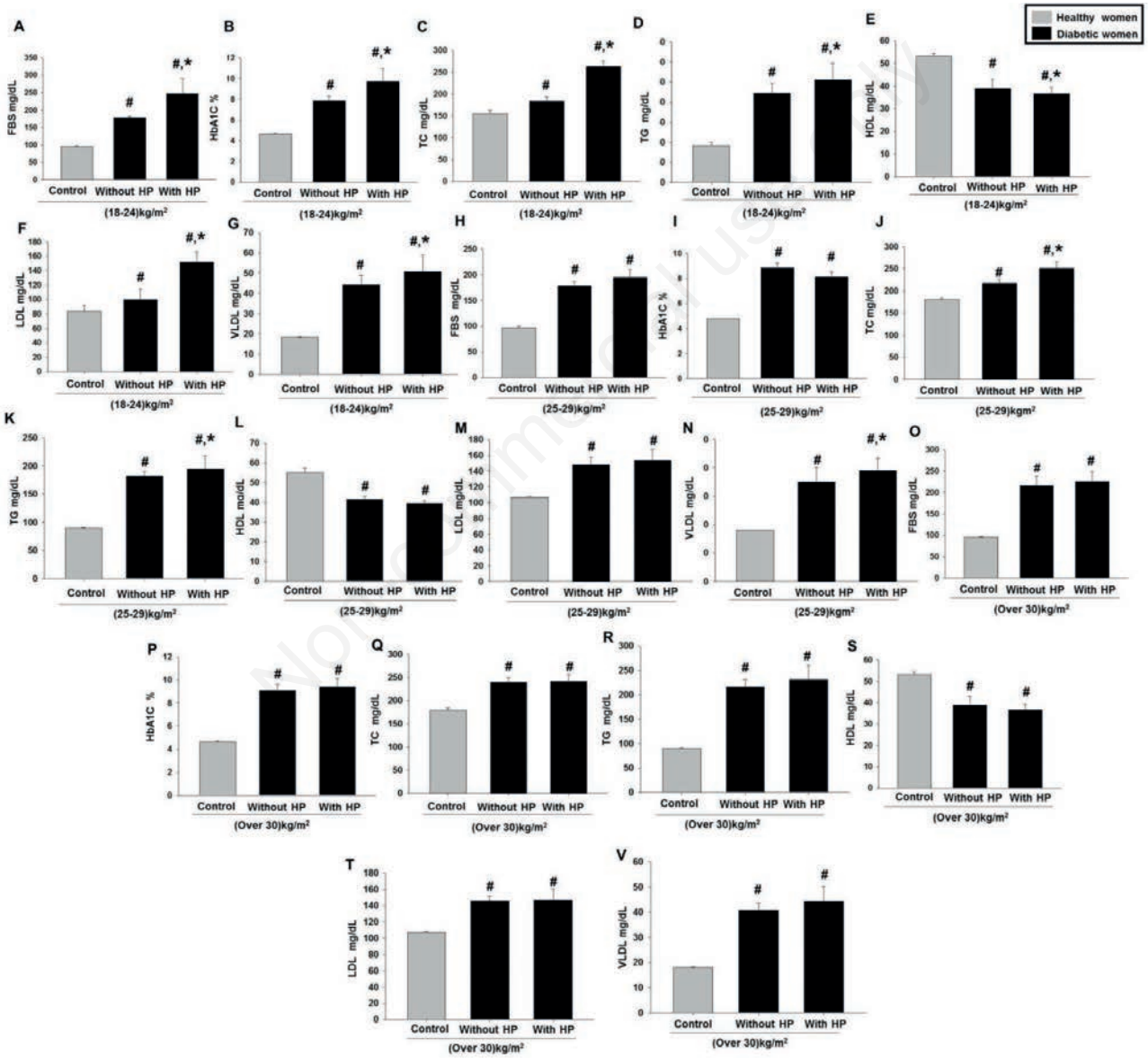

Figure 1. Serum levels of FBS (A, H, and O), HbA1C (B, I, and P), TC (C, J, and Q), TG (D, K, and R), HDL (E, L, and S), LDL (F, M, and T) and VLDL (G, N, and V) in diabetic women (n=34) according to BMI 18-24, 25-29 and over 30 kg/m^2. Black boxes represent diabetic women with and without HP. Grey boxes represent healthy control group. Data represent means ± SEM . #P <0.05 *versus* control and *P <0.05 with HP *versus* without HP.

1A). Moreover, we also noticed that the levels of FBS were higher in T2DM women with HP (247.00±43.00 mg/dL) than in those without HP (178.33±3.66 mg/dL) at F= 50.794 with 2 degrees of freedom and p= 0.001, according to BMI 18-24 kg/m^2 (Figure 1A). In addition, our results indicate significant differences in the levels of HbA1C and lipid profiles (TC, TG, HDL, LDL and VLDL) in T2DM women with and without HP as compared with control groups (Figure 1B-G).

Next, we examined the levels of FBS, HbA1C, and lipid profiles according to BMI 25-29 kg/m^2 in T2DM female patients with and without HP. We found that the serum levels of FBS, HbA1C and lipid profiles were significantly increased in T2DM female patients with and without HP comparing to control groups (Figure 1H-N). The statistical analysis showed that the levels of FBS, HbA1C, TC, TG, LDL-C, and VLDL-C increased by 47.62%, 43.39%, 31.62%, 47.36%, 52.30%, and 51.39, respectively (P=0.0001, 0.001, 0.001, 0.001, 0.0001, and 0.0001), in the serum of T2DM women (Figure 1 H-K, M and N). Conversely, we noticed that the serum levels of HDL-C significantly decreased (39.51±0.81 mg/dL and p=0.001) in these patients as compared to the control group (Figure 1L).

Then, the serum levels of FBS, HbA1C and lipid profiles were also examined in terms of BMI over 30 kg/m^2 in T2DM women. Our results showed a significant difference between women with T2DM and those in control group in regard to the serum levels of FBS, HbA1C, TC, TG, LDL-C, HDL-C and VLDL-C (Figure 1 O-V). However, serum examinations of HDL-C showed a significant reduction (32%) at F=57.103 with 2 degrees of freedom and P=0.001 in the serum levels of T2DM female patients comparing to control group (Figure 1 S). By the comparison between T2DM female patients with and without HP having BMI over 30 kg/m^2, the results showed no statistical differences in the serum levels of FBS, HbA1C and lipid profile (Figure 1 O-V).

Levels of FBS, HbA1C and lipid profile in T2DM male patients with and without HP according to BMI (18-24, 25-29 and Over 30 kg/m^2)

It was interesting to explore whether the gender factor is involved in T2DM development. In the current study, we examined the levels of FBS, HbA1C and lipid profile in men with T2DM in regard to HP according to BMI 18-24 kg/m^2 (Figure 2 A-G). Our results indicate that the levels of FBS, HbA1C, TC, TG, LDL-C, and VLDL-C were substantially higher in men with T2DM comparing with healthy control group (Figure 2 A-D, F and G). Conversely, the levels of HDL-C were significantly reduced by 37% (F= 45.619 with 2 degrees of freedom and P=0.001) in the serum of T2DM male patients as compared to control group (Figure 2 E). Then, we examined the differences between the T2DM men with and without HP in the levels of FBS, HbA1C and some lipid profile. We observed no significant differences in the serum levels of TC (Figure 2C) and HDL-C (Figure 2E) between T2DM men with and without HP.

Also, we examined the levels of FBS, HbA1C and lipid profile in T2DM men linked to HP according to BMI 25-29 kg/m^2 (Figure 2 H-N). We demonstrated that the levels of FBS, HbA1C, TC, TG, LDL-C, and VLDL-C were significantly increased in men with T2DM as compared to healthy control group (Figure 2 H-K, M and N). Whereas, our results showed that the levels of HDL-C were significantly reduced in men with T2DM compared to the control group (Figure 2L). The results demonstrated a significant increase in the levels of TC, TG, LDL-C, and VLDL-C (265.14±13.05 mg/dL, 176.57±11.40 mg/dL, 169.57±18.27 mg/dL, and 33.85±2.21 mg/dL, respectively) in T2DM men with HP compared to those without HP (197.80±5.95 mg/dL, 132.30±15.06 mg/dL, 130.00±5.84 mg/dL, and 31.10±3.46 mg/dL (P= 0.0001, 0.0007, 0.0125, and 0.01) respectively, (Figure 2 J, K, M and N).

Then, the levels of FBS, HbA1C and lipid profile were also estimated in T2DM men with HP according to BMI Over 30 kg/m^2 (Figure 2 O-V). We found that the levels of FBS, HbA1C, TC, TG, LDL-C, and VLDL-C increased significantly by 43%, 41%, 36%, 40%, 56%, and 57%, respectively (P=0.001) in the serum of T2DM men compared to the control group (Figure 2 O-R, T and V). However, the results indicate a significant reduction (34%) in HDL serum levels of T2DM men compared to control group (Figure 2 S). In addition, with respect of HP, we studied the differences between T2DM men with and without HP in term of the levels of HbA1C and some lipid profile (Figure 2 O-V). Our results showed that the levels of FBS, and LDL-C were significantly increased in T2DM men with HP compared to T2DM men without HP (Figure 2 O and T). Conversely, we found no differences between T2DM men with and without HP in the serum levels of HbA1C and some lipid profile (Figure 2 P-S and V).

Immunological response in T2DM female and male patients with and without HP according to BMI (18-24, 25-29 and over 30 kg/m^2)

The statistical analysis showed significant differences in the levels of CRP in T2DM women compared to the control group according to BMI 18-24 and 25-29, whereas, the results showed the levels of CRP were only significant in over 30 kg/m^2 group as compared with control group (Figure 3 A and C). We found that the levels of CRP are significantly increased by 40% and 46%, respectively (P=0.001) in T2DM women according to the 18-24 and 25-29 kg/m^2 BMI groups (Figure 3A and C). Considering HP, our results showed that the levels of CRP were significantly increased in T2DM women with HP compared to T2DM women without HP only in overweight subjects (Figure 3E), and showed no statistical differences between T2DM women with and without HP (Figure 3 A and C).

Our results showed that the levels of IFN-γ were decreased in the serum of T2DM women compared to healthy control group (Figure 3 B, D, and F). Additionally, we did not find any significant difference in the levels of IFN-γ between T2DM women with and without HP.

Moreover, we also examined the immune response in T2DM men. First, the levels of CRP were measured in the serum of T2DM men with and without HP as well as in the serum of healthy control group (Figure 3 G, I, and K). Our data showed a significant difference between T2DM men and healthy control group according to BMI 18-24, 25-29 and over 30 kg/m^2 (Figure 3 G, I, and K). The results indicate that the levels of CRP were significantly increased by 50%, 53% and 65%, respectively (P=<0.001) according to BMI 18-24, 25-29 and over 30 kg/m^2 (Figure 3 G, I, and K). In addition, with respect of HP in BMI 25-29 kg/m^2, we found that the levels of CRP significantly increased in T2DM men without HP compared to T2DM men with HP (Figure 3 I), while our results showed no statistical differences between T2DM men with and without HP in BMI 18-24 and over 30 kg/m^2 (Figure 3 G and K).

Moreover, we examined the role of IFN-γ in T2DM men. Our results showed that the levels of IFN-γ were reduced in the serum of T2DM men compared to healthy control group and according to BMI 18-24, 25-29 and over 30 kg/m^2, respectively (Figure 3 H, J, and L). Whereas, there were no significant differences between T2DM men with and without HP (Figure 3 H, J, and L).

Body fat percentage and waist/hip ratio and their association with FBS, TC, and CRP in T2DM female and male patients

After that, the percentage of body fat and waist/hip ratio in both T2DM women and men was examined. Notably, the results showed a clear-cut increase in the percentage of body fat in diabetic women with and without HP (F= 97.705 with 2 degrees of freedom and p=0.001) compared to control group (Figure 4 A). Moreover, the waist/hip ratio was significantly increased in diabetic women with and without HP compared to the control (F= 31.659 with 2 degrees of freedom and P=0.001; Figure 4 B). Additionally, we also examined the body fat percentage in diabetic men. The results showed a significant increase in the percentage of body fat in diabetic men with and without HP (F= 220.974 with 2 degrees of freedom and P=0.001) compared to the control group (Figure 4 C). Furthermore, the waist/hip ratio was also examined and we found that it greatly increased in diabetic men with and without HP (F= 33.831 with 2 degrees of freedom and P= 0.001; Figure 4 B).

Finally, the statistical analysis showed a strong relationship of the waist/hip ratio with FBS (y=-745.58x + 885.12; R^2=0.616, P=0.01), TC (y=-639.85x + 828.3; R^2=0.6822, P=0.001), and CRP (y=-20.066x + 22.444; R^2=0.6421, P=0.001) in diabetic women with and without HP (Figure 4 E-G). Moreover, the results showed a strong correlation of the waist/hip ratio with FBS (y=-357.72x + 526.16; R^2=0.6026, P=0.01), TC (y=-468.11x + 713.61; R^2=0.6119, P=0.001), and CRP (y=-10.469x + 14.435; R^2=0.6358, P=0.001) in diabetic men with and without HP (Figure 4 H-J).

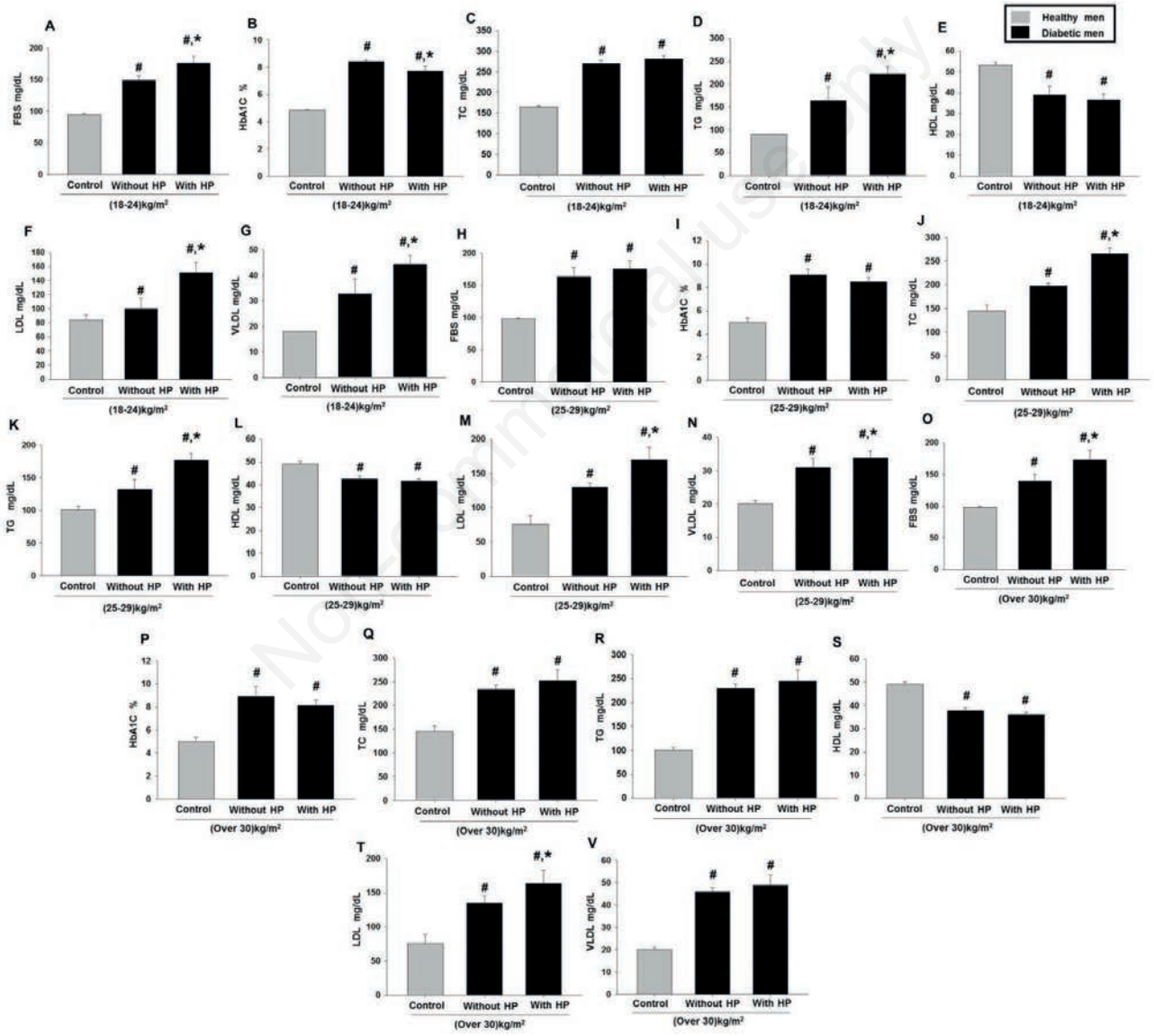

Figure 2. Serum levels of FBS (A, H, and O), HbA1C (B, I, and P), TC (C, J, and Q), TG (D, K, and R), HDL (E, L, and S), LDL (F, M, and T) and VLDL (G, N, and V) in diabetic men (n=44) according to BMI 18-24, 25-29 and over 30 kg/m^2). Black boxes represent diabetic men with and without HP. Grey boxes represent healthy control group. Data represent means ± SEM. # P <0.05 *versus* control and *P <0.05 with HP *versus* without HP.

Discussion

DM has been identified as a chronic disease linked to hyperglycemia and it can be developed via a variety of risk factors such as obesity.[2,19] In the present study, we found, not surprisingly, a significant association between investigated parameters (FBS, HbA1C, and lipid profile) and the presence of T2DM in both women and men. In addition, the levels of TC, TG, LDL-C and VLDL-C are associated with the incidence of HP in T2DM men in term of overweight (BMI 25-29 kg/m^2). Thus, the present findings suggest that regulating these lipid profiles could be a therapeutic strategy in T2DM patients (especially male patients) suffering from HP.

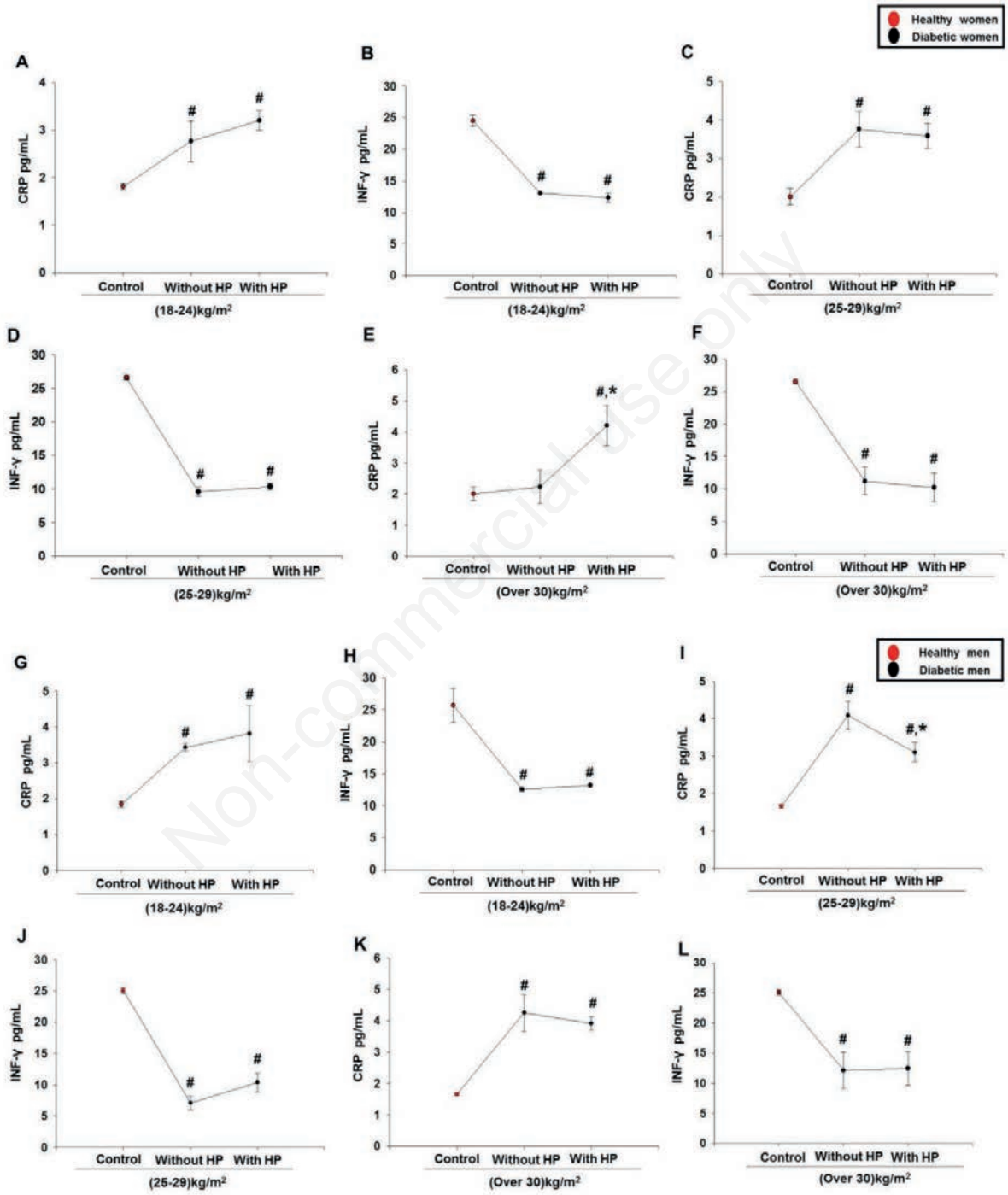

Figure 3. Serum levels of CRP and IFN-γ in diabetic women according to BMI 18-24 kg/m^2 (A and B), BMI 25-29 kg/m^2 (C and D), BMI over 30 kg/m^2 (E and F), and serum levels of CRP and IFN-γ in diabetic men according to BMI 18-24 kg/m^2 (G and H), BMI 25-29 kg/m^2 (I and J), BMI over 30 kg/m^2 (K and L). Black circles represent diabetic women or diabetic men with and without HP. Red circles represent healthy control group. Data are expressed as means ± SEM. #P <0.05 *versus* control and *P <0.05 *versus* without HP.

Recent studies found that BMI and lipid profile play an important role in the incidence of T2DM.[9,20] Moreover, BMI is known to be used as an indicator for the total body adiposity that greatly involves T2DM development.[21] In this study, we demonstrate that the levels of lipids were significantly increased in both T2DM women and men compared to the healthy control group and according to their BMI (18-24, 25-29 and over 30 kg/m^2). However, our findings indicate that the levels of HDL-C were significantly reduced in all T2DM patients compared to the healthy control group and according to their BMI (18-24, 25-29 and over 30 kg/m^2). Our results are in line with the findings by Omodanisi *et al.*[22] It is now known that a crucial element influencing insulin action is the increased availability and intracellular buildup of lipids.[23] In fact, insulin resistance can in turn led independently to abnormal lipid profile because of the event of hyperglycemia and this could explain why the levels of lipid profile were increased in T2DM patients.[24] Moreover, the insulin resistance has been implicated in excessive production of VLDL and apolipoprotein-C-III (ApoC-III) by the liver, and also in increased absorption of chylomicrons in the gastrointestinal tract. It is important to mention here that insulin resistance is also implicated in decreasing of HDL-C and apolipoprotein A-I (apA-I). Furthermore, insulin resistance leads to increasing of hydrolysis of TG, as well as in increasing of liver lipase.[25–27]

HP has been known to be a common disorder that greatly impacts the public health and this could be due to its potential consequence of stroke, heart failure and kidney disease. Subsequently, HP is a major source of morbidity and mortality.[28] HP has demonstrated to affect 30% of adults in Europe and USA with additional 30% at high risk of T2DM.[29] In the present study, we noticed that HP significantly linked to T2DM in male patients. Our results showed a substantial increase in the levels of TC, TG, LDL-C and VLDL-C in overweight T2DM men with HP. Our data are in line with a recent study that observed HP can be one of the common risk factors for DM patients.[30]

Immune responses are well known to have an essential contribution in the development of T2DM. It has been shown that

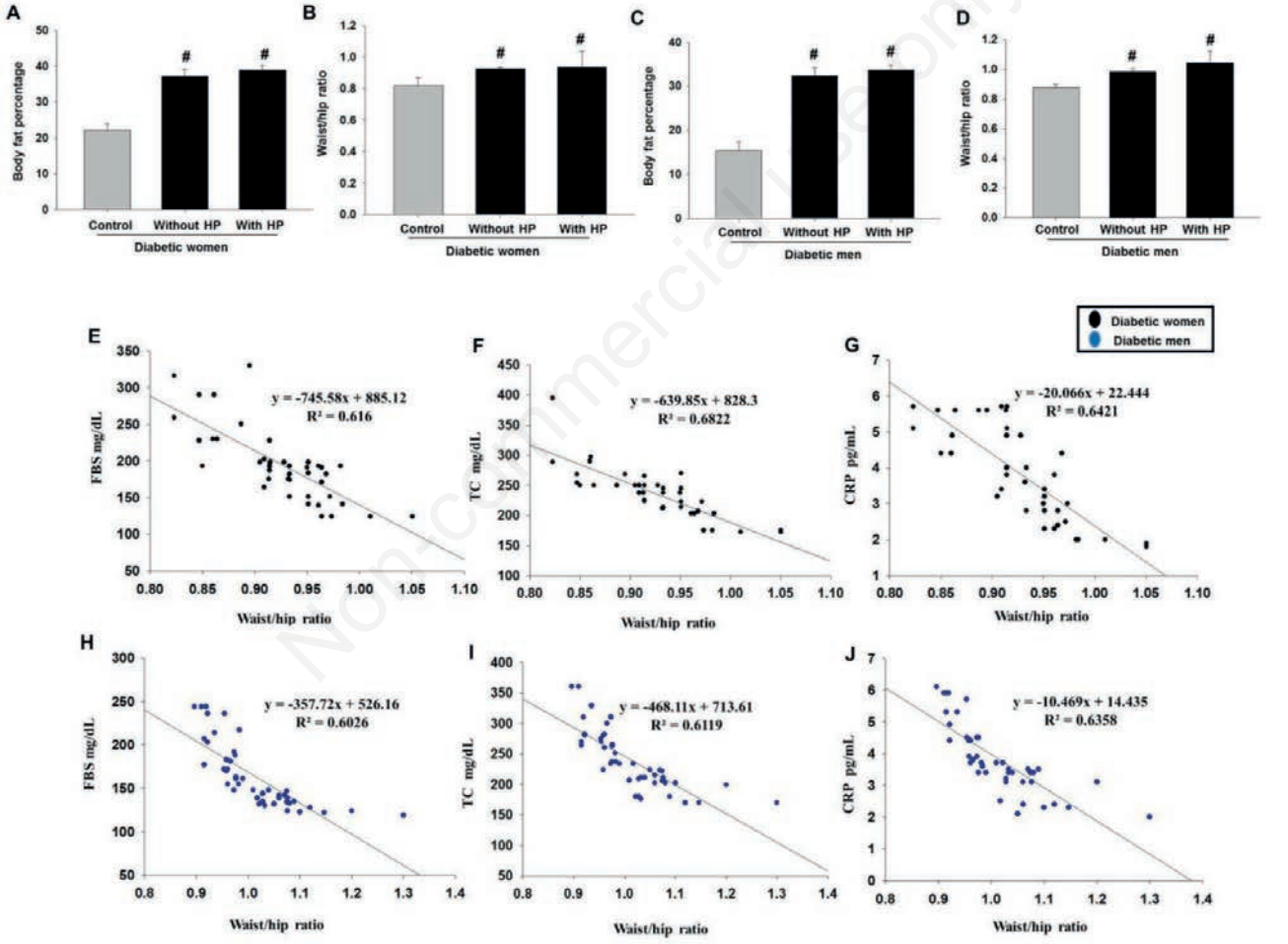

Figure 4. Percentage of body fat and waist/hip ratio and their relationship with FBS, TC, and CRP in T2DM diabetic women and diabetic men (n=78). (A) percentage of body fat and (B) waist/hip ratio in diabetic women, and (C) percentage of body fat and (D) waist/hip ratio in diabetic men. Association between waist/hip ratio with (E) FBS, (F) TC, and (G) CRP as represented by (y=-745.58x + 885.12, R^2=0.616), (y=-639.85x + 828.3; R^2=0.6822), and (y=-20.066x + 22.444; R^2=0.6421), respectively, in diabetic women. Additionally, association between waist/hip ratio with (H) FBS, (I) TC, and (J) CRP as represented by (y=-357.72x + 526.16; R^2=0.6026), (y=-468.11x + 713.61; R^2=0.6119), and (y=-10.469x + 14.435; R^2=0.6358), respectively, in diabetic men. Black circles represent diabetic women and blue circles represent diabetic men. Black boxes represent diabetic women or diabetic men with and without HP. Grey boxes represent healthy control groups. Data are expressed as means ± SEM. # P <0.05 *versus* control.

macrophages in adipose tissue of obese people greatly produce pro-inflammatory cytokines such as interleukin-1β (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α).[31] Moreover, it is believed that IFN-γ highly induces MHC class I and II expression on different cells such as β cells in the pancreas which is in turn involved in T2DM development.[31,32] In addition, it is revealed that low levels of IFN-γ have ability to provoke monocytes and macrophages to produce pro-inflammatory cytokines involved in T2DM development.[33] In the current study, we observed that low levels of IFN-γ could induce inflammation by increasing levels of CRP protein in T2DM patients. Our data is in agreement with recent findings showing that low levels of IFN-γ could participate in the release of pro-inflammatory cytokines.[33] In fact, we demonstrated that the levels of CRP were significantly increased in T2DM women (Figure 3 A, C, E), and men (Figure 3 G, I, and K). A previous study found that the levels of CRP significantly increased consistently with glucose increases in T2DM.[34–36] Moreover, increased levels of CRP have been shown to be connected with development of T2DM.[36,37] It is believed that increase levels of CRP could provoke insulin resistance via a variety of mechanisms, such as activation of adhesion molecules, complement cascade and releasing of thrombogenic factors.[35,38–41]

Some limitations have raised in the present study. First, we cannot completely rule out the possibility that there are further gender-related differences that our low sample size of 78 did not provide enough statistical power to identify. Moreover, it is difficult to figure out the role of pro-inflammatory mediators based on two parameters. The lipid profiles of T2DM linked to hypertension are provided by the study's results, which can be used for advanced lipid profile research and intervention despite these limitations.

Conclusions

The present data indicate a significant association between lipid profile and T2DM occurrence. Moreover, among lipids LDL-C has a strong association to HP in overweighed T2DM men. In addition, the low levels of IFN-γ have a powerful connection with the increased levels of CRP in both T2DM women and men. Furthermore, we have observed that the lipid profile is greatly associated with diabetes especially in patients hypertension and in both diabetic women and men. The regulation of TC, TG, LDL-C and VLDL-C could reduce T2DM burden and might attenuate diabetes incidence in the patients who are at the high risk such as those overweighed.

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