



Prevalence of Type 2 Diabetes among Adults, Duration Impact on Complications, Insulin Resistance, and Cortisol in Telafer District

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Abstract

Type 2 diabetes (T2DM) is one of the most prevalent chronic diseases worldwide, often accompanied by serious complications such as cardiovascular disease, dyslipidemia, and kidney disease. The study aimed to determine the prevalence of T2DM among adults in the Telafer district, Iraq (predominantly Turkmen), compared with previous data from Mosul city (predominantly Arabs). In addition to investigating the effect of the disease duration on the likelihood of developing common complications, insulin resistance, and cortisol levels. This community-based cross-sectional study included 350 adults, selected using stratified random sampling to determine the prevalence of T2DM. A case-control analysis was used to compare biochemical indicators and assess the prevalence of common complications among the three study groups: healthy individuals, T2DM <10 yrs, and T2DM ≥ 10 yrs. The prevalence of prediabetes and T2DM was found to be 23.14% and 19.42%, respectively. The study also indicated that obesity and family history among the district's residents played a significant role in the development of T2DM. Furthermore, the study showed a significant increase ($P < 0.05$) in the prevalence of common complications of T2DM and elevated cortisol levels over time. However, the highest insulin levels and insulin resistance index were observed in newly diagnosed patients. In conclusion, the level of T2DM among the population of the Telafer district is much higher than the one reported in Mosul. This discrepancy underlines the need for further prospective studies to explore the factors underlying this population that are demographic, genetic and lifestyle. Finally, prolonged disease is also a big part of increased incidence of common complications, insulin sensitivity and cortisol, highlighting the need for early detection and intervention.

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1. Introduction

Diabetes mellitus (DM) poses a significant challenge to the general health of the world. It is typified by persistently elevated blood sugar as a result of the failure of insulin functioning or secretion.[1]. The IDF Diabetes Atlas (2025) estimates the number of diabetic adults to be approximately 590 million across the world, which will increase to 853 million by 2050 [2]. It may cause severe complications like cardiovascular disease, nephropathy, retinopathy, and microangiopathy which can result in amputation of limbs.[3]. Type 2 diabetes (T2DM) is a civilizational disease; the latest data from the Non-Communicable Diseases Risk Factors Collaborative indicates that more than 95% of people with diabetes have T2DM [4]. It is a complex metabolic disorder characterized by high blood glucose levels resulting from tissue resistance to insulin, impaired insulin secretion, or both [5].

The duration of DM is a crucial factor in the worsening of metabolic disorders, as prolonged exposure to hyperglycemia leads to cumulative effects [6]. In this context, a recent cross-sectional study showed that approximately 61.3% of patients suffer from dyslipidemia [7]. On the other hand, a recent meta-analysis found that patients with DM for ten years or more face a

significantly increased risk of cardiovascular disease, compared to those with a shorter duration of the disease[8]. In general, the evidence confirms that the cumulative effect of T2DM promotes the overlap of dyslipidemia, obesity, and hypertension, which explains the persistent increase in cardiometabolic disease rates among patients [9].

In order to comprehend the mechanisms of T2DM complications, biological factors, including insulin resistance and cortisol levels, have to be studied [10]. It has also been reported by other studies that the degree of insulin resistance as expressed by the Triglyceride–Glucose Index (TyG) and Triglycerides/ HDL-Cholesterol (TG/HDL-c) is significantly greater in patients with T2DM compared with normal subjects [11]. Additionally, T2DM may be linked with cortisol fluctuations that are among the primary hormones to regulate metabolism and body response to stress [12]. Thus, it is valuable to follow the connection between the period of the disease and cortisol concentration to cognize changes in metabolism and hormones and, therefore, enhance preventive and therapeutic measures [13].

Regional research plays an important role in studying DM because it identifies the numerous variations that may contribute to its prevalence and the onset of its complications [14]. The Telafer district has several different socio-cultural and demographic aspects than the city of Mosul. Such differences are often accompanied by different dietary and lifestyle habits that could together contribute to the epidemiology of T2DM, but the relative importance of genetic versus environmental factors in this population is still not fully understood. Nonetheless, scientific research on the prevalence, duration and complications of the disease in the Telafer region is yet to be carried out. This gap in knowledge shows that there is a pressing need to conduct local epidemiological studies to define the health conditions of people with DM in this area. Thus, this research aims to evaluate the prevalence of T2DM in the adult population of Telafer and compare it with the findings of the earlier study on the same in Mosul city, also to determine the relationship between disease duration and chronic complications, Insulin resistance, and Cortisol levels.

2. Research Method

2.1 Study Design and Population

2.1.1 Volunteers and study protocol:

This study used a community-based cross-sectional study design to estimate the prevalence of T2DM in Telafer District, as well as a case-control study design to compare the biochemical and anthropometric measures between people with and without DM. The period of data collection was five months, between June and October 2025. The 350 adult volunteers were selected through a stratified random sampling technique at the household level to have a proportional representation of all the neighborhoods. People were randomly chosen in each household to ensure independence of data. Each Participant was subjected to structured interviews, anthropometric measurements and a blood sample was collected for biochemical analysis.

2.1.2 Exclusion Criteria:

To guarantee the validity of the diagnosis of T2DM and to decrease the effect of the confounding factors on the studied variables, the following groups were excluded: pregnant women, those with type 1 diabetes (T1DM), or hyperglycemia due to known reasons, as well as those with acute physical or psychological disorders, those taking medications that are likely to raise the level of blood glucose or cortisol, those on dialysis.

2.1.3 Epidemiological Part:

The participants were classified into three categories, namely, normal, prediabetic, and T2DM according to the American Diabetes Association (ADA) criteria based on the level of fasting blood glucose and glycated hemoglobin (HbA1c) values [15]. The differences between T2DM and T1DM diabetes were made in terms of age at diagnosis, treatment regimen, and the onset of symptoms..

2.1.4 Sample collection

All the participants contributed 5 ml of blood. Out of this volume, 2 ml was put in EDTA tubes and directly used to analyze HbA1c. The rest was put in a gel tube and left to coagulate within 20 minutes, and then centrifuged at a speed of 4000 rpm over a period of about 10 minutes. The serum samples were then kept at -20 o C to be prepared to undergo biochemical analyses. All blood samples were collected in the morning after an overnight fast and, for patients receiving insulin therapy, before their daily insulin administration.

2.2 Comparative Analysis:

2.2.1 Groups Classification:

For comparative analysis, participants were divided into three study groups:

Group 1: Individuals with T2DM diagnosed <10 yrs.

Group 2: Individuals with T2DM diagnosed ≥10 yrs.

Group 3: Control group (was selected from the healthy volunteers, using frequency matching to ensure that the age and sex distributions were consistent with those of the patient groups).

2.2.2 Serum determination of glucose and lipid profile

Fasting blood glucose levels, along with a range of lipid indices, were measured using enzyme analysis kits supplied by BIOLABO. Glucose and total cholesterol (T.C) levels were determined using the Trinder reaction-based oxidation method [16]. Triglycerides (T.G) were assessed via hydrolysis followed by the Trinder reaction [17] [16]. HDL levels were assessed according to the method described by Mohsen Ibrahim et al. (2013) [18]. All measurements were performed strictly according to the manufacturer's instructions, using a UV-Vis spectrometer at 500 nm. Low-density lipoprotein (LDL) was calculated using the Friedewald equation for samples containing triglyceride levels ≤ 400 mg/dL, while direct enzyme assays were used for samples exceeding this level [19].

2.2.3 Determination of HbA1c:

The HbA1c was assessed using the A1 Care™ HbA1c cartridge and analyzer, offered by the South Korean company i-SENS Inc., on the basis of the immuno-enzyme analysis principle.

2.2.4 Determination of Insulin hormone:

The Human Insulin Hormone Test Kit CLIA (catalogue No.: Insulin111) was used to determine serum insulin levels using a photochemical immunoassay principle, where antigen-antibody binding generates a light signal proportional to the analyte concentration [20]. Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was calculated using the formula: $\text{HOMA-IR} = [\text{glucose (mg/dL)} * \text{insulin } (\mu\text{U/mL})/405]$, using fasting values [21].

2.2.5 Determination of Cortisol hormone:

Serum cortisol levels were quantitatively determined using the Human Cortisol iCHROMA™ Kit (Catalogue No.: INS-CO-EN, Revision 10), manufactured by Boditech Med Inc., South Korea, based on an immunofluorescence principle, which is based on measuring the fluorescence intensity of antigen-antibody compounds bound to reagent antibodies, which are then read by an ichroma™ device [22].

2.3 Statistical analysis

Microsoft Excel and SPSS (version 25.0, 2021) were used for data analysis; group comparisons were carried out using ANOVA, with p-values < 0.05 deemed significant. The data were presented as means with standard error (SE).

3. Results And Discussion

3.1 Baseline Demographic Characteristics of the three study groups (healthy, T2D <10 yrs duration, and T2D ≥ 10 yrs duration)

Table 1 summarises the age and sex distribution across the three study groups: Control, T2DM <10 yrs, and T2DM ≥ 10 yrs. Statistical analysis using one-way ANOVA showed that the mean age was similar between the healthy control group and the two diabetic groups ($P = 0.095$). Furthermore, analysis of sex composition using the chi-square test showed no statistically significant difference between the groups ($P = 0.569$). In clinical studies, eliminating demographic bias is crucial to ensuring the reliability of the results. Therefore, the apparent homogeneity of the samples used in our current study across the three groups provides a standardised basis for subsequent biochemical and hormonal comparisons.

Table 1: Distribution of age and sex across the three study groups

Variable	Control group (n = 38)	T2DM <10 yrs (n = 38)	T2DM ≥ 10 yrs (n = 30)	P-value	Statistical test
Age (yrs) Mean $\pm$ SD	49.1 $\pm$ 12.1	46.2 $\pm$ 11.0	52.5 $\pm$ 12.1	0.095	One-way ANOVA
Sex (M/F)	18/20	21/17	18/12	0.569	Chi-square test

*Results interpreted as mean $\pm$ SD

P-value corresponding to the ANOVA and Chi-square tests.

A statistical test was done after considering the P-value = 0.05

3.2 Prevalence of prediabetes and T2DM within the study population

The current study included 350 adults from Telafer District, comprising 167 males (47.7%) and 183 females (52.3%). As shown in Table 2, overall prevalence analysis revealed that 23.14% of participants were identified as having prediabetic, while 19.42% as having T2DM. Regarding data classification by gender, the prevalence of both prediabetes and T2DM was higher among males compared to females; however, these differences did not reach statistical significance ($P > 0.05$).

Table 2: Distribution of participants according to glycemic status (Healthy, Prediabetic, and T2DM) among the population in Telafer district, with gender-based analysis.

Status	Total n (%)	Males n (%)	Females n (%)	P-value (Males vs. Females)
Healthy	201 (57.42)	87 (52.09)	114 (62.29)	0.057
Prediabetes	81 (23.14)	44 (26.34)	37 (20.21)	0.437
T2DM	68 (19.42)	36 (21.55)	32 (17.48)	0.628

* Results interpreted corresponding to the Chi-square test.

A statistical test was done after considering the P-value = 0.05

In fact, our findings in the Telafer district of Iraq were alarming compared to a 2024 study in Mosul (the capital of Nineveh Governorate), which indicated a significantly lower prevalence of prediabetes and T2DM, at 18.78% and 11.46%, respectively. That same study also reported a higher percentage of females with prediabetes than males, while a higher percentage of males with T2DM. This variation is a reflection of important epidemiological differences. The difference could be related to socio-environmental, dietary or ancestral factors, but in this study no genetic testing was done, so the results are only a demographic baseline.

The pattern of our findings is similar to that of international studies in other groups of ethnic populations. Recently, a study in the United States was completed and found that ethnic minorities (Hispanic and Black) had higher prevalence of DM than whites [23]. A Norwegian study also found that the rate of T2DM is significantly higher among immigrants from Asia and Africa compared to the native population [24]. Another UAE study revealed that UAE citizens had the highest prevalence rate of DM and prediabetes among the Arab and African residents of the UAE [25]. Recent studies suggest that differences in prevalence of DM in ethnic groups may be attributable to environmental and social factors, including income, education and housing, as well as to biological differences [26]. The risk of diabetes is also different across ethnic groups due to variation in physical activity and dietary patterns [27]. A recent review has shown that from a genetic point of view, the susceptibility to type 2 diabetes is linked to genetic variations between races, such as LCAT, APOE and FTO genes [28]. Lastly, the higher prevalence of T2DM and prediabetes in males than in females in our current study could be attributed to a number of biological, social, and behavioral differences. But in some communities men may be at higher risk due to unhealthy dietary patterns. In addition, premenopausal women are somewhat protected from risks due to their hormones; estrogen enhances glucose tolerance, and insulin sensitivity [29].

3.3 Comparative assessment of common clinical risk factors for T2DM between the healthy group and the patient group

Table 3 shows that the prevalence of obesity and a family history of T2DM were significantly higher ($P < 0.05$) in the diabetic group compared to the healthy group. However, there was no significant difference ($P > 0.05$) in the proportions of sedentary individuals and those with unhealthy diets between the two groups. Based on the above, we tentatively conclude that both fat accumulation and genetics are major contributors to the development of T2DM among residents of Telafer.

Table 3: Prevalence of common clinical risk factors in healthy and T2DM groups

Common risk factors of T2DM (n / %)	Healthy (n = 38)	T2DM (n = 68)	P-value
Obesity	7 / 18.4 %	26 / 38.2 %	0.03
Family history of T2DM	9 / 23.7 %	33 / 48.5 %	0.012
Sedentary lifestyle	24 / 63.1 %	49 / 72.1 %	0.343
Unhealthy diet	13 / 34.2 %	21 / 30.9 %	0.725

Obesity: BMI ≥ 30 kg/m².

Family history of T2DM: Includes first-degree relatives only (parents or siblings).

Sedentary lifestyle: Less than 150 minutes per week of moderate physical activity.

Unhealthy diet: High intake of sugars/fats and low intake of fruits, vegetables, and fibers

* Results interpreted corresponding to the Chi-square test

A statistical test was done after considering the P-value = 0.05

Several studies have demonstrated a strong correlation between the development of T2DM and obesity, family history, and advancing age. This is due to the association of these factors with IR and relative insulin deficiency [30]. Among the most recent studies is one conducted in Abha, Saudi Arabia, which found that 46.0% of individuals with T2DM were obese overall [31]. Another recent study conducted in Utah, USA, found that the risk of developing T2DM is strongly associated with having first-degree relatives with the disease. This study also indicated that obesity plays a mediating role between a family history of

DM and an individual's risk of developing the disease [32]. Supporting the latter point is the finding by Coral et al. (2023) that researchers have identified 48 single nucleotide polymorphisms (SNPs) associated with obesity and the inheritance of T2DM [33]. The excessive fat accumulation plays a key role in modulating numerous physiological functions, including appetite and insulin sensitivity, through disrupted adenokines and axosome secretion, resulting in a chronic inflammatory state and abnormal gene signaling [34]. Furthermore, high levels of free fatty acids (FFAs) directly contribute to a relative inhibition of enzymes in the glycolysis cascade, leading to a reduced ability of cells to absorb and metabolize glucose [35].

The results of the lack of statistically significant differences in sedentary lifestyle and unhealthy diet between the two type 2 diabetic patients and healthy individuals may suggest a similar behavior pattern. The instruments used were based on the (World Health Organization and American Diabetes Association) criteria, but may not have been sensitive enough to measure small changes in intensity of physical activity or individual dietary items, which could account for the non-statistically significant results.

3.4 Comparative assessment of common complications of T2DM across the three groups (healthy individuals, T2DM <10 yrs duration, and T2DM ≥10 yrs duration).

Table 4 shows a statistically significant ($P < 0.05$) progressive increase in the prevalence of heart diseases, hypertension, dyslipidemia, and impaired kidney function, starting from the healthy group through the group with T2DM for <10 yrs duration, up to the group with T2DM for ≥10 yrs duration. Regarding dyslipidemia, Table 5 confirmed the above findings. Statistically significant differences ($P < 0.05$) were observed in all lipid indices across the three groups. Overall, the analysis reveals a gradual deterioration in lipid levels, correlated with the presence and duration of diabetes. When compared to the healthy control group, TC, TG, and LDL cholesterol levels were significantly higher in the T2DM for <10 yrs duration, with an even more pronounced increase observed in the T2DM for ≥10 yrs duration. In contrast, a gradual decrease in HDL cholesterol levels was observed.

Table 4: Prevalence of common complications of T2DM across the three groups (healthy, T2DM <10 yrs , T2DM ≥10 yrs)

Common complications of T2DM (n / %)	Healthy (n = 38)	T2DM <10 yrs (n = 38)	T2DM ≥10 yrs (n = 30)	P-value
Dyslipidemia	6 / 15.8 %	11 / 28.9 %	18 / 60.0 %	0.0001
Heart diseases	5 / 13.2 %	7 / 18.4 %	13 / 43.3 %	0.009
Hypertension	6 / 15.8 %	15 / 39.4 %	21 / 70.0 %	0.0001
Impaired kidney function	3 / 7.9 %	7 / 18.4 %	13 / 43.3 %	0.002

* Results interpreted corresponding to the Chi-square test

A statistical test was done after considering the P-value = 0.05

Table 5: Blood lipid profile levels across the three groups (healthy, T2DM <10 yrs, T2DM ≥10 yrs)

Biomarker	Healthy (n=38) Mean ± SD	T2DM <10 yrs (n=38) Mean ± SD	T2DM ≥ 10 yrs (n=30) Mean ± SD	P- Value
T.C (mg/dL)	175.7 ± 18.8	196.6 ± 24.1	232.1 ± 33.2	0.0001
HDL (mg/dL)	53.4 ± 8.7	48.1 ± 9.9	40.0 ± 9.9	0.0002
LDL (mg/dL)	94.1 ± 25.2	116.0 ± 20.1	144.5 ± 31.4	0.0001
T.G (mg/dL)	118.0 ± 21.9	158.3 ± 27.5	208.4 ± 42.4	0.0001

* Results interpreted as mean ± SD

P-value corresponding to the ANOVA test.

A statistical test was done after considering the P-value = 0.05

The results of several recent studies are consistent with our current findings. For example, a recent study conducted in South Korea found a significant association between longer duration and increased incidence of dyslipidemia, hypertension, and chronic kidney disease, and increased risk of myocardial infarction and stroke even at low LDL levels [36]. A Turkish review published in 2025 stated that a long duration of T2DM increases the likelihood of hypertension, and that co-occurring the two more than doubles the risk of cardiovascular and kidney diseases [37].

IR in adipose tissue leads to elevated levels of FFA in the blood. This results in increased production of very low-density lipoproteins (VLDL) by the liver and impaired lipid metabolism. This, in turn, leads to decreased levels of HDL and the formation of small, dense LDL particles. Furthermore, the accumulation of lipid mediators further disrupts insulin signalling pathways and exacerbates metabolic dysfunction [35]. In parallel, chronic hyperglycemia contribute to endothelial dysfunction, increased oxidative stress, and activation of inflammatory pathways, leading to hypertension through mechanisms including sodium

retention, activation of the renin-angiotensin-aldosterone system [37]. At the kidney level, chronic exposure to hyperglycemia and hypertension leads to structural disfunction in glomeruli, resulting in hyperfiltration and albuminuria. Also, increased Reactive Oxygen Species (ROS) production activate the inflammatory pathway through increased inflammatory cytokines and chemokines, which in turn activate growth factors such as Transforming Growth Factor Beta (TGF- β) and Connective Tissue Growth Factor (CTGF), ultimately leading to renal fibrosis [38]. Evidence also suggests that the gradual accumulation of the above mentioned harmful effects of chronic hyperglycemia increases the risk and severity of these complications [5].

3.5 Fasting Insulin, IR, and cortisol alterations across the three study groups (healthy, T2DM <10 yrs , and T2DM $\geq$ 10 yrs)

Table 6 demonstrates clear and statistically significant differences ($P < 0.05$) among the three groups with respect to fasting values for insulin levels, IR index, and cortisol levels. Insulin and IR values were significantly higher in T2DM patients compared to healthy individuals. This increase was more pronounced in patients with T2DM <10 yrs compared to those with T2DM $\geq$ 10 yrs, although values in both diabetic groups remained higher than in the control group. Cortisol levels showed a different pattern, with a slight increase in the hormone level observed in the T2DM < 10 yrs group compared to the healthy group, while in patients with T2DM $\geq$ 10 yrs, cortisol levels increased dramatically, which explains the overall statistical significance.

Table 6: Insulin, HOMA-IR, and Cortisol levels across the three study groups

Parameters	Healthy (n=38) Mean $\pm$ SD	T2DM <10 yrs (n=38) Mean $\pm$ SD	T2DM $\geq$ 10 yrs (n=30) Mean $\pm$ SD	P- Value
Fasting insulin (μ IU/mL)	6.6 $\pm$ 2.3	23.5 $\pm$ 8.2	16.1 $\pm$ 8.3	0.0001
(HOMA-IR)	1.42 $\pm$ 0.57	5.70 $\pm$ 1.80	3.52 $\pm$ 1.14	0.0001
Fasting cortisol (μ g/dL)	11.94 $\pm$ 5.01	12.75 $\pm$ 4.02	15.95 $\pm$ 4.97	0.002

* Results interpreted as mean $\pm$ SD

P-value corresponding to the ANOVA test.

A statistical test was done after considering the P-value = 0.05

The results of our current study are consistent with a longitudinal American study on patients with T2DM , which found that C-peptide secretion decreased with disease duration in approximately half of patients [39]. A South Korean study (2024) confirmed that beta-cell function in T2DM deteriorates with disease duration; however, the most influential factor is not disease duration alone, but rather the current glucose level (HbA1c) at the time of testing [8]. Conversely, an Iraqi study indicated the opposite results [40]. This decrease in insulin secretion could be attributed to beta-cell depletion resulting from metabolic stress, particularly glycolipotoxemia, in addition to fibrosis and inflammatory changes [41]. What reinforces the results of our study is that most patients in the third group ($\geq$ 10 yrs) required insulin injections, indicating that their reduced IR does not reflect actual improvement, but rather a decline in the beta cells' ability to produce the hormone. Regarding cortisol levels, a 2020 study conducted in Gaza showed that T2DM patients have higher levels of the hormone compared to healthy individuals. This increase was directly proportional to the duration of the disease and was explained by hyperactivity of the Hypothalamic–Pituitary–Adrenal Axis (HPA) resulting from continuous stress, in addition to chronic psychological tension and the complications associated with the disease [42]. It is worth noting here that hypercortisol is also implicated in exacerbating the pancreas's insulin deficiency, increasing IR, and raising blood glucose levels, creating a vicious cycle that worsens metabolic status [43]. These findings highlight the importance of early intervention to reduce pancreatic exhaustion. Also emphasizing the need to consider the role of cortisol as a stress hormone in the disease process, this opens the door for broader future studies to investigate its long-term effects on DM patients.

4. Study Limitations:

While the current study makes substantive contributions, there are a number of limitations that need to be noted. One of the most significant of these was the cross-sectional design, which was not able to definitively determine causal relationships between the length of disease and subsequent metabolic and hormonal abnormalities. In addition, the study was conducted in one part of Iraq, which may restrict the generalizability of the results to Turkmen groups in other regions of Iraq. Also, some nuances of individual differences in the nuances of nutrition and habitually active were not taken into account by the study, which can influence the metabolism pattern. Last, while we have enough statistical power from our 3-month time frame and sample size to support the core hypotheses, we do appreciate larger cohorts. Future studies are recommended to use wider, multi-center, prospective, longitudinal designs for better generalizability.

5. Conclusion:

This work shows a markedly high prevalence of T2DM and prediabetes among adults in Telafer district than the previous data from Mosul. The results indicate that the regional demographic and environmental factors should be explored more in-depth to better understand the precise role of these factors in disease development. We also found that the longer the duration of the disease, the greater the risk of common complications, the more elevated the cortisol levels and the more stressed the beta-cells. Therefore, the results of this study highlight the need to monitor the metabolic syndrome longitudinally and provide a base for future prospective studies that can be used for genetic analysis and further provide personalized preventive and therapeutic measures for this population.

6. Acknowledgments

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7. Declarations

7.1 Ethics approval and consent to participate

This study complied with the Declaration of Helsinki regarding sampling. Informed consent was obtained from all volunteers, and their data and identities were protected.

7.2 Consent for publication

Not applicable.

7.3 Availability of Data and Materials

Data will be provided upon receiving a valid request.

7.4 Conflicts of interest

The author declares that there is no conflict of interest

7.5 Funding

The author did not receive any funding.

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انتشار داء السكري من النوع الثاني بين البالغين، وتأثير مدة الإصابة على المضاعفات، ومقاومة الأنسولين، ومستويات الكورتيزول في قضاء تلعفر

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الخلاصة:

يُعد داء السكري من النوع الثاني (T2DM) أحد أكثر الأمراض المزمنة انتشاراً على مستوى العالم، وغالباً ما يترافق مع مضاعفات خطيرة كأمراض القلب والأوعية الدموية، واضطرابات دهون الدم، وأمراض الكلى. هدفت الدراسة لتحديد معدل انتشار السكري من النوع الثاني بين البالغين في قضاء تلعفر بالعراق (ذي الأغلبية التركمانية)، ومقارنة النتائج ببيانات سابقة مستمدة من مدينة الموصل (ذات الأغلبية العربية). كما بحثت الدراسة في تأثير مدة الإصابة بالمرض على احتمالية حدوث المضاعفات الشائعة، ومقاومة الأنسولين، ومستويات الكورتيزول. اعتمدت الدراسة المنهج المقطعي الميداني (Community-based cross-sectional study) وشملت 350 بالغاً تم اختيارهم باستخدام أسلوب المعاينة العشوائية الطبقية. كما استُخدم تحليل الحالات والشواهد (Case-control analysis) للمقارنة بين المؤشرات البيوكيميائية وتقييم انتشار المضاعفات الشائعة عبر ثلاث مجموعات: الأصحاء، ومرضى السكري بمدة إصابة أقل من 10 سنوات، ومرضى السكري بمدة إصابة 10 سنوات فأكثر. أظهرت النتائج أن معدل انتشار مقدمات السكري وداء السكري من النوع الثاني بلغ 23.14% و 19.42% على التوالي. كما بينت الدراسة أن السمنة والتاريخ العائلي المرضي يؤديان دوراً جوهرياً في تطور الإصابة بالمرض بين سكان القضاء. علاوة على ذلك، لوحظ ارتفاع معنوي إحصائياً ($P < 0.05$) في معدل انتشار المضاعفات الشائعة للسكري ومستويات الكورتيزول مع مرور الوقت. وفي المقابل، سُجلت أعلى مستويات للإنسولين ومؤشر مقاومة الإنسولين لدى المرضى حديثي التشخيص. استنتجت الدراسة أن معدل انتشار داء السكري من النوع الثاني في قضاء تلعفر أعلى بشكل ملحوظ مقارنة بالنتائج المسجلة مسبقاً في مدينة الموصل. ويؤكد هذا التباين على الحاجة إلى مزيد من الدراسات المستقبلية لاستكشاف العوامل الكامنة وراء هذه الفئة السكانية، وهي العوامل الديموغرافية والوراثية ونمط الحياة. وأخيراً، يُعد طول مدة المرض أيضاً جزءاً كبيراً من زيادة معدل حدوث المضاعفات الشائعة، وضعف حساسية الأنسولين، ومستويات الكورتيزول، مما يُبرز الحاجة إلى الكشف المبكر والتدخل المناسب.