OXIDATIVE DNA DAMAGE AND METABOLIC RISK FACTORS IN PREDIABETES: A CASE-CONTROL STUDY IN DUHOK, IRAQ

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ABSTRACT

Background: Prediabetes, a precursor to type 2 diabetes mellitus, defined by glucose metabolism abnormalities resulting from insulin resistance and/or β-cell dysfunction. Recent interest has emerged in oxidative DNA damage as a potential contributor to metabolic dysregulation, particularly via 8-hydroxy-2'-deoxyguanosine (8-OHdG), a biomarker of oxidative stress.

Objective: This study aimed to evaluate serum levels of 8-OHdG in prediabetic individuals and explore its relationship with some metabolic risk factors in comparison to healthy individuals.

Methods: A case-control study was performed on 160 participants (80 prediabetic and sex and age matched 80 healthy individuals) at the Central Laboratory in Duhok, Iraq. Clinical and biochemical assessments included BMI, fasting blood sugar (FBS), HbA1c, insulin, HOMA-IR, and serum 8-OHdG levels. ELISA was used to quantify 8-OHdG. Data were statistically analyzed using SPSS v25, with significance set at $p \leq 0.05$.

Results: Individuals with prediabetes exhibited markedly elevated serum 8-OHdG levels (5.13±1.12 ng/ml) in comparison to healthy controls (3.66±1.02 ng/ml, $p<0.01$), along with elevated FBS, HbA1c, insulin, and HOMA-IR. No significant gender disparities in 8-OHdG levels were found. Higher levels of 8-OHdG were noted in Prediabetics with increased BMI, central obesity, and insulin resistance. A similar trend was seen in healthy individuals, but the differences were not statistically significant.

Conclusion: The study highlights a significant association between oxidative DNA damage and prediabetes, particularly in overweight, centrally obese, and insulin-resistant individuals. These findings indicate that oxidative stress plays a pivotal role in early glucose metabolism disturbances and could be a potential target for early intervention strategies.

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Keywords: Prediabetes, Oxidative stress, 8-OHdG, Insulin resistance, Central obesity, DNA damage.

Prediabetes is often asymptomatic but this metabolic glucose derangement poses a considerable risk to progress to type 2 diabetes mellitus over time[1]. The pathophysiology behind glucose abnormality in prediabetes is mainly driven by two main ways: insulin resistance and/or β-cell dysfunction[2]. Together, these two pathologies lead to metabolic imbalance that predisposes to glucose intolerance, particularly in the presence of other risk factors as central obesity, sedentary lifestyle, dyslipidemia and hypertension[3,4]. Oxidative stress regarded as a significant contributor to the pathogenesis of prediabetes as well as diabetes[5]. Oxidative stress results from an imbalance between the production of reactive oxygen species and the body's ability to neutralize them[6]. In prediabetic states, hyperglycemia-induced oxidative stress can exacerbate insulin resistance by

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disrupting insulin signaling pathways and promoting chronic low-grade inflammation[7]. Reactive oxygen species also leads to endothelial dysfunction by diminishing nitric oxide bioavailability, contributing to vascular complications even before progressing to clinically evident diabetes[8]. Deoxyribonucleic Acid (DNA) strand breaks and base modifications in its structure occurs due to Reactive oxygen species, including oxidation of guanine residues and lead to production of 8-hydroxy-2'deoxyguanosine (8-OHdG) - an oxidized nucleoside of DNA[9]. Measuring the serum level of 8-OHdG which is a recognized biomarker of oxidant-induced DNA damage has been used in many researches using Enzyme-Linked Immunosorbent Assay (ELISA) and High-performance liquid chromatography (HPLC) methods[10]. 8-OHdG is the most frequently detected and studied DNA lesion[11]. In recent years, several clinical studies have analyzed serum levels of 8-OHdG in relation to many metabolic disturbances including prediabetes and diabetes[12]. Though evidence exists to indicate an association between oxidative DNA damage and metabolic dysfunction, the underlying processes involved demand further investigation in the context of prediabetes. Additionally, there are little data available in our locality regarding the link between prediabetes and oxidative DNA damage which prompt us to carry this study.

Aim: To investigate the serum levels of 8-OHdG and its association with some related risk factors in subjects with Prediabetes in comparison to healthy individuals.

MATERIALS AND METHODS:

This case controls study was conducted at central laboratory in Duhok governorate, Kurdistan region of Iraq, from October 2024 to January 2025, involving individuals visiting the lab for annual checkup. Apparently healthy individuals from health care workers, college students and their relatives asked to participate in the study as volunteers and those who agreed were introduced to the study. A non-probability convenience sampling method was employed, selecting individuals who met the selected criteria for prediabetes screening. Prior to sampling, all participants provided with detailed and clear explanation of the study's protocol, emphasizing the importance of honesty in their responses and asked to participate in the study. Each participant gave their written and oral informed consent. The study's protocol received approval from the council of the college of health science at the university of Duhok as well as ethics committee at the Duhok directorate of health with a reference number (25092024-8-27). A total of 160 subjects (80 Prediabetics and 80 age and sex matched healthy individuals) included in this study. A pretested questionnaire designed to gather data on age, gender, residence, and occupation, smoking habits and family history of diabetes, cardiovascular diseases (CVD), thyroid diseases and personal history of prediabetes. History of using medication, vitamins and mineral taken by participants were documented. Weight, height and waist circumference were recorded. Body mass index was calculated as dividing weight in kg by height in square meter. According to BMI participants were categorized as underweight (BMI less than 18.5), Normal (BMI between 18.5 and 24.99), Overweight (BMI more than 24.99) and obese (BMI more than 30). Inclusion criteria for prediabetic subjects included all individuals who met the criteria for prediabetes according to American Diabetes Association were included[2]. These includes HBA1c levels between 5.7% and 6.4%. Fasting Plasma Glucose level between 100 mg\dL and 125 mg\dL (impaired fasting glucose). Oral Glucose Tolerance Test: post two hour blood glucose level between 140 mg\dL and 199 mg\dL after consuming 75 grams of glucose solution (impaired glucose

tolerance). Exclusion criteria for prediabetic subjects includes Diabetes, cardiovascular, rheumatoid, renal, hepatic and thyroid diseases, any history of malignancy, pregnancy, Alcohol consumption and Individuals taking medications, vitamins and/or minerals six months prior to sampling. Inclusion criteria for healthy individuals includes: Apparently healthy subjects, no personal and family history of prediabetes and diabetes, no history of chronic diseases, non-alcoholic drinkers and not taking medications, vitamins and/or minerals six months prior to sampling. After an overnight fast 5ml of venous blood were obtained by venipuncture then separated into two portions. A volume of (3mL) was collected in a gel separated tube with no anti-coagulant and the remaining (2ml) collected into the EDTA tube with anticoagulant for HbA1c estimation using latex agglutination-inhibition immunoassay by Cobas 6000 Roche. For a two-hours, 75-gram oral glucose tolerance test (OGTT), a second blood sample was collected from each individual after consuming a 75-gram glucose solution. The Cobas 6000 Roche (open, automated, discrete and random access) clinical chemistry analyzer was used to measure serum levels of insulin using electrochemiluminescence technique and glucose using colorimetric method, while the remaining of the fasting tubes serum was carefully transferred to pre-labeled aliquot tubes and preserved at -80°C for later estimation of 8-hydroxy-2'-deoxyguanosine (8-OHdG) using ELISA technique. Homeostatic Model Assessment-Insulin Resistance (HOMA-IR) was calculated by using a specific formula for HOMA-IR estimation: HOMA-IR = Fasting glucose (mg\dL) X fasting insulin (uU\ml)/405. Participants were divided into three groups, No insulin resistance (HOMA-IR less than 3), moderate insulin resistance (HOMA-IR between 3 and 5), sever insulin resistance (HOMA-IR more than 5)[9].

Statistical analysis: The analysis of all the data was conducted using the Statistical Package for Social Science (SPSS 25, IBM: USA). The values of the laboratory parameters are presented as a mean along with the standard deviation (SD). Independent student t-test was used to assess differences in serum analyte among groups. Significance of association between various risk factors for categorical data was assessed by using Chi-square test. A cutoff point considered statistically significant was established at a P-value of ≤0.05.

RESULTS:

The Mean± SD of serum 8-OHdG level of prediabetes subjects was significantly higher when compared to and healthy individuals (p <0.01). The levels of HOMA-IR, FBS, and insulin were markedly elevated in individuals with prediabetes when compared to healthy subjects (p= <0.01) across all parameters. The level of HbA1c for prediabetes subjects was significantly higher in comparison to healthy individuals (p= 0.044). (Table 1).

Table 1 General characteristics of prediabetes subjects and healthy individuals

Parameter	Prediabetics mean ± SD	Healthy Individuals mean ±SD	P-value
Age (years)	46.3± 9.93	45.78± 9.39	0.73
BMI (Kg/m2)	31.97± 5.55	25.88± 5.64	<0.01
8-OHdG (ng/ml)	5.13±1.12	3.66±1.01	<0.01
HOMA-IR	5.04±3.69	2.69±1.52	<0.01
HbA1c%	6.48±0.48	5.04±0.48	0.044
FBS (mg/dL)	107.5±8.21	90.53±7.35	<0.01
Insulin(uU/mL)	18.98±13.68	12.07±6.89	<0.01

No significant difference was observed in Mean+SD of serum 8-OHdG levels when comparing male to female in prediabetes subjects (p=0.6588) and healthy individuals (p=0.78) (Table 2).

Table 2 Serum 8-OHdG levels stratified by gender

	Gender	n (%)	mean ±SD	P value
Prediabetics	Male	41(51.25)	5.07± 1.018	0.65
	Female	39(48.75)	5.18± 1.23	
Healthy Individuals	Male	41(51.25)	3.62±0.99	0. 78
	Female	39(48.75)	3.69±1.04	

The mean serum 8-OHdG levels was significantly higher in prediabetes subjects who are overweight and obese collectively as compared with those with normal weight (p=0.025). In healthy individuals, higher mean values of serum 8-OHdG level were observed in both overweight and obese groups when compared to those of normal weight; however, the difference was not statistically significant (p=0.24) (Table 3).

Table 3. Serum 8-OHdG levels stratified by body mass index

	BMI	n (%)	8-OHdG mean ±SD	P value
Prediabetics	Normal	9(11.25)	4.15± 1.09	0.025
	Overweight + Obese	71(88.75)	5.16± 1.12	
Healthy Individuals	Normal	45 (31.25)	3.66 ±1.01	0.24
	Overweight + Obese	35(68.75)	3.95±3.95	

The mean values of serum 8-OHdG level was higher in the centrally obese subjects when compared to normal waist circumference in both prediabetes subjects and healthy individuals and the difference was statistically significant in prediabetes subjects (p=0.05) but the difference in healthy individuals was near statistically significant value (p=0.09) (Table 4).

Table 4. Serum 8-OHdG levels stratified by waist circumference

	W.C.	n (%)	mean ±SD	P value
Prediabetics	Centrally obese	64(80)	5.25± 1.09	0.05
	Normal	16(20)	4.62± 1.12	
Healthy Individuals	Centrally obese	31(56.3)	3.88± 1.18	0.09
	Normal	49(43.8)	3.46± 0.91	

In subjects with insulin resistance (HOMA-IR >3.0), the mean values of serum 8-OHdG levels was higher when compared to insulin sensitive subjects (HOMA-IR <3.0), both in prediabetes subjects and healthy individuals. The difference was statistically significant among prediabetes subjects (p=0.03) but the difference in healthy individuals was not statistically significant (p=0.46) (Table 5).

Table 5. Serum 8-OHdG levels stratified by HOMA-IR

	HOMA-IR	n (%)	mean ±SD	P value
Prediabetics	>3.0	60(75)	5.92±1.61	0.03
	<3.0	20(25)	5.09± 1.31	
Healthy Individuals	>3.0	25 (31.25)	3.85±1.08	0.46
	<3.0	55(68.75)	3.57±0.98	

The mean values of serum 8-OHdG level was higher in the smokers when compared to non-smokers subjects but the difference did not reach the statistically significant cut off point in both prediabetes subjects (p=0.25) and healthy individuals (p=0.10) (Table 6).

Table 6. Serum 8-OHdG levels stratified by smoking habit

	Smoking	n (%)	mean ±SD	P value
Prediabetics	Smokers	23(28.75)	5.12± 0.93	0.25
	Non-Smokers	57(71.25)	4.87± 1.33	
Healthy Individuals	Smokers	30(22.5)	3.82 ± 1.46	0.10
	Non-Smokers	50(77.5)	3.31± 1.45	

DISCUSSION:

The striking finding of this study was that mean serum 8-OHdG levels were significantly elevated among prediabetic individuals compared to age and sex matched healthy individuals, suggesting increased oxidative stress and oxidative DNA damage which may contribute to the pathophysiology of insulin resistance and glucose dysregulation in individuals with prediabetes and eventual progression to type 2 diabetes mellitus. This study evaluated serum 8-hydroxy-2'-deoxyguanosine (8-OHdG) levels, which is one of a well-recognized marker of oxidative DNA damage [14]. Significantly higher HbA1c and fasting blood glucose as well as higher BMI, insulin levels, and HOMA-IR values were observed in prediabetic individuals when compared to age and sex matched healthy individuals. This is consistent with previous studies and reports which indicates that increased adiposity and insulin resistance have pivotal role that impairs plasma glucose metabolism and glucose tolerance [15]. The findings of the current investigation indicated that 8-OHdG levels were significantly elevated ($p < 0.01$) in individuals with prediabetes when compared with healthy individuals. Our findings confirm the hypothesis of increased oxidative stress and oxidative DNA damage in early glucose dysregulation of either insulin resistance or prediabetes [16, 17]. Mahmoud et al also found in their studies that oxidative DNA damage is increased in prediabetic and diabetic populations which aligned with our results [16]. No significant differences in 8-OHdG levels were detected in either group when analyzing both genders. Our findings are in accordance with those of Al-dulaimi et al who found no sex specific differences in oxidative stress markers among prediabetic individuals [18]. These finding suggests that gender may not influence oxidative DNA damage in prediabetic states. We found in our study that Prediabetic subjects with higher BMI had significantly elevated 8-OHdG levels, indicating a positive association between adiposity and oxidative stress. This is consistent with the others findings who demonstrated that obesity is one of the major contributors to systemic oxidative stress via increased adipocyte-derived reactive oxygen species [16]. In healthy individuals, while the trend was similar, it was not statistically significant, possibly due to lower levels of systemic inflammation in the absence of insulin resistance. In this study we found a significantly higher mean serum levels of 8-OHdG in centrally obese Prediabetics compared to those with normal waist circumference. This observation reinforces the role of excess visceral fat which is a potent source of ROS through increased mitochondrial activity, chronic inflammation, and adipokine imbalance resulting in oxidative stress and oxidative DNA damage as well as early glucose

metabolic abnormalities. We also found in this study that mean serum levels of 8-OHdG in centrally obese healthy individuals was higher compared to those with normal waist circumference but the difference here was near statistical significant cutoff point. The near-significant difference among healthy individuals suggests that central obesity might initiate oxidative damage even before biochemical markers of glucose dysregulation become apparent. Our results showed that mean serum levels of 8-OHdG in Prediabetics with insulin resistance was significantly higher compared to insulin-sensitive counterparts, which agrees with other study results which reported that hyperinsulinemia leads to an excess production of mitochondrial reactive oxygen species, resulting in oxidative DNA damage [19]. This may underscores the association between insulin resistance and oxidative stress. The absence of a significant difference in the healthy group may indicate that oxidative stress intensifies once a threshold of metabolic impairment is crossed. These findings suggest that oxidative DNA damage may not only coexist with insulin resistance but could contribute to its progression. Smokers in the present study had higher mean serum 8-OHdG levels in both groups, consistent with the known role of smoking in increasing oxidative stress through free radical formation[20,21]. However, the differences did not reach statistical significance, possibly due to relatively small sample size and/or underreporting of smoking habits.

CONCLUSION:

This study highlighted a significant rise in mean serum 8-OHdG levels among prediabetic individuals, indicating increased oxidative stress and DNA damage during early glucose dysregulation. The findings support a strong association between oxidative DNA damage marker and insulin resistance, particularly in individuals with higher BMI and central obesity. Screening for prediabetes with larger sample size especially for high risky group and strategies to reduce oxidative stress including a balanced diet rich in antioxidants as well as weight reduction and controlling comorbidities is recommended to prevent complications associated with dysglycemia.

Conflict of interest: The authors declare no conflict of interest.

Limitations: The relatively small sample size was the major limitation of the present study which may not enough explore the pathophysiology behind oxidative DNA damage and glucose dysregulation in prediabetic subjects. The study was limited by the absence of several markers for assessing DNA damage.

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پوختە

زیانی DNA یا ئوکسیداتی وچەند فاکتەرێن میتابولزمێ ل دە ف کەسێن پێش قوناغا ئێشا شەکرێ ل دهوکێ – هەرێما کوردستانا عیراق

پێشەکی و ئارمانج: قوناغا پێش نەخوشیا شەکرێ بارودوخەکێ دەستپێکی بو نەخوشیا شەکرێ ژ جورێ دووێ دهێتە هژمارتن ئو دبیتە ئەگەرێ تێکچونێ د مێتابولیزما گلوکوزی دا ژ ئەگەرێ بەرگریکرنا ئەنسولینێ و لاوازکرنا خانێن بێتایێ ل بنکریاسێ. ل ڤان دوماهیێان، گرنگی زێدەتر هاتە دان ب رولێ ژ ناڤچونا DNA ژ ئەگەرێ فشارا ئوکسانێ کو ئەگەرەکێ هاریکارە ژبو تێکچونا مێتابولیزمی، ب تایبەتی ب رێکا 8-هایدروکسی-2'- دیوکسی گوانوسین(8-OHdG) ، ئەو نیشاندەرەکێ ژیانی یێ دیارە بو فشارا ئوکسانێ.

ئارمانجا ئەڤێ ڤەکولینێ هەلسەنگاندنا ئاستێ 8-OHdG د پلازما ئەوان کەسێن کو توشبوویێن قوناغا پێش نەخوشیا شەکرێ نە و ڤەدیتنا پەیوەندیا وێ ب هندەك پیڤەرێن مێتابولیزمی ب بەراورد کرن دگەل کەسێن ساخلەم.

رێکێن کاری: ڤوکولین لسەر حالەتێ ١٦٠ بەشداربوویان هاتە کرن (٨٠ توشبوویێن قوناغا پێش نەخوشیا شەکرێ و ٨٠ کەسێن ساخلەم) ل تاقیگەها ناڤەندی یا پارێزگەها دهوکێ - عیراق. هەمی هەلسەنگاندنێن پزیشکی و بیوکیمیایی پێکهاتبوون ژ : پیڤەرێ بارسەیا لەشی(BMI) ، شەکرا خینێ یا روژیگری(FBS) ، هیموگلوبینا شەکرێ(HbA1c) ، ئەنسولین، بەرگریا ئەنسولینێ(HOMA-IR) ، و رێژەیا 8-OHdG د پلازمایێ دا. تەکنیکا ELISA ژبو پیڤانا-8 OHdGهاتیە بکارئینان. ب رێکا بەرنامێ SPSSڤێرژن ٢٥ ئەنجام هاتینە شیکارکرن، دگەل دەستنیشانکرنا ئاستێ ڕابەرێ ئامارێ لسەر p. ≤0.05

ئەنجام: د ئەنجامان دا دیاربوو رێژەیا 8-OHdG ب شێوەیەکێ بەرچاڤ یا بلند بوو لدەف توشبوویێن قوناغا پێش نەخوشیا شەکرێ (5.13±1.12 نگ/مل) بەراوردی دگەل کەسێن ساخلەم (3.66±1.02) نگ/مل،). (p<0.01هەروەسا زێدەبوونا واتایی دFBS ، HbA1c، ئەنسولین، و HOMA-IR لدەف گروپێ توشبوویێن قوناغا پێش نەخوشیا شەکرێ هاتە دیتن. چ جیاوازییێت واتایی نە هاتنە دیتن د ناڤبەرا رەگەزان دا د رێژەیێن 8-OHdG. رێژەیێن بلندتر ژ 8-OHdG هاتنە دیتن لدەف کەسێن خودان کێشا زێدە، قەلەویا ناڤەندی، بەرگریا ئەنسولینێ. هەمان تشت لدەف کەسێن ساخلەم ژی هاتە دیتن بەلێ بێی ڕابەرەکێ ئامارێ یێ دیارکری.

دەرئەنجام: ئەنجامێن ئەڤێ ڤەکولینێ پەیوەندیەکا بهێز دناڤبەرا ژناڤچوونا DNA ژ ئەگەرێ فشارا ئوکسانێ و ئاستێ پێش نەخوشیا شەکرێ دیارکر، ب تایبەتی د حالەتێن کێشا زێدە و قەلەویا ناڤەندی و بەرگریا ئەنسولینێ. ئەڤ ئەنجامە رولێ تەوەری یێ فشارا ئوکسانێ د تێکچوونا مێتابولیزما گلوکوزی یا پێشەکی دیاردکەت، کو ئاماژێ ب وێ چەندێ ددەت شیانێن ژناڤچوونا وێ دگەل ستراتیجیێن خوپاراستنا پێشەکی یە.

الخلاصة

تلف الحمض النووي التأكسدي وبعض عوامل الخطورة الأيضية في مرحلة ما قبل السكري في دهوك، إقليم كردستان العراق

الخلفية والأهداف: تعد مرحلة ما قبل السكري حالة تمهيدية لداء السكري من النوع الثاني، وتتميز باضطرابات في استقلاب الجلوكوز ناجمة عن مقاومة الإنسولين وضعف وظيفة خلايا بيتا في البنكرياس. في الآونة الأخيرة برز الاهتمام بدور تلف الحمض النووي الناتج عن الإجهاد التأكسدي كعامل مساهم في الخلل الأيضي، لاسيما من خلال 8-هيدروكسي-2′-ديوكسي غوانوزين (8-OHdG)، الذي يعتبر مؤشر حيوي معروف للإجهاد التأكسدي.

تهدف هذه الدراسة إلى تقييم مستويات 8-OHdG في مصل الدم لدى الأفراد المصابين بمرحلة ما قبل السكري، واستكشاف علاقته ببعض المعايير الأيضية مقارنة بالأفراد الأصحاء.

طرق البحث: تم إجراء دراسة حالة وشاهد شملت 160 مشاركًا (80 مصابا بمرحلة ما قبل السكري و80 شخصا سليما) في المختبر المركزي بمحافظة دهوك، العراق. شملت التقييمات السريرية والبيوكيميائية كلا من: مؤشر كتلة الجسم (BMI)، سكر الدم الصائم (FBS)، الهيموغلوبين السكري (HbA1c)، الإنسولين، مقاومة الإنسولين (HOMA-IR)، ومستوى 8-OHdG في مصل الدم. تم استخدام تقنية ELISA لقياس 8-OHdG، وتم تحليل البيانات باستخدام برنامج SPSS الإصدار 25، مع تحديد مستوى الدلالة الإحصائية عند $p \leq 0.05$.

النتائج: أظهرت النتائج أن مستويات 8-OHdG كانت أعلى بشكل ملحوظ لدى المصابين بمرحلة ما قبل السكري (5.13±1.12 نغ/مل) مقارنة بالأصحاء (3.66±1.02 نغ/مل، $p<0.01$). كما وجدت زيادات ملحوظة في FBS، HbA1c، الإنسولين، وHOMA-IR لدى مجموعة مرحلة ما قبل السكري. لم تسجل فروق معنوية بين الجنسين في مستويات 8-OHdG. لوحظت مستويات أعلى من 8-OHdG لدى الأفراد الذين يعانون من زيادة الوزن، السمنة المركزية، ومقاومة الإنسولين. كما أظهر الأفراد الأصحاء اتجاها مشابها، لكن دون دلالة إحصائية واضحة.

الاستنتاج: تشير نتائج هذه الدراسة إلى وجود علاقة قوية بين تلف الحمض النووي الناتج عن الإجهاد التأكسدي ومرحلة ما قبل السكري، لاسيما في حالات زيادة الوزن والسمنة المركزية ومقاومة الإنسولين. تسلط هذه النتائج الضوء على الدور المحوري للإجهاد التأكسدي في اضطرابات استقلاب الجلوكوز المبكرة، مما يشير إلى إمكانية استهدافه ضمن استراتيجيات الوقاية المبكرة