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Research Article

Comparison between the Effects of Hypertension on Diabetic Mellitus with Endothelin Function

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Abstract

Background: Diabetes mellitus and hypertension are among the most prevalent chronic diseases worldwide; both strongly associated with vascular and metabolic complications. Endothelin-1 is a potent vasoconstrictor that plays a key role in vascular tone regulation and endothelial function.

Objective: To evaluate serum Endothelin-1 levels, lipid profile parameters (total cholesterol, LDL-C, VLDL-C, HDL-C, triglycerides), and HbA1c in patients with type 2 diabetes and essential hypertension compared with healthy controls, in order to assess their association with vascular injury and metabolic risk.

Methods: A case-control study was conducted in the Department of Biochemistry, College of Medicine, University of Baghdad. Between November 2024 and February 2025, a sample of patients were recruited from Baghdad Teaching Hospital and Ibn Al-Bitar Center for Cardiac Surgery Hospital. A study on 180 adult males (aged 40–50 years), divided into three groups: 60 with type 2 diabetes mellitus (Group A), 60 with essential hypertension (Group B), and 60 healthy controls (Group C). Blood samples were collected after an overnight fast for measurement of HbA1c, lipid profile, and Endothelin-1 using a Sandwich-ELISA kit.

Results: Significant differences were observed among the three groups. Group A showed markedly higher HbA1c, triglycerides, and VLDL-C, along with significantly lower HDL-C compared with Groups B and C. Both Groups A and B had significantly elevated total cholesterol and LDL-C compared with controls. Endothelin-1 levels were significantly higher in diabetics than in hypertensive patients and controls.

Conclusion: Both diabetes and hypertension impair metabolic health, with diabetes showing more severe effects on lipid metabolism and glycemic control. Elevated endothelin-1 levels in diabetics may contribute to increased vascular risk, highlighting the need for early metabolic monitoring and intervention.

Keywords: Diabetes mellitus; Endothelin-1; Hypertension; Lipid profile; Vvascular complications.

Introduction

Diabetes mellitus (DM) is defined by elevated blood glucose levels and is regarded as a public health issue since it causes long-term metabolic disorders determined by behavioral, environmental, and hereditary variables (1). In just 34 years, the number of DM patients has quadrupled globally (from 108 million in 1980 to 422 million in 2014), while the global incidence of diabetes in individuals over 18 has increased from 4.7% to 8.5%. (2). Arterial hypertension (AH) is a long-term illness that is defined by a blood pressure (BP) level where the advantages of treatment exceed the hazards (3). Systolic blood pressure (SBP) ≥ 140 mm Hg and/or diastolic blood pressure (DBP) ≥ 90 mm Hg are considered indicators of AH, under the European Society of Cardiology / European Society of Hypertension (ESC/ESH) recommendations. Globally, AH is a major contributor to cardiovascular disease (CVD) and mortality (4). In addition, it causes numerous organs to malfunction, such as the kidneys, heart, blood vessels (5), and eyes, as well as a higher chance of brain injury (6). A characteristic of all forms of DM is elevated blood glucose. Many of the harmful cellular effects of diabetes mellitus, particularly in endothelial cells, are thought to be mediated by elevated levels of circulating glucose (7). DM is known to cause decreased vasodilation in response to acetylcholine. This effect is thought to be caused by endothelial nitric oxide synthase's decreased nitric oxide production and activity (8). In contrast to arterial smooth muscle cells, macrovascular endothelial cells might be especially sensitive to the extracellular glucose environment. The extent to which endothelial cells are directly impacted by acute blood glucose elevation in vivo and whether this effect is mainly mediated by secondary mechanisms are unknown (9). Endothelin-1 (ET-1) is a potent mitogen and vasoconstrictor. It has a strong vasoconstrictor effect on the vasculature and is important for regulating the vascular tone throughout the body. The human body uses ET-1 for a number of purposes, including immune cell recruitment during

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inflammation and ion transport in the gastrointestinal tract (10).

This study aimed to compare how DM and hypertension (HTN) affect blood vessels.

Patients and Methods

A case-control study was conducted in the Department of Biochemistry, College of Medicine, University of Baghdad. Between November 2024 and February 2025, a sample of patients was recruited from Baghdad Teaching Hospital and Ibn Al-Bitar Center for Cardiac Surgery Hospital. The study protocol was approved by the Department of Clinical Biochemistry Research Ethics Committee.

A total of 180 males were grouped as follows: Group I included 60 males with DM, Group II included 60 males with essential HTN not caused by another medical condition and with no diabetes, and Group III included 60 healthy individuals as a control group. In order to identify any systemic conditions that could affect the evaluated biochemical parameters, such as hepatic or renal disorders, participants were evaluated through a thorough medical history review and a structured questionnaire. Those with a documented history of cardiovascular diseases, particularly congenital heart abnormalities, were excluded. To further reduce the possibility of confounding variables, participants who smoked or showed signs of alcohol or drug addiction were not enrolled in the study.

Following an overnight fast, five milliliters (5ml) of venous blood were drawn from each participant, transferred onto a gel tube and left to clot at room temperature, the sample was then centrifuged for 10 minutes at 896 RCF (xg). The extracted serum was moved to Eppendorf safe-lock tubes and stored at -20°C for further analysis. Endothelin-1 was determined using the Sandwich-ELISA kit.

Statistical analysis: Version 25 of the Statistical Package for Social Sciences (SPSS) was used to analyze the data. Categorical variables were displayed as percentages, medians with interquartile ranges, and continuous data as means ± standard deviations. For continuous variables, the analysis employed the independent t-test and the ANOVA test; while for categorical variables, the Chi-square test ($\chi 2$ test) was employed. P-values below 0.05 were regarded as significant.

Results

Group A showed the highest mean total cholesterol (189.7 ± 43.96 mg/dl) followed by Group B (183.3 ± 25.40 mg/dl), both significantly higher than the control group (150.7 ± 21.68 mg/dl) ($p < 0.001$). Triglycerides were also markedly elevated in Group A (201.2 ± 97.16 mg/dl) compared to Group B (147.2 ± 48.38 mg/dl) and controls (99.2 ± 20.76 mg/dl) ($p < 0.001$). HDL-C was lower in Group A (43.0 ± 10.72 mg/dl) than Group B (47.8 ± 7.31 mg/dl) and the control group (48.3 ± 7.14 mg/dl) ($p < 0.001$). Furthermore, LDL-C was higher in Groups A and B (103.3 ± 34.05 and 102.3 ± 27.63 mg/dl) versus controls (82.7 ± 25.64 mg/dl) ($p = 0.012$). VLDL-C also showed higher values in Group A (40.0 ± 9.15 mg/dl) and Group B (30.2 ± 10.88 mg/dl) compared to controls (19.8 ± 4.14 mg/dl) ($p = 0.008$), Table 1.

Table 1: Comparison between study groups' mean biochemical parameter levels

Biochemical parameters	Study groups			*P* - Value*
	Group A Mean ± SD	Group B Mean ± SD	Control Group Mean ± SD	
Cholesterol (mg/dl)	189.7 ± 43.9	183.3 ± 25.4	150.7 ± 21.6	< 0.001
Triglyceride (mg/dl)	201.2 ± 97.1	147.2 ± 48.38	99.2 ± 20.76	< 0.001
HDL-C (mg/dl)	43.0 ± 10.72	47.8 ± 7.31	48.3 ± 7.14	< 0.001
LDL-C (mg/dl)	103.3 ± 34.05	102.3 ± 27.63	82.7 ± 25.64	0.012
VLDL-C (mg/dl)	40.0 ± 9.15	30.2 ± 10.88	19.8 ± 4.14	0.008

*** ANOVA-test at 0.05 level: Significant difference between more than two independent means**

Post hoc tests (LSD) were run to confirm the differences in the mean level of biochemical parameters between the three groups. Group A had significantly higher mean levels of triglycerides, and very low-density lipoprotein (VLDL-C) than groups B and C. At the same time, group A had significantly lower mean levels of high-density lipoprotein (HDL-C) than the other two groups. No significant difference was detected between group A and group B regarding cholesterol and low-density lipoprotein (LDL-C) concentrations, Table 2.

Table 2: Multiple comparisons of biochemical parameters between the study groups using Post hoc (LSD) test

Biochemical	Study groups		Mean diff.	*P*- value
Cholesterol (mg/dl)	Group A	Group B	9.458	0.234
	Group A	Group C	39.03	< 0.001
	Group B	Group C	29.58	0.002
Triglyceride (mg/dl)	Group A	Group B	53.93	0.001
	Group A	Group C	98.09	< 0.001
	Group B	Group C	48.05	0.013
	Group A	Group B	- 4.841	0.020
HDL-C (mg/dl)	Group A	Group C	- 5.295	0.011

	Group B	Group C	- 0.453	0.848

Table 2:

Biochemical	Study groups	Mean diff.	*P*- value	Biochemical
LDL-C (mg/dl)	Group A	Group B	0.941	0.891
	Group A	Group C	20.65	0.003
	Group B	Group C	19.71	0.014
VLDL-C (mg/dl)	Group A	Group B	9.801	0.004
	Group A	Group C	20.21	< 0.001
	Group B	Group C	10.41	0.007

***Multiple comparisons of biochemical parameters between the three groups using Post hoc (LSD) test**

Table 3 shows that the levels of serum ET-1 were significantly higher in Group A (51.1 ± 1.96 pg/ml) compared to Group B (30.9 ± 1.75 pg/ml) and the control group (15.7 ± 0.97 pg/ml), with a highly significant difference among the groups ($P < 0.001$).

Table 3: Comparison of the study groups endothelin concentrations

	Study groups			
Variable	Group A Mean ± SD	Group B Mean ± SD	Control Group Mean ± SD	P - Value
Endothelia (pg/ml)	51.1 ± 1.96	30.9 ± 1.75	15.7 ± 0.97	< 0.001

*** Significant difference at the 0.05 level between more than two independent means as determined by the ANOVA test**

To verify the variations in the mean endothelin level among the groups under study, post hoc tests (LSD) were performed. ET-1 concentrations in group a patients were significantly higher than those in groups B and C (51.1 pg/ml vs. 30.9 pg/ml and 15.7 pg/ml, $P < 0.001$). However, group B had a substantially higher mean ET-1 concentration than group C (30.9 pg/ml vs. 15.7 pg/ml, $P < 0.001$), Table 4.

Table 4: Post hoc tests (LSD) to validate the variations in the study groups' mean endothelin-1 concentrations

	Study Groups			
	Group A Mean ± SD	Group B Mean ± SD	Control Group Mean ± SD	*P*- Value
Endothelin-1 (pg/ml)	51.1 ± 1.96	30.9 ± 1.75		< 0.001
	51.1 ± 1.96		15.7 ± 0.97	< 0.001
		30.9 ± 1.75	15.7 ± 0.97	< 0.001

*** Significant difference of means using Post hoc test (LSD) at 0.05 levels**

Discussion

In the current study, females were not included in the study groups because of the influence of hormonal fluctuations particularly related to the menstrual cycle, menopause, and hormone replacement therapy - on the metabolic parameters and cardiovascular functions. These hormonal variations can affect insulin sensitivity, lipid metabolism, endothelial function, and blood pressure regulation, thereby, introducing additional biological variability into the data (11).

Low-density lipoprotein cholesterol is without a doubt the most important lipoprotein in the spectrum. Numerous studies have demonstrated that the only variables linked to DM in high-risk populations are plasma triglyceride levels. A high-fat diet lowers relative HDL-C, speeds up the onset of DM, and increases the liver production of TG (12).

The study findings indicate that both Group A and Group B exhibited significantly higher cholesterol levels compared with the control group, suggesting that dyslipidemia is a shared metabolic disturbance in DM and HTN. However, Group A presented a more adverse lipid profile, characterized by markedly elevated TGs and significantly reduced HDL-C levels. These alterations are consistent with the metabolic impact of insulin resistance, which commonly elevates TGs and contributes to HDL-C reduction. Given that high TGs and low HDL-C are established risk factors for cardiovascular disease, the lipid abnormalities observed particularly in diabetic patients highlight an increased cardiovascular risk and underscore the importance of early lipid monitoring and targeted intervention in these populations. In comparison to the control group, Groups A and B had significantly higher levels of LDL-C and VLDL-C. The lack of a significant difference in LDL-C levels between Groups A and B, however, indicates that elevated LDL-C, a key atherogenic component, is a result of both DM and HTN these results are in agreement with Bani Salameh, *et al.* (13) and Li, *et al.* (14).

Hypertension causes dyslipidemia through a number of indirect pathways, even though it does not directly alter lipid metabolism the way DM does. Prolonged HTN affects the vascular endothelium, which lowers the availability of nitric oxide (NO) and encourages oxidative stress and inflammation. These pathological alterations enhance the oxidation of circulating lipids and disrupt the metabolism of lipoproteins. Furthermore, oxidative stress and inflammation are made worse by the activation of the Renin-Angiotensin-Aldosterone System (RAAS), which is frequently seen in HTN. This leads to increased LDL oxidation and reduced lipid clearance (9).

Insulin resistance, which is common in people with type 2 DM and HTN with metabolic syndrome, results in decreased hepatic absorption of LDL, which causes LDL-C to build up in the plasma. Damage to the endothelium brought on by high blood pressure also increases vascular permeability, which permits more LDL particles, especially tiny, dense LDL, to enter the sub-endothelial region, where they are more vulnerable to oxidation (15). This process promotes atherosclerosis and has been documented in prior studies, including those by Salameh, *et al.* (13) and Li, *et al.* (14), whose findings support the current study findings regarding lipid abnormalities in both diabetic and hypertensive groups.

Chronic high blood pressure damages the endothelium, impairing nitric oxide production and promoting inflammation and oxidative stress, which contributes to altered lipoprotein metabolism and lipid oxidation. HTN frequently causes activation of the RAAS, which has been connected to elevated oxidative stress and inflammation, which may impair lipid clearance and increase LDL oxidation. Many hypertensive patients exhibit insulin resistance, particularly those with metabolic syndrome. This insulin resistance may contribute to the same dyslipidemic profile observed in type 2 DM, including elevated TGs, low HDL, and a small dense LDL (16).

Significant variations in biochemical markers among the three experimental groups are shown by the LSD post hoc analysis results. In comparison to Groups B and C, Group A showed significantly greater levels of TGs, VLDL-C, and HbA1c. Given that poor glycemic control and lipid abnormalities are characteristics of metabolic syndrome and type 2 DM, these findings indicate a higher degree of metabolic dysregulation in Group A (17).

Endothelin-1, a potent vasoconstrictor implicated in vascular damage, showed marked elevation in diabetic patients as compared to hypertensive patients and the control group in the current study. This indicates a strong association between increased ET-1 levels and DM, with or without an additional pronounced elevation, in hypertensive patients. Because diabetics have a much higher concentration of ET-1, it is possible that hyperglycemia contributes to endothelial dysfunction, which is a major contributing factor to the development of vascular complications. This concurs with the study by Kostov (18) and Mayyas, *et al.* (19). Elevated levels of ET-1 have been observed in conditions such as HTN, atherosclerosis, and chronic inflammatory diseases, reflecting its involvement in vascular tone regulation and pro-inflammatory responses (20,21).

The significant increase in endothelin levels in Group A could be indicative of a more severe or advanced pathological state, possibly associated with a higher degree of vascular injury or endothelial stress. In contrast, the moderate increase in Group B suggests a less severe endothelial disturbance, whereas, the control group maintains baseline ET-1 levels, consistent with normal endothelial function.

Limitations

1. Single-Center Design: Participants were recruited from two hospitals in Baghdad, which can restrict the generalization of the results to different populations or geographical areas.

2. Exclusion of Lifestyle Factors: Smoking, alcohol use, and drug addiction were not addressed, as well as dietary patterns, physical activity levels, and socioeconomic status, which may impact lipid profiles and glycemic control.

3. Lack of Longitudinal Data: The absence of follow-up limits insights into how the measured parameters evolve or how effective interventions might alter vascular outcomes.

Conclusion

Both diabetes and hypertension impair metabolic health, with diabetes showing more severe effects on lipid metabolism and glycemic control. Elevated endothelin-1 levels in diabetics may contribute to increased vascular risk, highlighting the need for early metabolic monitoring and intervention.

Authors' declaration

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's Approval-Ethical Clearance: The project was approved by the local ethical committee of (College of Medicine, University of Baghdad) according to the code number (353A) on (28/ 11/ 2024).

Conflict of Interest: None

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Data availability: Upon reasonable request, the corresponding author will make the data sets generated and/or analyzed during the current work available.

Author contributions:

Study conception & design: (Haider K. Al Haddad, Shifaa J. Ibrahim, Ameen Abdulhasan Al-Alwany). Literature search: (Haider K. Al Haddad, Shifaa J. Ibrahim, Ameen A. Al Al-Wany). Data acquisition: (Haider K. Al Haddad, Shifaa J. Ibrahim, Ameen A. Al Al-Wany). Data analysis & interpretation: (Haider K. Al Haddad, Shifaa J. Ibrahim, Ameen A. Al Al-Wany). Manuscript preparation: (Haider K. Al Haddad, Shifaa

J. Ibrahim, Ameen A. Al Al-Wany). Manuscript editing & review: (Haider K. Al Haddad, Shifaa J. Ibrahim, Ameen A. Al Al-Wany).

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مقارنة بين آثار ارتفاع ضغط الدم ومرض السكري على مستوى الإندوثيلين-1

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الخلاصة:

خلفية البحث: يعد كل من داء السكري وارتفاع ضغط الدم من اكثر الامراض المزمنة شيوعا عالميا، ويرتبطان ارتباطا وثيقا بالمضاعفات الوعائية والتمثيل الغذائي. يعد الإندوثيلين-1 مضيق وعائي قوي ويلعب دورا أساسيا في تنظيم إنقباض الأوعية الدموية ووظيفة البطانة الوعائية.

المنهجية: أجريت دراسة حالة–شاهد في قسم الكيمياء الحياتية / كلية الطب / جامعة بغداد. وخلال الفترة من تشرين الثاني 2024 إلى شباط 2025، تم جمع العينات من المرضى من مستشفى بغداد التعليمي ومركز ابن البيطار لجراحة القلب. أجريت الدراسة على 180 من الذكور البالغين الذين تتراوح أعمارهم بين 40 و50 سنة، تم تقسيمهم إلى ثلاث مجموعات: 60 مريضا بالسكري من النوع الثاني المجموعة (A)، 60 مريضا بارتفاع ضغط الدم الأساسي بدون السكري المجموعة (B)، و60 فردا غير مريض كمجموعة ضابطة المجموعة (C). تم جمع عينات الدم بعد صيام خلال الليل وتحليلها لتحديد مستوى الدهون. تم استخدام مجموعة Sandwich-ELISA لتحديد مستوى الاندوثيلين.

النتائج: لوحظت فروق معنوية واضحة بين المجموعات الثلاث. أظهرت المجموعة A ارتفاعا ملحوظا في مستوى الهيموغلوبين السكري التراكمي، والدهون الثلاثية، والبروتين الدهني منخفض الكثافة جدا، مع انخفاض معنوي في البروتين الدهني عالي الكثافة مقارنة بالمجموعتين B وC. كما سجلت كل من المجموعتين A وB مستويات أعلى من الكوليسترول الكلي و البروتين الدهني منخفض الكثافة مقارنة بالمجموعة الضابطة. بالإضافة إلى ذلك، كان مستوى الإندوثيلين-1 أعلى بشكل معنوي لدى مرضى السكري مقارنة بمرضى ارتفاع الضغط والأصحاء.

الاستنتاجات: يؤثر كل من داء السكري وارتفاع ضغط الدم سلبا على صحة التمثيل الغذائي، مع تأثيرات أشد لداء السكري على استقلاب الدهون ومستوى السكر في الدم. وقد يسهم ارتفاع مستويات الإندوثيلين-1 لدى مرضى السكري في زيادة خطر الإصابة بأمراض الأوعية الدموية، مما يبرز الحاجة إلى المراقبة والتدخل المبكرين في مجال التمثيل الغذائي.

مفتاح الكلمات: السكري، الإندوثيلين-1، ارتفاع ضغط الدم، ملف الدهون، السكر التراكمي، مضاعفات وعائية.