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# Relationship between inflammatory biomarkers and insulin resistance in Iraqi hypertensive patients

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*Abstract:* **Background: Hypertension is a global health issue characterised by elevated blood pressure levels, leading to an increased risk of cardiovascular morbidity and mortality. Insulin resistance is the inability of the liver, adipose tissue, and skeletal muscle to respond to insulin. Several prior studies have reported a correlation between impaired insulin sensitivity and elevated inflammatory markers. These conditions are believed to be associated with increased risk of incident hypertension by promoting endothelial dysfunction and inflammatory processes.**

**Objective: The current study aimed to investigate the association between insulin resistance (IR) and multiple inflammatory biomarkers in hypertensive patients.**

**Materials and methods: In this case-control study, 150 volunteers with normotension and 150 hypertensive patients participated. The enzyme-linked immunosorbent assay (ELISA) was used to measure serum levels of insulin, tumour necrosis factor-alpha (TNF), high-sensitivity C-reactive protein (hs-CRP), and interleukin–6 (IL-6). Body mass index (BMI) and homeostasis model assessment of insulin resistance (HOMA-IR) were computed. To ascertain the relationship between insulin levels and HOMA-IR with the pro-inflammatory indicators, logistic regression analysis was employed.**

**Results: Hypertensive individuals had significantly higher P values ($P < 0.05$) and higher serum levels of hs-CRP, IL-6, and TNF-α compared with normotensive volunteers. Elevated insulin levels and HOMA-IR index were also markedly observed ($P < 0.001$). Furthermore, positive correlations were found between insulin levels and the HOMA-IR index and inflammatory biomarkers ($P < 0.05$), but no association was found between IL-6 levels and the HOMA-IR index ($P = 0.119$).**

**Conclusion: Hypertensive patients have significantly elevated levels of hs-CRP, TNF-a, and IL-6, which may be crucial in the pathophysiology and development of hypertension. These levels are also markedly correlated with high levels of insulin and HOMA-IR index among hypertensive patients.**

*Keywords:* **Insulin, hs-CRP, TNF-a; IL-6; hypertension, Iraqi population.**

## 1. INTRODUCTION

Hypertension, a prevalent condition characterized by elevated blood pressure levels, represents a significant public health challenge affecting millions globally [(1,2)]. Among its various manifestations, high levels of systolic blood pressure (SBP) and diastolic blood pressure (DBP) pose severe risks for cardiovascular morbidity and mortality [(3)].

The interplay of several biological mechanisms contributes significantly to elevated systolic blood pressure (SBP) and diastolic blood pressure (DBP) in hypertensive patients. One of them is that inflammation plays a crucial role in hypertensive cardiac remodelling, as elevated levels of plasma inflammatory markers, including high-sensitivity C-reactive protein (hs-CRP), tumor necrosis factor-alpha (TNF), and interleukin–6 (IL-6), were associated with a risk of incident hypertension [(4-6)]. It has been reported that pro-inflammatory markers not only cause damage to the cardiac tissue but also increase vascular stiffness [(5)].

The intricate relationship between inflammation and the pathology associated with essential hypertension, including insulin resistance, remains incompletely understood. However, there is an increasing amount of data to suggest that inflammation is an important connecting factor between metabolic syndrome and hypertension, which are well-known contributors to cardiovascular disease [7].

Hypertension is often linked to insulin resistance, a condition in which the body's inability to respond to insulin leads to metabolic disturbances such as elevated glucose levels and dyslipidemia [8-9]. These metabolic alterations can exacerbate hypertension by promoting endothelial dysfunction and inflammatory processes [8,10]. For instance, some studies emphasise a correlation between elevated inflammatory markers and reduced insulin sensitivity, suggesting that low-grade inflammation plays a role in these conditions [11]. Additionally, sympathetic nervous system activity may be enhanced by insulin resistance, leading to elevated blood pressure [12]. Particularly, the Iraqi hypertensive patients have a higher risk of insulin resistance development due to factors including genetic factors, poor dietary habits, and obesity. Therefore, the study aimed to determine the relationship between insulin resistance (IR) and multiple inflammatory biomarkers in hypertensive patients.

## 2. SUBJECTS AND METHODS

### 2.1. Study Ethics

The Medical Research and Ethnics Committee at the Biochemistry Department, College of Medicine, Kufa University, approved the study protocol. Subsequently, written informed consent has been obtained from each subject prior to enrolment.

### 2.2. Study Population

The current case-control study was performed on 300 individuals at the Al-Sader Teaching Hospital in Najaf, Iraq, from September 2025 to February 2026. The Patient selection was performed with inclusion and exclusion criteria. The study's practical work was performed at the Department of Clinical Biochemistry, College of Medicine, Kufa University. A standard questionnaire was prepared to collect data on gender and age, height and weight, disease duration, medications used, current smoking status, and family history.

### 2.3. Inclusion and exclusion criteria

A total of 150 hypertensive patients (≥ 18 years) of both genders volunteered to participate in this study and were selected as recommended by the American Heart Association using a mercury sphygmomanometer. Participants with acute or chronic inflammatory disorders, diabetes, or pregnancy were not allowed to participate in the study. Similar to the enrolled patients, a control group of 150 normotensive individuals was also enrolled. They were chosen at random from the general population of employees and their family members, taking into account factors such as age, weight, and gender. Additionally, no overt systemic illness was present in any of these people.

### 2.4. Data collection

At the time of data collection, the participants' blood pressure was measured. This was done to identify hypertensive patients who met the study's criteria (systolic blood pressure>140 mmHg or diastolic blood pressure>90 mmHg). The subject's right arm was measured twice at 10-minute intervals, and the participant's blood pressure was determined for the study by averaging the two values. The body mass index (BMI) was calculated after the subjects' height and weight were recorded. The homeostasis model assessment of insulin resistance (HOMA-IR) equation was used to measure insulin resistance [13]. To prevent blood clotting, 5 ml of fasting blood from each participant was collected and placed into ethylenediaminetetraacetic acid (EDTA) tubes. After that, serum was kept at -20 C for biochemical analyses. The glucose oxidase method was used to measure fasting blood glucose levels. Using an enzyme-linked immunosorbent assay (ELISA) kit (Elabscience, China) according to the manufacturer's instructions, the levels of fasting serum insulin, IL-6, TNF-α, and hs-CRP were measured.

### 2.5. Statistical Analysis

For continuous variables, values are shown as mean ± SD; for categorical variables, values are shown as numbers (%). The student's t-test for continuous variables was used to compare clinical and demographic parameters across groups. Using Pearson's correlation, the relationship between specific inflammatory biomarkers and insulin resistance was evaluated. Version 17.0 of SPSS software (SPSS Inc., Chicago, IL, USA) was used for all statistical analyses, and P≤ 0.05 was considered a significant value.

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## 3. RESULTS

Three hundred subjects participated in this study: one hundred fifty subjects were hypertensive patients, of which 48.66% were females, and one hundred fifty subjects were non-hypertensive subjects (56% males and 44% females). Demographic characteristics of the study groups are shown in Table 1. Our findings indicated that systolic and diastolic blood pressures in hypertensive patients were higher than in non-hypertensive subjects, with a significant p-value (< 0.001). However, there was no statistically significant difference in age, gender, or BMI between the two groups (p> 0.05).

As shown in Table 2, fasting insulin levels and the HOMA-IR index were significantly higher in hypertensive patients than in non-hypertensive subjects (P < 0.001). Whereas no significant difference was observed among study groups in FBS (p = 0.069).

Importantly, the data showed that inflammatory biomarker levels of hs-CRP, IL-6, and TNF-α differed significantly between hypertensive and non-hypertensive controls. They were significantly increased in patients with hypertension, with a p-value (< 0.001) as summarised in Table 3.

**Table 1: The Characteristics of the Study Population**

| | Population | | |
|---|---|---|---|
| **Variables** | Hypertensive (n=150) | Non-hypertensive (n=150) | **P-value** |
| **Age (years)** | 48.21 ± 7.32 | 46.40 ± 11.50 | 0.106 |
| **Gender:** | | | |
| **Male** | 77 (51.34%) | 84 (56%) | 0.418 |
| **Female** | 73 (48.66%) | 66 (44%) | 0.114 |
| **BMI (Kg/m$^2$)** | 28.21 ± 2.72 | 27.71 ± 2.78 | < 0.001* |
| **SBP (mm Hg)** | 152.3 ± 5.88 | 122.5 ± 10.35 | < 0.001* |
| **DBP (mm Hg)** | 96.7 ± 3.75 | 75.80 ± 3.45 | |

*P ≤ 0.05 was considered a significant value. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.

**Table 2: The metabolic characteristics of the study population**

| | Population | | |
|---|---|---|---|
| **Variables** | Hypertensive (n=150) | Non-hypertensive (n=150) | **P-value** |
| **FBS (mmol/L)** | 5.30 ± 0.48 | 5.19 ± 0.53 | 0.069 |
| **Insulin (µIU/ml)** | 17.06 ± 6.28 | 11.29 ± 5.50 | < 0.001* |
| **HOMA-IR** | 4.01 ± 1.50 | 2.63 ± 1.36 | < 0.001* |

*P ≤ 0.05 was considered a significant value. FBS, fasting blood sugar.

**Table 3: Levels of inflammatory biomarkers in hypertensive and non-hypertensive subjects**

| | Population | | |
|---|---|---|---|
| **Variables** | Hypertensive (n=150) | Non-Hypertensive (n=150) | **P-value** |
| **hs-CRP (mg/l)** | 9.17 ± 3.47 | 5.88 ± 2.84 | < 0.001* |
| **IL-6 (pg/ml)** | 7.38 ± 2.79 | 5.81 ± 2.93 | < 0.001* |
| **TNF-α (pg/ml)** | 8.44 ± 2.73 | 6.26 ± 1.74 | < 0.001* |

*P ≤ 0.05 was considered a significant value. IL-6: interleukin-6; TNF: tumour necrosis factor; hs-CRP: high-sensitivity C-reactive protein.

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Logistic regression analysis was used to evaluate the association between serum insulin levels and HMOA-IR and the different inflammatory biomarkers among hypertensive patients, as tabulated in Table 4. Among patients with hypertension, the findings showed that hs-CRP and TNF-α levels were significantly and positively correlated with serum insulin levels and the HOMA-IR index. The correlation coefficients of hs-CRP = 0.138, and TNF-α = 0.453 with insulin, and HOMA-IR with hs-CRP = 0.148 and TNF- α= 0.453. The p-value being < 0.05. Additionally, a positive correlation was observed between IL-6 and insulin levels in hypertensive patients (P < 0.05). On the other hand, there was no significant positive association of IL-6 levels with the HOMA-IR index (P = 0.119).

**Table 4: Correlation coefficients of insulin and HOMA-IR with inflammatory biomarkers in hypertensive patients**

| Biomarkers | Insulin | | HOMA-IR | |
|---|---|---|---|---|
| | r | P-value | r | P- value |
| **hs-CRP (mg/l)** | 0.138 | < 0.05 | 0.148 | < 0.05 |
| **IL-6 (Pm/ml)** | 0.242 | < 0.05 | 0.128 | 0.119 |
| **TNF-α (pg/ml)** | 0.453 | < 0.05 | 0.386 | < 0.05 |

r, Pearson's correlation coefficient; IL-6: interleukin-6; TNF: tumour necrosis factor; hs-CRP: high sensitive C- reactive protein.

## 4. DISCUSSION

The data from this study showed that fasting serum insulin levels and the HOMA-IR index were significantly elevated among hypertensive patients, as tabulated in Table 2. Our findings are in line with other studies that have proven a strong association between insulin resistance and the incidence of hypertension [7, 14, 15].

Insulin, a key vasodilator in skeletal muscle, enhances blood flow and glucose transport, but is impaired in insulin-resistant states [16]. It relaxes precapillary arterioles, facilitating transcapillary insulin transport and enhancing microvascular recruitment through nitric oxide (NO) production [17]. Insulin-induced NO production is mediated by the stimulation of nitric oxide synthase via the pathway of phosphatidylinositol 3-kinase (PI3K), like other metabolic insulin signaling [18]. Moreover, insulin also stimulates vasoconstriction through the production of the vasoconstrictor endothelin-1 (ET-1), dependent on mitogen-activated protein kinase (MAPK) [18].

Insulin resistance, a key component of metabolic syndrome, which includes hypertension, obesity, and dyslipidemia, is caused by downregulation of insulin signalling, primarily mediated by insulin receptor substrates in effector organs [19]. Insulin resistance leads to compensatory hyperinsulinemia, activating the Na+/H+ exchanger, Na+/K+ ATPase, Na-K-2Cl cotransporter, sodium-bicarbonate cotransporter (NBCe1), and the epithelial sodium channel, thereby increasing sodium retention in the kidneys and resulting in hypertension [7].

Our study found that hypertensive patients had significantly higher serum hs-CRP, IL-6, and TNF-α levels than normotensive subjects, as shown in Table 3.3. These results are consistent with data from former studies in hypertensive patients. Several studies have found the highest levels of hs-CRP in hypertensive patients across different populations [5, 20-22]. A study reported that levels of serum hs-CRP were associated with an increased risk of incident hypertension in Hong Kong [23]. Moreover, Prior studies have shown that hypertensive patients had higher IL-6 and TNF-α levels than normotensive subjects [15, 22-25]. In addition, a meta-analysis of cohort studies reported that high levels of circulating inflammation markers were found to be significantly associated with the risk of hypertension [21].

Studying the relationship between cytokines and hypertension is important because inflammatory mediators play a crucial role in the pathophysiological processes underlying high blood pressure. It has been shown that cytokines, including IL-6, TNF-α, and hs-CRP, cause vascular inflammation, which correlates with the severity of hypertension. For example, obesity-associated inflammation increases cytokine release, which in turn promotes insulin resistance, a risk factor for hypertension. Studies have elucidated that the chronic low-grade inflammatory environment propagates endothelial dysfunction, a precursor to hypertension [5,11].

Inflammation causes fibrosis and vascular remodelling, which may lead to hypertension through vascular wall thickening, vessel contraction, and cell proliferation (26). The Multi-Ethnic Study of Atherosclerosis's prospective analysis revealed

that a 20% – 40% increase in the incidence of hypertension is associated with variations in the circulations of hs-CRP and IL-6 (27). Researchers have shown that lowering circulating TNF-α levels can lower blood pressure in patients who are spontaneously hypertensive [25, 28].

It has been documented that the immune system contributes to the aetiology of hypertension. It is proposed that both innate and adaptive immunity cells infiltrate the vessels of hypertensive patients [25]. These cells also secrete inflammatory cytokines, including hs-CRP, IL-6, and TNF-α, which enhance the vascular and ultimately the tissue damage [29]. Hs-CRP and TNF-a have been shown to suppress nitric oxide synthesis in the endothelium, promoting vasospasm, platelet aggregation, oxidative processes, leukocyte adhesion, and thrombus formation [29]. Moreover, TNF-α induces vascular smooth muscle cell proliferation, thereby thickening the vascular wall and decreasing the lumen diameter, increasing peripheral resistance and thereby increasing blood pressure [30]. The liver is the source of CRP production [31], which is regulated by IL-6 and results in the formation of PDGF, which leads to contraction of vascular smooth muscle cells and proliferation of fibroblasts [32]. In addition, PDGF elevates reactivity to low-density lipoprotein, constricts arterial walls, and increases blood pressure [33]. This harms vascular endothelial cells (VECs), decreasing vasodilator synthesis and increasing vasoconstrictor synthesis.

Studies on humans and experimental models have shown that systemic inflammation increases arterial stiffness and the activity of the renin-angiotensin-aldosterone system (RAAS), which increases renal sodium reabsorption, enhancing blood volume and systemic vascular resistance, leading to hypertension [5, 34-35].

This study also found that fasting serum insulin and the HOMA-IR index were positively associated with inflammatory biomarker levels, but no correlation was observed between IL-6 and the HOMA-IR index, as presented in Table 3.4. These findings are in accordance with data obtained from previous studies [7, 15, 36]. A study suggested that TNFα and insulin resistance were associated in hypertensive patients [15]. Furthermore, TNF-α was found to play an important role in producing insulin resistance by suppressing the insulin signalling pathway [36].

The results indicate that hs-CRP is consistently associated with insulin resistance. For IL-6, there was no significant correlation with HOMA IR, and no causal inference can be made. But, considering that IL-6 is the main inducer of hepatic synthesis of hs-CRP, the role of IL-6 in promoting systemic insulin resistance through increased CRP production cannot be excluded; maybe IL-6 is acting indirectly via inflammation at insulin target tissues [32, 37].

The interplay between inflammation and insulin sensitivity suggests that inflammatory biomarkers may serve as critical indicators of metabolic dysfunction. Additionally, research comparing hypertensive populations reveals that individuals experiencing chronic inflammation exhibit altered gene expression profiles, highlighting the emergence of inflammation as a pivotal determinant of insulin sensitivity [30]. Chronic inflammation significantly influences insulin sensitivity, serving as a critical link between inflammatory pathways and metabolic disorders such as hypertension [7]. Evidence showed that obesity and other metabolic syndromes lead to creating a pro-inflammatory environment, which correlates inversely with insulin sensitivity and impairs insulin signaling, resulting in reduced uptake of glucose into the target tissues such as adipose tissue and muscle, exacerbating conditions like hypertension [11, 38].

## 5. CONCLUSION

The current study found that insulin resistance and hypersecretion of pro-inflammatory biomarkers were strongly associated with an increased risk of developing hypertension. Besides, the study observed a strong correlation between insulin resistance and inflammatory biomarkers in individuals with hypertension. These data indicate that low-grade inflammation may contribute to the development of insulin resistance among hypertensive subjects.

### Author Contributions

All authors have contributed to reviewing literature, writing the manuscript, reading and approving the final version of the manuscript.

### Declaration of Interest

The authors have nothing to declare.

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