ORIGINAL RESEARCH PAPER
Volume - 15 | Issue - 01 | January - 2026 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

METABOLIC DETERMINANTS OF BLOOD PRESSURE IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSION: FOCUS ON INSULIN RESISTANCE

Endocrinology

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ABSTRACT

Background: Essential hypertension is frequently associated with insulin resistance, even in the absence of overt metabolic comorbidities. This study aimed to evaluate the relationship between insulin resistance and different stages of hypertension. **Methods:** This cross-sectional observational study was conducted at LLRM Medical College, Meerut (Uttar Pradesh). Newly diagnosed hypertensive patients were recruited from the outpatient department (OPD). A total of 300 patients were enrolled and classified into three groups according to the stages of hypertension as defined by the American Heart Association 2017 guidelines. Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Pearson's correlation coefficient was used to evaluate the relationship between blood pressure parameters and insulin resistance indices. **Results:** Mean serum insulin levels and mean HOMA-IR values increased progressively with advancing stages of hypertension, with the difference being statistically significant ($p < 0.05$). Pearson's correlation analysis demonstrated a weak but positive correlation between systolic blood pressure (SBP) and HOMA-IR ($r = 0.311$) as well as serum insulin levels ($r = 0.292$). Similarly, diastolic blood pressure (DBP) showed a weak positive correlation with HOMA-IR ($r = 0.278$) and serum insulin levels ($r = 0.267$). **Conclusion:** The findings of this study suggest that insulin resistance is positively associated with essential hypertension, even in the absence of obesity and other metabolic comorbidities. This association becomes more pronounced with increasing stages of hypertension and is observed for both systolic and diastolic blood pressure.

KEYWORDS

INTRODUCTION

Essential hypertension is a major public health problem and a leading contributor to cardiovascular morbidity and mortality worldwide, particularly in developing countries like India [1,2]. Despite extensive research, the pathophysiology of essential hypertension remains complex and multifactorial, involving genetic, environmental, neurohormonal, and metabolic factors.

Insulin resistance has been increasingly recognized as an important metabolic abnormality associated with essential hypertension. It is characterized by reduced tissue responsiveness to insulin, resulting in compensatory hyperinsulinemia [3]. Insulin resistance may contribute to elevated blood pressure through multiple mechanisms, including increased renal sodium retention, sympathetic nervous system activation, stimulation of the renin–angiotensin–aldosterone system, endothelial dysfunction, and vascular smooth muscle proliferation [4–6].

Although insulin resistance is commonly linked to obesity, diabetes mellitus, and metabolic syndrome, several studies have demonstrated its independent association with essential hypertension, even in non-obese individuals without overt metabolic comorbidities [7,8]. This association is particularly relevant in Asian populations, where insulin resistance occurs at lower body mass indices [9].

The American Heart Association 2017 guidelines introduced lower blood pressure thresholds and redefined stages of hypertension, highlighting the importance of early detection and risk stratification [10]. However, limited Indian data exist evaluating the relationship between insulin resistance and different stages of hypertension using these updated criteria. Therefore, this study aimed to assess insulin resistance using HOMA-IR and its correlation with blood pressure parameters across stages of hypertension in newly diagnosed patients.

MATERIALS AND METHODS

Study Population

This cross-sectional observational study was conducted at LLRM Medical College, Meerut (Uttar Pradesh). Newly diagnosed hypertensive patients were recruited from the outpatient department (OPD). Elevated Blood Pressure and Hypertension were defined according to the American Heart Association 2017 guidelines. A total of 300 patients were enrolled in the study after obtaining written informed consent. Ethical approval was granted by the Institutional Ethics Committee prior to commencement of the study.

Inclusion Criteria

Patients aged ≥18 years with newly diagnosed hypertension, defined as systolic blood pressure (SBP) ≥120 mmHg and/or diastolic blood pressure (DBP) ≥80 mmHg as per AHA 2017 guidelines, were included. Only treatment-naïve patients who had not received any prior antihypertensive therapy were enrolled.

Exclusion Criteria

Patients with diabetes mellitus, chronic kidney disease, or thyroid disorders were excluded. Subjects with obesity or increased body mass index as per World Health Organization criteria [11], and those fulfilling criteria for metabolic syndrome based on the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) guidelines [12], were also excluded from the study.

Study Design

This was a cross-sectional, observational study. The study population was divided into three groups according to the stages of hypertension defined by the American Heart Association 2017 guidelines [13]:

- **Group A:** Elevated blood pressure
- **Group B:** Stage 1 hypertension
- **Group C:** Stage 2 hypertension

Study Procedure

All participants underwent detailed history taking and thorough clinical examination, including measurement of body mass index (BMI) and fundus examination. Blood pressure was measured in accordance with AHA 2017 guidelines.

Insulin resistance was assessed using the surrogate marker Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) [14]. Higher HOMA-IR values indicate greater insulin resistance. HOMA-IR was calculated using the following formula:

$$\text{HOMA-IR} = \frac{\text{Fasting insulin (mIU/L)} \times \text{Fasting plasma glucose (mg/dl)}}{405}$$

All patients were also evaluated with kidney function tests, liver function tests, thyroid function tests, fasting lipid profile, blood glucose profile including HbA1c, and complete haemogram. Additional investigations were performed as clinically indicated.

Statistical Analysis

Data were tabulated in Microsoft Excel and analyzed using IBM SPSS Statistics version 25. Quantitative variables with parametric distribution were expressed as mean ± standard deviation (SD). Comparison of quantitative variables across more than two groups was performed using one-way analysis of variance (ANOVA). A p value of <0.05 was considered statistically significant.

Correlation between two quantitative variables was assessed using

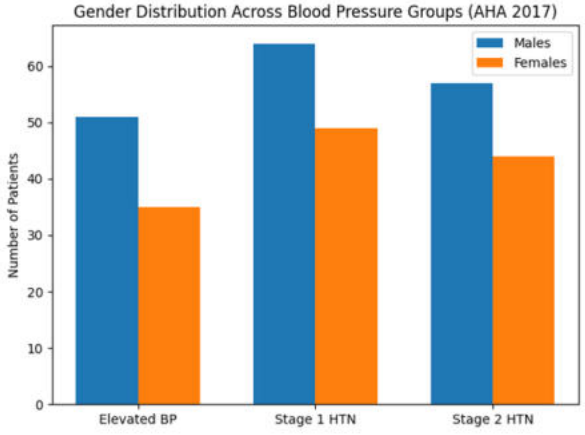
Pearson's correlation coefficient. Correlation analysis was considered statistically significant at a p value of <0.01.

Observations

Of the 300 patients enrolled in the study, 86 subjects had elevated blood pressure (systolic blood pressure [SBP] 120–129 mmHg and diastolic blood pressure [DBP] <80 mmHg). A total of 113 patients were classified as having stage 1 hypertension (SBP 130–139 mmHg and/or DBP 80–89 mmHg), while 101 subjects had stage 2 hypertension (SBP >140 mmHg and/or DBP ≥90 mmHg), as per the American Heart Association 2017 guidelines.

Baseline Characteristics

The gender distribution revealed 51 males and 35 females in Group A (elevated blood pressure), 64 males and 49 females in the stage 1 hypertension group, and 57 males and 44 females in the stage 2 hypertension group (Figure 1).



Baseline characteristics including age, body mass index (BMI), fasting blood sugar (FBS), and glycated hemoglobin (HbA1c) were comparable across all three groups. Patients included in the study were non-obese and non-diabetic, and none fulfilled criteria for metabolic syndrome

S. No.	Parameter	Elevated BP	Stage 1 Hypertension	Stage 2 Hypertension	p-value
1	Mean Age (years)	51.81 ± 6.43	52.91 ± 12.61	54.63 ± 12.92	0.51
2	Mean BMI (kg/m²)	22.4 ± 1.3	22.1 ± 1.2	21.9 ± 1.3	0.59
3	Mean FBS (mg/dL)	83.25 ± 8.41	86.67 ± 9.02	88.85 ± 7.89	0.57
4	Mean HbA1c (%)	5.36 ± 0.42	5.48 ± 0.39	5.52 ± 0.37	0.08
5	Mean SBP (mmHg)	124	134	152	—
6	Mean DBP (mmHg)	76	84	94	—

Table 1 shows the insulin levels and HOMA-IR values in the three study groups. It was seen that as the stage of hypertension progressed, mean insulin levels and mean HOMA-IR also increased with a significant difference among the groups (p - value < 0.05).

Parameter	Elevated BP	Stage 1 Hypertension	Stage 2 Hypertension	*p*-value
Serum insulin (mIU/L)	5.12 ± 2.88	7.94 ± 4.62	9.56 ± 6.91	<0.001
HOMA-IR	1.14 ± 0.67	1.76 ± 1.05	2.08 ± 1.58	<0.001

Table II: Insulin Level And HOMA-IR In Different Study Groups.

Parameter	Systolic Blood Pressure (SBP)	*p*-value	Diastolic Blood Pressure (DBP)	*p*-value
Serum insulin (µIU/mL)	0.304	<0.001	0.271	0.001
HOMA-IR	0.286	<0.001	0.259	0.001

Table III: Correlation Of Blood Pressure With Insulin Resistance And HOMA-IR.

In all three groups, correlation analysis demonstrated a **modest but statistically significant positive correlation** between systolic blood pressure and serum insulin levels as well as HOMA-IR. Similarly, diastolic blood pressure showed a **modest yet statistically significant positive correlation** with both serum insulin levels and HOMA-IR

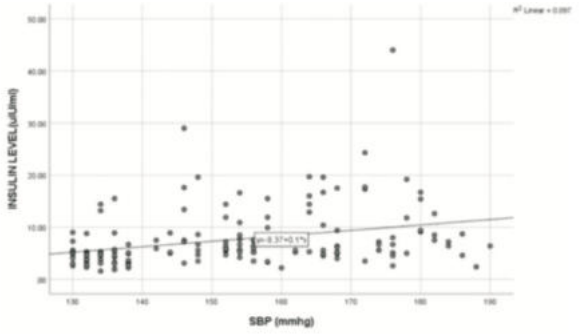


Fig. 2: *Scatter plot between SBP and insulin level.*

Pearson's correlation co-efficient r (0.311) between SBP and insulin level with p value < 0.001; depicting weak significant positive correlation.

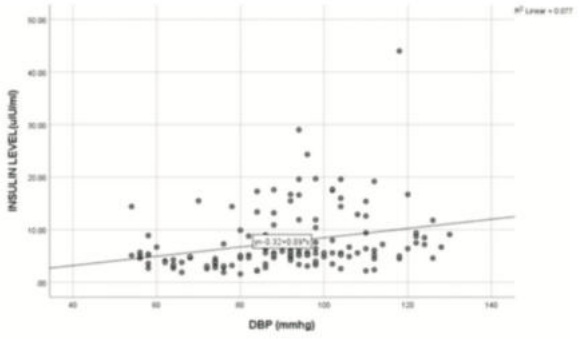


Fig. 3: *Scatter plot between DBP and insulin level.*

Pearson's correlation co-efficient r (0.278) between DBP and insulin level with p value of 0.001 suggesting weak positive significant correlation.

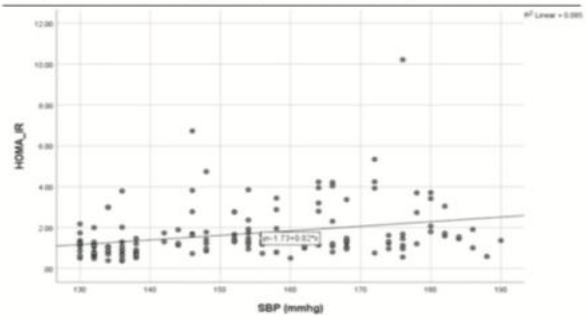


Fig. 4: *Scatter plot between SBP and HOMA-IR.*

Pearson's correlation co-efficient (0.292) between SBP and HOMA-IR with p value of < 0.001 suggesting positive correlation that too significant.

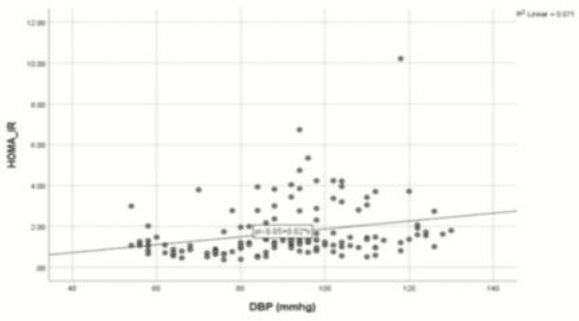


Fig. 5: *Scatter plot between DBP and HOMA-IR.*

Pearson's correlation co-efficient (0.267) between DBP and HOMA-IR with p value 0.001 suggesting significant positive correlation.

DISCUSSION

Insulin resistance and hypertension frequently coexist and are key components of the metabolic syndrome. Although the exact pathogenesis of essential hypertension remains incompletely understood, multiple environmental, genetic, and metabolic factors have been implicated, with insulin resistance being an important contributor. Independent of its metabolic effects, insulin plays a role in blood pressure regulation by enhancing renal sodium reabsorption, thereby promoting sodium retention and volume expansion [7]. Hyperinsulinaemia is also known to stimulate the sympathetic nervous system, which further contributes to elevation of blood pressure. Under physiological conditions, insulin induces endothelial nitric oxide production, leading to vasodilatation; however, in insulin-resistant states, this vasodilatory response is impaired, resulting in relative vasoconstriction and increased vascular resistance [8]. These mechanisms collectively may predispose insulin-resistant individuals to the development of hypertension.

In addition, patients with essential hypertension have been shown to exhibit defects in insulin receptor signaling. Reduced insulin action at the insulin receptor leads to diminished activation of receptor tyrosine kinase, decreased receptor autophosphorylation, and impaired phosphorylation of insulin receptor substrate-1 (IRS-1). This results in reduced activation of phosphatidylinositol-3-kinase, decreased glucose transport into skeletal muscle, and impaired peripheral glucose uptake. Compensatory hyperinsulinaemia subsequently develops to maintain normoglycaemia [9]. Thus, the coexistence of insulin resistance and hypertension may represent a bidirectional or non-causal relationship, with shared pathophysiological pathways.

Several studies support an association between essential hypertension and insulin resistance. Demissie et al. demonstrated that hypertension, insulin resistance, and oxidative stress are associated with shorter leukocyte telomere length, suggesting accelerated cellular aging in hypertensive individuals, largely mediated by insulin resistance [10]. Sinha et al. also reported a higher prevalence of insulin resistance among essential hypertensive subjects compared with normotensive controls in a South Asian population [11]. Other studies have proposed that hyperinsulinaemia-induced sympathetic overactivity may cause microvascular structural changes, leading to increased blood pressure and reduced peripheral glucose uptake [12]. Similar positive associations between insulin resistance and hypertension have been reported by Haffner et al. [13], Welborn et al. [14], and Penesova et al. [15].

In the present study, patients were classified into three categories-elevated blood pressure, stage 1 hypertension, and stage 2 hypertension-as per AHA 2017 guidelines. All groups were comparable with respect to age, sex, body mass index, fasting blood glucose, and HbA1c. Patients with obesity and metabolic syndrome were excluded, thereby minimizing confounding metabolic influences. We observed a progressive and statistically significant increase in fasting serum insulin levels and HOMA-IR with advancing stages of hypertension. Both serum insulin levels and HOMA-IR showed a significant positive correlation with systolic and diastolic blood pressure.

However, not all studies have demonstrated a consistent association. Baba et al. [16] and Every et al.[17]did not find a significant relationship between serum insulin levels and blood pressure in non-obese, middle-aged individuals. These discrepancies suggest heterogeneity in the insulin–hypertension relationship, possibly due to differences in study populations, methodologies, and confounding factors.

Overall, although substantial evidence supports a positive association between insulin resistance and essential hypertension, the causal direction remains unresolved. Insulin resistance and hypertension share common lifestyle and pathophysiological mechanisms, including chronic inflammation, endothelial dysfunction, and sympathetic overactivity. Therefore, lifestyle modification and pharmacological interventions that improve insulin sensitivity may have a beneficial role in reducing the risk of hypertension, metabolic syndrome, and subsequent cardiovascular disease.

CONCLUSION

Based on our observations, we conclude that insulin resistance is positively associated with essential hypertension even in the absence of obesity and other metabolic comorbidities. This association is more pronounced with increasing stages of hypertension and is evident for both systolic and diastolic blood pressure. Therefore, early identification of insulin resistance and timely implementation of appropriate lifestyle modification and pharmacological interventions may help in the prevention and management of both insulin resistance and hypertension, irrespective of which condition is detected first.

Limitation Of The Study

As this was a single-center study conducted in a population from a single ethnic background, the generalizability of the findings is limited. Larger multicenter studies and meta-analyses involving diverse populations are required to further explore these observations and to evaluate their role in preventing the development of metabolic syndrome, dyslipidaemia, and diabetes mellitus in patients with essential hypertension without obesity or other comorbidities.

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