

# Impact of Reduced Oxygen Carrying Capacity on Hypertension: A Comparative Study of Hemoglobin and Hematocrit Levels in Hypertensive and Normotensive Individuals

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## ABSTRACT

**Background:** Hypertension is a leading modifiable risk factor for cardiovascular morbidity and mortality. While the role of neurohumoral and vascular mechanisms in hypertension is well established, the contribution of oxygen-carrying capacity - reflected by hemoglobin and hematocrit levels - remains underexplored. Alterations in these parameters can influence blood viscosity, vascular resistance, and tissue oxygenation, thereby affecting cardiovascular regulation. This study aimed to assess hemoglobin and hematocrit levels in hypertensive and normotensive individuals and evaluate their relationship with blood pressure.

**Methodology:** A cross-sectional comparative study was conducted on 80 adults aged 25-60 years, including 40 hypertensive and 40 normotensive participants matched for age and sex. Blood pressure was measured using a mercury sphygmomanometer after adequate rest, and venous blood samples were analyzed for hemoglobin (g/dL), hematocrit (%), and red blood cell (RBC) count using an automated hematology analyzer. Statistical analyses were performed using SPSS v25.0. Independent *t*-tests compared group means, Pearson's correlation assessed associations, and multiple regression determined independent predictors of blood pressure. A *p*-value < 0.05 was considered significant.

**Results:** Hypertensive individuals exhibited significantly lower hemoglobin ( $13.21 \pm 1.09$  g/dL vs  $14.13 \pm 0.94$  g/dL,  $p = 0.003$ ) and hematocrit ( $39.5 \pm 3.4$  % vs  $42.0 \pm 3.0$  %,  $p = 0.012$ ) compared to normotensives. Within the hypertensive group, hemoglobin and hematocrit showed moderate positive correlations with systolic and diastolic pressures ( $r = 0.38-0.43$ ,  $p < 0.05$ ). Regression analysis identified hemoglobin ( $\beta = 0.36$ ,  $p = 0.006$ ) and hematocrit ( $\beta = 0.29$ ,  $p = 0.018$ ) as independent predictors of systolic pressure ( $R^2 = 0.42$ ).

**Conclusion:** The coexistence of lower mean hemoglobin and hematocrit in hypertensive individuals, along with a positive intra-group correlation with blood pressure, indicates a complex interplay between oxygen-carrying capacity and cardiovascular regulation. Both reduced oxygen transport and increased blood viscosity may contribute to altered hemodynamics in hypertension. Routine assessment of hematological indices could enhance understanding and management of hypertensive patients.

**Keywords:** Hypertension; Hemoglobin; Hematocrit; Blood viscosity; Cardiovascular regulation

## INTRODUCTION

Hypertension is one of the most prevalent chronic non-communicable diseases and a major modifiable risk factor for cardiovascular morbidity and mortality worldwide. Global epidemiological estimates suggest that elevated blood pressure contributes substantially to the burden of ischemic heart disease, stroke, chronic kidney disease, and premature death, affecting more than one billion individuals globally.<sup>[1]</sup> Persistent elevation of arterial pressure leads to progressive target-organ damage involving the heart, brain, kidneys, and vasculature.<sup>[2]</sup>

The pathophysiology of hypertension is multifactorial and has traditionally been explained by the “mosaic theory,” which proposes that multiple interacting mechanisms - including genetic predisposition, sympathetic nervous system activation, renin-angiotensin-aldosterone system dysregulation, endothelial dysfunction, oxidative stress, and alterations in renal sodium handling - collectively contribute to sustained elevation of blood pressure.<sup>[3]</sup> In addition to these well-established mechanisms, hemorheological factors such as blood viscosity and red blood cell characteristics have increasingly been recognized as potential modulators of vascular resistance and circulatory dynamics.<sup>[4]</sup>

Oxygen-carrying capacity of blood is primarily determined by hemoglobin concentration and hematocrit, which together influence oxygen delivery to peripheral tissues. Hemoglobin is the iron-containing protein within erythrocytes responsible for oxygen transport from the lungs to tissues, whereas hematocrit represents the proportion of blood volume occupied by red blood cells. Alterations in these parameters may significantly affect systemic hemodynamics, since hematocrit is a major determinant of whole-blood viscosity and vascular resistance.<sup>[5]</sup> Increased hematocrit elevates

blood viscosity, which may raise peripheral vascular resistance and subsequently influence arterial pressure.<sup>[6]</sup>

Conversely, reduced hemoglobin levels may impair oxygen delivery and induce compensatory cardiovascular responses aimed at maintaining adequate tissue perfusion. Such adaptations may include increased cardiac output, activation of sympathetic and renin-angiotensin systems, and changes in vascular tone.<sup>[7]</sup> Chronic tissue hypoxia may also promote endothelial dysfunction, oxidative stress, and alterations in nitric oxide bioavailability, thereby contributing to vascular remodeling and increased arterial stiffness.<sup>[8]</sup> These physiological changes suggest a potential relationship between oxygen-carrying capacity and the regulation of systemic blood pressure.

Several epidemiological studies have explored the association between hematological parameters and hypertension. Large population-based investigations have demonstrated that higher hemoglobin or hematocrit levels are associated with increased blood pressure and a greater prevalence of hypertension.<sup>[9]</sup> For instance, findings from the Gubbio Population Study showed a positive relationship between hematocrit levels and both systolic and diastolic blood pressure in a community-based cohort.<sup>[10]</sup> However, the relationship between reduced oxygen-carrying capacity and hypertension remains less clearly understood. Most existing studies primarily focus on elevated hemoglobin or hematocrit levels rather than examining the potential role of lower values in influencing cardiovascular regulation.

Understanding the interaction between hemoglobin, hematocrit, and blood pressure may provide additional insight into the physiological determinants of hypertension. Given the high prevalence of anemia and altered hematological parameters in many populations, particularly in developing

countries, evaluating the relationship between oxygen-carrying capacity and blood pressure could have important clinical and physiological implications.

Therefore, the present study was undertaken to compare hemoglobin and hematocrit levels between hypertensive and normotensive adults and to examine their association with blood pressure parameters. By exploring these relationships, the study aims to contribute to a better understanding of the role of oxygen transport variables in the pathophysiology of hypertension.

## METHODOLOGY

This study was designed as a cross-sectional comparative investigation conducted in the Department of Physiology, MGM Medical College, Aurangabad after obtaining approval from the Institutional Ethics Committee (MGM-ECRHS/PHYSIOLOGY/MD/2024/02).

Eighty adult participants aged 25–60 years were enrolled, comprising 40 clinically diagnosed hypertensive individuals (study group) and 40 age- and sex-matched normotensive controls (control group). Participants were recruited from the outpatient department (OPD) and health check-up clinics of MGM Hospital. Hypertensive subjects were selected based on established criteria—systolic blood pressure (SBP)  $\geq 140$  mmHg and/or diastolic blood pressure (DBP)  $\geq 90$  mmHg, or current use of antihypertensive medication as per the Joint National Committee (JNC 8) guidelines. Normotensive subjects were healthy volunteers with SBP  $< 130$  mmHg and DBP  $< 85$  mmHg and no history of hypertension. Participants with anemia due to chronic diseases, renal disorders, diabetes mellitus, thyroid dysfunction, smoking, alcohol abuse, or on medications influencing erythropoiesis or blood viscosity were excluded to eliminate confounding factors.

After obtaining written informed consent, participants underwent a detailed clinical evaluation. Height and weight were

measured using a stadiometer and calibrated digital scale, respectively, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared ( $\text{kg}/\text{m}^2$ ). Blood pressure was recorded in the right arm in a seated position using a standard mercury sphygmomanometer following a 10-minute rest period. Three readings were taken at 2-minute intervals, and the mean value was used for analysis. Systolic, diastolic, and mean arterial pressure (MAP) were computed to assess cardiovascular status.

Venous blood samples were collected under aseptic precautions in the morning hours after an overnight fast of 8–10 hours. Hemoglobin concentration (g/dL) was determined by the cyanmethemoglobin method using an automated hematology analyzer. Hematocrit (%) was estimated via the microhematocrit method and verified with automated cell counter readings. Red blood cell (RBC) count ( $\times 10^6/\mu\text{L}$ ) was also recorded to assess erythrocyte indices. All assays were performed in the institutional clinical laboratory following standard quality control procedures.

## Statistical Analysis

Data were entered and analyzed using IBM SPSS Statistics (Version 25.0). Results were expressed as mean  $\pm$  standard deviation (SD). Normality of data was assessed using the Shapiro–Wilk test. Comparisons between hypertensive and normotensive groups were made using independent samples *t*-test for continuous variables. Pearson's correlation coefficients were calculated to examine relationships between hemoglobin, hematocrit, and blood pressure parameters. Multiple linear regression analysis was performed to identify independent predictors of systolic and diastolic blood pressure after adjusting for confounding variables such as age, BMI, and sex. A *p*-value  $< 0.05$  was considered statistically significant.

## RESULTS

**Table 1. Baseline Demographic and Clinical Characteristics of the Study Population (n = 80)**

Variable	Hypertensive Group (Mean ± SD)	Normotensive Group (Mean ± SD)	p-value
Age (years)	52.1 ± 10.3	50.4 ± 9.1	0.28
BMI (kg/m <sup>2</sup> )	27.2 ± 3.1	24.3 ± 2.6	<b>0.001 *</b>
Systolic BP (mmHg)	148.2 ± 10.1	120.6 ± 7.9	<b>&lt; 0.001 *</b>
Diastolic BP (mmHg)	92.4 ± 8.2	78.1 ± 6.1	<b>&lt; 0.001 *</b>

Table 1 summarizes the demographic and clinical characteristics of the study population. The mean age of hypertensive participants was 52.1 ± 10.3 years, comparable to 50.4 ± 9.1 years in the normotensive group ( $p = 0.28$ ), indicating adequate age matching. Body mass index (BMI) was significantly higher among hypertensive subjects (27.2 ± 3.1 kg/m<sup>2</sup>) compared to normotensive controls (24.3 ±

2.6 kg/m<sup>2</sup>,  $p = 0.001$ ). As expected, both systolic and diastolic blood pressures were markedly elevated in the hypertensive group (148.2 ± 10.1 mmHg and 92.4 ± 8.2 mmHg, respectively) compared with normotensives (120.6 ± 7.9 mmHg and 78.1 ± 6.1 mmHg, both  $p < 0.001$ ). These findings confirm appropriate group differentiation and the presence of established hypertension in the study cohort.

**Table 2. Comparison of Hemoglobin, Hematocrit, and RBC Count Between Groups**

Parameter	Hypertensive (Mean ± SD)	Normotensive (Mean ± SD)	p-value
Hemoglobin (g/dL)	13.21 ± 1.09	14.13 ± 0.94	<b>0.003 *</b>
Hematocrit (%)	39.5 ± 3.4	42.0 ± 3.0	<b>0.012 *</b>
RBC Count (×10 <sup>6</sup> /μL)	4.52 ± 0.39	4.81 ± 0.31	<b>0.021 *</b>

As shown in Table 2, hypertensive individuals exhibited a modest but statistically significant reduction in hemoglobin and hematocrit values compared with normotensive subjects. The mean hemoglobin concentration in hypertensives was 13.21 ± 1.09 g/dL versus 14.13 ± 0.94 g/dL in normotensives ( $p = 0.003$ ). Similarly, mean hematocrit was 39.5 ± 3.4% in hypertensives and 42.0 ± 3.0% in

normotensives ( $p = 0.012$ ). The RBC count followed a similar trend, being slightly lower among hypertensives (4.52 ± 0.39 × 10<sup>6</sup>/μL) compared with controls (4.81 ± 0.31 × 10<sup>6</sup>/μL,  $p = 0.021$ ). These results suggest a relative reduction in oxygen-carrying capacity in hypertensive individuals, even within clinically normal ranges of hematological parameters.

**Table 3. Correlation Between Blood Pressure and Oxygen-Carrying Parameters**

Variables	Pearson's r (Hypertensive)	p-value (Hypertensive)	Pearson's r (Normotensive)	p-value (Normotensive)
Hemoglobin vs SBP	0.41	0.008 *	0.18	0.27
Hemoglobin vs DBP	0.38	0.014 *	0.16	0.31
Hematocrit vs SBP	0.43	0.005 *	0.22	0.18
Hematocrit vs DBP	0.40	0.009 *	0.19	0.24

Table 3 presents the correlations between blood pressure indices and oxygen-carrying parameters. In the hypertensive group, hemoglobin demonstrated a moderate

positive correlation with systolic blood pressure ( $r = 0.41$ ,  $p = 0.008$ ) and diastolic pressure ( $r = 0.38$ ,  $p = 0.014$ ). Hematocrit also correlated positively with both systolic

( $r = 0.43, p = 0.005$ ) and diastolic pressure ( $r = 0.40, p = 0.009$ ). In contrast, correlations were weaker and statistically insignificant in the normotensive group. These findings indicate that within the hypertensive

population, higher hemoglobin and hematocrit levels are associated with greater blood pressure elevations, possibly reflecting increased blood viscosity and vascular resistance.

**Table 4. Multiple Linear Regression Analysis for Predictors of Systolic Blood Pressure**

Independent Variable	Standardized $\beta$	t-value	p-value	R <sup>2</sup> (Model Fit)
Hemoglobin	0.36	2.87	0.006 *	<b>0.42</b>
Hematocrit	0.29	2.45	0.018 *	
Age	0.12	1.05	0.30	
BMI	0.25	2.11	0.040 *	

The results of multiple linear regression are summarized in Table 4. After adjusting for age, BMI, and sex, hemoglobin remained an independent predictor of systolic blood pressure ( $\beta = 0.36, p = 0.006$ ), while hematocrit also showed a significant but weaker association ( $\beta = 0.29, p = 0.018$ ). BMI was another significant covariate ( $\beta =$

$0.25, p = 0.04$ ). The overall model explained approximately 42% of the variance in systolic blood pressure ( $R^2 = 0.42$ ), underscoring the multifactorial nature of hypertension where hematological and anthropometric variables contribute significantly.

**Table 5. Association Between Hemoglobin Status and Hypertension (2x2 Analysis)**

Hemoglobin Status	Hypertensive (n, %)	Normotensive (n, %)	$\chi^2$ value	p-value
Abnormal Hb (<12 or >16 g/dL)	9 (22.5%)	6 (15.0%)	0.74	0.38
Normal Hb (12–16 g/dL)	31 (77.5%)	34 (85.0%)		

Table 5 presents the association between hemoglobin status and hypertension after categorizing participants into normal and abnormal hemoglobin groups. The original three-category distribution (low, normal, and high hemoglobin) was collapsed into two categories to satisfy the assumptions of the chi-square test, as more than 20% of cells in the initial analysis had expected counts less than five. In the revised analysis, 22.5% of hypertensive subjects had abnormal hemoglobin levels compared to 15.0% of normotensive individuals. However, this difference was not statistically significant ( $\chi^2 = 0.7400, p = 0.3896$ ). These findings suggest that although a higher proportion of hypertensive individuals exhibited abnormal hemoglobin levels, there was no statistically significant association between hemoglobin status and hypertension in this study population.

## DISCUSSION

The present study investigated the relationship between oxygen-carrying

capacity, reflected by hemoglobin and hematocrit levels, and blood pressure in hypertensive and normotensive adults. The main findings of this study were: (1) hypertensive individuals exhibited significantly lower mean hemoglobin and hematocrit levels compared with normotensive controls; (2) within the hypertensive group, hemoglobin and hematocrit showed moderate positive correlations with both systolic and diastolic blood pressure; and (3) regression analysis identified hemoglobin and hematocrit as independent predictors of systolic blood pressure after adjusting for potential confounders such as age and body mass index.

Several epidemiological studies have previously reported associations between hematological parameters and blood pressure regulation. Large population-based analyses have demonstrated that higher hemoglobin or hematocrit concentrations are associated with elevated blood pressure and increased prevalence of hypertension. For example,

Atsma et al. reported that hemoglobin concentration was positively associated with systolic and diastolic blood pressure in a large cohort of healthy individuals, suggesting that erythrocyte-related factors may contribute to vascular resistance and cardiovascular regulation.<sup>[9]</sup> Similarly, findings from the Gubbio Population Study demonstrated that hematocrit levels were significantly correlated with both systolic and diastolic blood pressure in community-dwelling adults, even after adjustment for age and other confounding variables.<sup>[10]</sup> These observations support the concept that hemorheological factors influence systemic hemodynamics.

In contrast to several previous studies reporting higher hemoglobin or hematocrit values among hypertensive individuals, the present study observed slightly lower mean hemoglobin and hematocrit levels in the hypertensive group compared with normotensive controls. However, despite these lower mean values, hemoglobin and hematocrit still demonstrated positive correlations with blood pressure within the hypertensive population. This apparent paradox suggests that the relationship between oxygen-carrying capacity and blood pressure may be complex and influenced by multiple physiological mechanisms rather than a simple linear association.

One possible explanation for this observation is the role of compensatory cardiovascular responses to reduced oxygen delivery. Reduced hemoglobin concentration can impair tissue oxygen transport and trigger adaptive responses aimed at maintaining adequate oxygenation. These compensatory mechanisms may include increased cardiac output, sympathetic nervous system activation, and stimulation of the renin-angiotensin-aldosterone system.<sup>[7]</sup> Such physiological responses may contribute to elevations in arterial pressure as part of systemic circulatory adaptation.

Another important factor is the influence of hematocrit on blood viscosity and vascular resistance. Hematocrit is one of the principal determinants of whole-blood viscosity, and

increases in hematocrit can substantially elevate vascular resistance.<sup>[6]</sup> Experimental and rheological studies have demonstrated that relatively small increases in hematocrit may lead to disproportionately larger increases in blood viscosity and resistance to flow, thereby influencing arterial pressure.<sup>[4]</sup> These hemorheological effects may explain the positive correlations observed between hemoglobin, hematocrit, and blood pressure in the hypertensive group in the present study.

Furthermore, chronic hypoxia and altered oxygen delivery may also contribute to vascular dysfunction. Hypoxia-related changes in endothelial function, oxidative stress, and nitric oxide bioavailability can promote vasoconstriction, vascular remodeling, and increased arterial stiffness, all of which may contribute to the development or progression of hypertension.<sup>[8]</sup> Therefore, both reduced oxygen transport and increased blood viscosity may act through different physiological pathways to influence cardiovascular regulation.

The findings of the present study suggest that oxygen-carrying capacity may play a modulatory role in blood pressure regulation, particularly within hypertensive individuals. The coexistence of lower average hemoglobin levels alongside positive correlations between hematological parameters and blood pressure indicates that multiple interacting mechanisms may be involved. It is therefore plausible that both extremes of oxygen-carrying capacity—reduced hemoglobin leading to compensatory cardiovascular activation and increased hematocrit leading to elevated viscosity—may influence blood pressure through distinct physiological pathways.

In addition, categorical analysis of hemoglobin status, performed after appropriate grouping to satisfy statistical assumptions, showed no significant association with hypertension ( $p = 0.3896$ ), although a higher proportion of hypertensive individuals exhibited abnormal hemoglobin levels.

## CONCLUSION

The present study demonstrated that hypertensive individuals had modestly lower hemoglobin and hematocrit levels compared with normotensive controls, while within the hypertensive group, higher values of these oxygen-carrying parameters were positively correlated with both systolic and diastolic blood pressures. Regression analysis confirmed hemoglobin and hematocrit as independent predictors of systolic pressure after controlling for confounders. These findings suggest alterations in oxygen-carrying capacity, whether due to reduced hemoglobin or elevated viscosity, may play a contributory role in the regulation of blood pressure and may influence the pathophysiology of hypertension.

### Declaration by Authors

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