



“STUDY OF HAEMATOLOGICAL PARAMETERS IN HYPERTENSION AND THEIR CORRELATION WITH BLOOD PRESSURE.”

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ABSTRACT

Context: Increasing evidence has underlined the importance of haematological parameters in the Pathophysiology of hypertension and its contribution to the associated complications. However the correlation between haematological parameters and blood pressure levels is not clearly understood. **Aims:** Study was done to evaluate the

haematological parameters in patients with hypertension and to check their correlation, if any, between these parameters and systolic and diastolic blood pressure. **Settings and Design:** Its observational study. **Methods and Material:** Hemoglobin (Hb), Packed cell volume (PCV) and Red blood cell (RBC) count were measured in thirty hypertensive and thirty normotensive subjects. Statistical analysis used: Pearson's correlation analysis was performed to study the correlation between these parameters and systolic and diastolic blood pressure. **Results:** Hemoglobin (Hb), packed cell volume (PCV) correlated negatively with systolic and diastolic blood pressure in hypertensive. No significant correlation was observed between these parameters and blood pressure levels in normotensive controls. **Conclusions:** Lesser hemoglobin level was observed in hypertensive subjects as denoted by decreased Hb levels and decreased PCV.

KEYWORDS: Haematological parameters, Hypertension, Correlation, Hemoglobin (Hb), packed cell volume (PCV) and Red blood cell (RBC) count.

INTRODUCTION

Hypertension is one of the important public health problems worldwide. A recent report on the global burden of hypertension indicates that nearly one million adults had hypertension in

2000, and this is expected to increase to 1.56 million by 2025.^[1] A meta-analysis of hypertension prevalence rates in India showed a significant rise in the prevalence of the disease over the years and the disease burden in India are now almost comparable to those in the USA.^[2] Hypertension is the most common cardiovascular disease and major cardiovascular risk factor that cause significant morbidity and mortality. Complex interaction of multiple vascular effectors including the activation of the sympathetic nervous system, renin–angiotensin–aldosterone system and the inflammatory mediators is attributed to the pathophysiology of hypertension.

Primary (essential) hypertension is the most common, 90–95% among all form of hypertension.^[3] Excessive sympathetic activity is consistently present in hypertensive patients since their childhood. Multiple mechanisms by which sympathetic over activity could cause both the metabolic syndrome and hypertension have been documented.^[4] Sympathetic over activity which is related to the stress has major impact on the cardiovascular, autonomic and hematological parameters. Long standing hypertension (sympathetic overactivity) might progress to coronary artery disease (CAD)^[5], congestive heart failure (CHF), end stage renal disease (ESRD), stroke or atherosclerotic events.^[6,7] Hypertension if not treated leads to cardiac and renal failure. Previously it is shown that hypertensives have tendency for lesser hemoglobin level as compare to normotensives.^[8,9] Nevertheless, the available data is not conclusive and the association between human blood pressure and oxidative stress remains to be elucidated. So the present study was undertaken to assess the haematological parameters in randomly selected normotensive and essential hypertensive subjects and to study the relationship, if any, between arterial blood pressure levels and these parameters.

MATERIALS AND METHODS

Cross sectional design was used for this study. The study protocol was approved by Institutional Ethics Committee.

Subjects

The study included thirty newly diagnosed hypertensive patients never treated previously for hypertension. All patients were between the ages of 23 to 37 years. Hypertension was defined according to the criteria of the VII Joint National Committee (SBP >140 mmHg and DBP > 90 mmHg). Thirty normotensive healthy volunteers served as controls. Blood pressure was measured using a mercury sphygmomanometer according to the recommendations of the British Hypertension Society. After explaining the study details, informed consent was

obtained from all the participants. Patients with cerebrovascular or coronary artery disease, congestive heart failure, diabetes, renal or liver disease and any active viral or bacterial infection were excluded from the study. Smokers, alcoholics and subjects taking any medication known to affect haematological parameters were also excluded.

Table 1. General characteristics of study population.

Variable		Control (n=30)	Hypertensives (n = 30)
Age (years)		30.93±7.23	32.82 ± 8.23
Sex	Male	30	30
	Female	00	00
SBP		123.13 ± 5.14	148.8 ± 7.35*
DBP		81.67 ± 73.57	93.87 ± 3.34*

Data shown are means ± SD, * $p < 0.05$ vs. control

In all subjects 3 ml venous blood sample was collected in Na-EDTA (1mg/ml) tubes and subjected to hematological analysis. Hematological parameters are assessed by *ABX MICROS 60 CS/CT16* complete blood cell counter machine. Hemoglobin, Packed cell volume (PCV), RBC counts were recorded.

Statistical analysis

Data were expressed as mean ± SD. Student t test was used to assess statistical differences between the groups. Pearson's correlation analysis was performed to study the correlation between hematological parameters and systolic and diastolic blood pressure. Differences were considered statistically significant at $p < 0.05$.

RESULTS

Thirty hypertensive and thirty age matched normotensive controls were included in the study. Baseline characteristics of both groups are shown in table 1.

Table 2. Haematological parameters in normotensive and hypertensive subjects.

Variable	Control (n=30)	Hypertensives (n = 30)
Hemoglobin (g/dl)	13.81± 0.80	13.32 ± 1.03*
PCV %	37.11 ± 3.41	34.92 ± 3.12*
RBC count/micro L	4.3 ± 0.45	4.25 ± 0.40

Data shown are means ± SD, * $p < 0.05$ vs. control

Hematological parameters in both groups are shown in Table 2. Hb level and PCV are significantly lesser in hypertensive group compared to controls ($p < 0.05$) while there is no significant difference in RBC count ($p > 0.05$). We further studied the relationship between

these three parameters and systolic and diastolic blood pressure levels in both groups. Pearson correlation coefficients assessed between the different parameters and systolic and diastolic BP. HB and PCV correlated negatively with SBP and DBP in hypertensive subjects (fig. 1A and 1B). RBC count showed no correlation with SBP and DBP in hypertensive subjects (fig 1C). No correlation was observed between any of these parameters with SBP and DBP in controls (fig 1D, 1E and 1F).

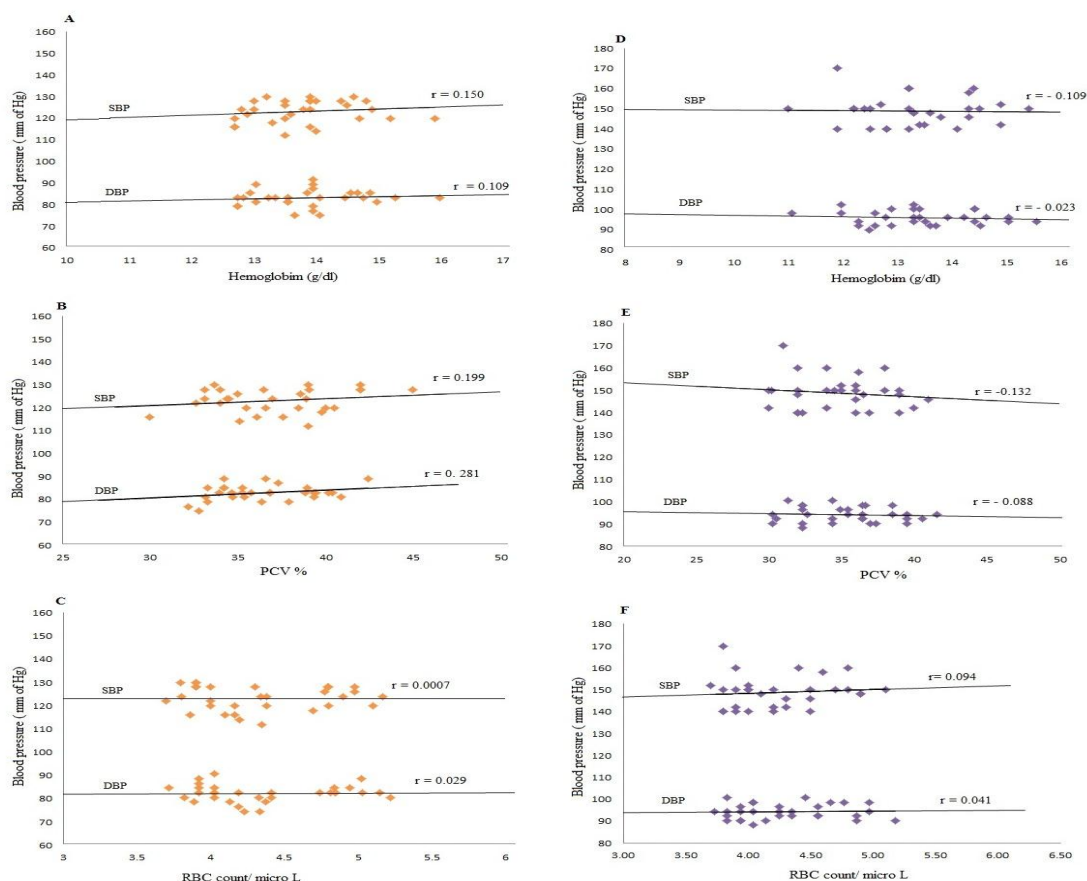


Figure 1. Pearson Correlation (r) between SBP or DBP and Hb, PCV and RBC count (1A, 1B, 1C) and Hypertensive subjects (1D, 1E, 1F)

DISCUSSION

Most common variant of hypertension is essential hypertension, where increased sympathetic overactivity is responsible for majority of cases.^[10] The present study was done to evaluate the haematological parameters in the patients with essential hypertension and to assess the relationship, if any, between these parameters and systemic blood pressure levels. We observed that HB and PCV levels were significantly lesser in the patients of essential hypertension. It is suggested that in hypertension, sympathetic activation stimulates heart, elevating cardiac output, causing naturally mediated vasoconstriction, and augmenting renin

secretion and tubular reabsorption of sodium, increasing total body fluid volume.^[11] There are multiple factors responsible for lesser hemoglobin level in cardiovascular disease like hemodilution^[12], pro-inflammatory cytokines, malnutrition due to right-sided heart failure, iron deficiency, decreased bone marrow perfusion and drug therapy for hypertension (like angiotensin converting enzyme inhibitors, aspirin), decreased erythropoietin production and decreased iron supply for erythropoiesis. Several of these mechanisms act simultaneously, and the anemia is the result of a complex interaction between them.^[13,14] Long standing high blood pressure affects kidney, heart and many organs leading to chronic anemia.^[15,16] Sympathetic overactivity associated increase in β_2 mediated renin release results in hemodilution via aldosterone.^[17] This sympatho-adrenal axis induce hemodilution is responsible for lesser Hb in hypertensives as compared to normotensives.^[18] In CHF pseudo anemia is mainly because of hemodilution besides other factors.^[19] The present study showing statistically significant lesser hemoglobin and PCV level in hypertensive patients confirms the results of previous studies. However whether the hemodilution is cause or the consequence of hypertension is not clear and it needs further evaluation. We further examined the possible relationship between these parameters and systemic blood pressure levels. Negative correlation was observed between Hb and PCV level and systolic and diastolic blood pressure in hypertensive subjects. These findings point to the fact that severity of lesser Hb and PCV level parallels the degree of rise in blood pressure in patients with hypertension. The correlation observed in this study assumes a great importance due to the fact that low Hb level might contributes to the complications of hypertension including heart failure. Thus it seems logical that normalization of BP may considerably check haematological parameters and reduce the complications of hypertension. Classic antihypertensive agents such as some β -adrenergic blockers, reduces sympathetic over activity. In view of their ability to decrease the sympathetic over activity in addition to their blood pressure lowering effect these agents may prove beneficial in long term treatment of hypertension.

CONCLUSION

Reduced Hb and PCV level in patients with hypertension as evidenced by the significant changes in the levels the haematological parameters. Moreover, these parameters correlate with the systolic and diastolic blood pressure in hypertension. Thus attempts to lower blood pressure could prove beneficial in such patients by improving Hb and PCV level.

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REFERENCES

1. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. *Lancet*. 2005 Jan 15-21; 365(9455): 217-23. PubMed PMID: 15652604. Epub 2005/01/18. eng.
2. Gupta R. Trends in hypertension epidemiology in India. *J Hum Hypertens.*, 2004 Feb; 18(2): 73-8. PubMed PMID: 14730320. Epub 2004/01/20. eng.
3. Carretero OA, Oparil S. Essential hypertension. Part I: definition and etiology. *Circulation.*, 2000 Jan 25; 101(3): 329-35. PubMed PMID: 10645931. Epub 2000/01/25. eng.
4. Julius S, Nesbitt S. Sympathetic overactivity in hypertension. A moving target. *Am J Hypertens.*, 1996 Nov; 9(11): 113S-20S. PubMed PMID: 8931844. Epub 1996/11/01. eng.
5. Fogari R, Zoppi A. [Is sympathetic hyperactivity a coronary risk factor?]. *Cardiologia*. 1993 Dec; 38(12 Suppl 1): 427-34. PubMed PMID: 8020045. Epub 1993/12/01. E l'iperattività simpatica un fattore di rischio coronarico? ita.
6. Julius S. Effect of sympathetic overactivity on cardiovascular prognosis in hypertension. *European heart journal*. 1998 Jun; 19 Suppl F: F14-8. PubMed PMID: 9651730. Epub 1998/07/04. eng.
7. Julius S. The evidence for a pathophysiologic significance of the sympathetic overactivity in hypertension. *Clin Exp Hypertens*. 1996 Apr-May; 18(3-4): 305-21. PubMed PMID: 8743023. Epub 1996/04/01. eng.
8. Aswini Dutt.R RC, Niranjana murthy.H.L, Satish Kumar NS, Shankar Bhat.K. Do hypertensives have tendency for lesser hemoglobin concentration? *National Journal Of Basic Medical Sciences*. 2010 Oct-Dec; 1.
9. Jadeja U, J. M. Jadeja, and Shobha Naik. . "COMPARATIVE STUDY OF HAEMOGLOBIN CONCENTRATION IN HYPERTENSIVE AND NORMOTENSIVE SUBJECTS..". *Indian Journal of Applied Basic Medical Sciences.*, 2011 July; 13(17): 7.

10. Hall JE, Granger JP, Carmo JM, Silva AA, Dubinon J, George E, et al. Hypertension: Physiology and Pathophysiology. *Comprehensive Physiology*.
11. Somers VK. Cardiovascular manifestations of Autonomic disorders. 9th ed. Bonow M, Zipes and Libby, editor. India: Elsevier., 2011; 13.
12. Androne AS, Katz SD, Lund L, LaManca J, Hudaihed A, Hryniewicz K, et al. Hemodilution is common in patients with advanced heart failure. *Circulation*. 2003 Jan 21; 107(2): 226-9. PubMed PMID: 12538419. Epub 2003/01/23. eng.
13. Efstratiadis G, Konstantinou D, Chytas I, Vergoulas G. Cardio-renal anemia syndrome. *Hippokratia*. 2008 Jan; 12(1): 11-6. PubMed PMID: 18923761. Pubmed Central PMCID: PMC2532969. Epub 2008/10/17. eng.
14. Opasich C, Cazzola M, Scelsi L, De Feo S, Bosimini E, Lagioia R, et al. Blunted erythropoietin production and defective iron supply for erythropoiesis as major causes of anaemia in patients with chronic heart failure. *European heart journal*. 2005 Nov; 26(21): 2232-7. PubMed PMID: 15987710. Epub 2005/07/01. eng.
15. Guidi GC, Lechi Santonastaso C. Advancements in anemias related to chronic conditions. *Clin Chem Lab Med.*, 2010 Sep; 48(9): 1217-26. PubMed PMID: 20618092. Epub 2010/07/14. eng.
16. Kes P, Basic-Jukic N, Juric I, Basic-Kes V. [The cardiorenal syndrome and erythropoietin]. *Acta Med Croatica*. 2008; 62 Suppl 1: 21-31. PubMed PMID: 18578329. Epub 2008/06/27. Srcano-bubrezní sindrom i eritropoetin. hrv.
17. Kotchen TA. Harrison's Principles of Internal Medicine. 18th ed. Longo DL FA, Kasper DL, Hauser SL, Jansen JL, et al. editors, editor. New York: MacGraw Hill 2012. 18 p.
18. Aswini Dutt.R RC, Niranjana murthy.H.L, Satish Kumar NS, Shankar Bhat.K. Do hypertensives have tendency for lesser hemoglobin concentration? *National Journal Of Basic Medical Sciences.*, 2010 Oct-Dec; 1.
19. Adlbrecht C, Kommata S, Hulsmann M, Szekeres T, Bieglmayer C, Strunk G, et al. Chronic heart failure leads to an expanded plasma volume and pseudoanaemia, but does not lead to a reduction in the body's red cell volume. *European heart journal*. 2008 Oct; 29(19):2343-50. PubMed PMID: 18701467. Epub 2008/08/15. eng.