Study of eosinophil to monocyte ratio and its association with arterial blood pressure in hypertensives

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ABSTRACT

Background: Total white blood cell (WBC) count is an effective marker of chronic inflammation. Granulocyte, specifically eosinophil play significant role in endothelial dysfunction, vasoconstriction, inflammation, and thrombosis. Along with eosinophil, previous studies reported increased activity of monocytes in hypertension (HT). Nowadays, in a medical research, we are using mathematical calculations, which is more accurate in this study. Aim and Objectives: This study was conducted to find out association of total WBC count and eosinophil to monocyte ratio (EMR) in hypertensives and normotensives. Materials and Methods: In study group, 50 hypertensives aged 40–60 years, males and females with the history of previously diagnosed HT >1 year duration, or taking antihypertensive medications with blood pressure (BP) Systolic BP (SBP) >140 mm Hg, Diastolic BP >90 mmHg were taken. In control group, 50 normotensives aged 40–60, males and females with SBP <140 mmHg, DBP <90 mmHg were taken, who were matching socioeconomically with study group. BP was measured, and venous blood samples were collected with proper aseptic precautions. Results: Study shows significantly (P < 0.05) high total WBC count, Eosinophil count and Monocyte count in hypertensives. EMR is also high but it is statistically not significant. Conclusion: High total WBC count, eosinophil count and monocyte count predicts risk of adverse cardiovascular events but change in EMR is not associated with HT.

KEY WORDS: Eosinophil; Monocyte; Eosinophil to Monocyte Ratio; Hypertension

INTRODUCTION

Due to modernization, there are significant changes in dietary habits, lifestyle, physical activity, etc., in our society. All these factors are responsible for increased prevalence of hypertension (HT) in populations. HT exerts a substantial public health burden on cardiovascular health status and healthcare systems in India. Long-term HT can lead to adverse cardiovascular events, including coronary heart disease and stroke.[1-3] Total white blood cell (WBC) count is a simple but effective marker of chronic inflammation, it may also contribute to increasing microvascular capillary resistance, initiation of platelet aggregation, increased catecholamine levels, and there is considerable evidence of a link between inflammation and HT.[4-6]

Eosinophil, a granulocyte, plays an important role in endothelial dysfunction, vasoconstriction, inflammation, and thrombosis. It stimulates the activation and aggregation of platelets. Moreover, they ease the formation of thrombosis through inhibition of thrombomodulin.[7-10] Eosinophil have powerful vasoconstrictor and procoagulant effect; hence, we tried to find out the relationship between eosinophil count and HT.[11] Earlier studies indicated that monocyte activation...
is increased in HT.[12] Monocytes from patients with essential HT show elevated secretion patterns of pro-inflammatory cytokines, an increased expression of adhesion molecules, and an increased adhesion to vascular endothelial cells.[13]

Individual blood counts have more tendency to fluctuate. Nowadays, in a medical research, we are using mathematical calculations of different types of blood cells and their relationship with a disease. Since HT is one of cause behind major cardiovascular events, there is a need to develop new investigation strategies, which can predict cardiovascular risk early. Hence, we investigated the relationship of total WBC count and eosinophil to monocyte ratio (EMR) with HT, as the ratio of granulocyte and agranulocyte is more reliable than individual blood count.[14-16]

MATERIAL AND METHODS

A study was approved by the Institutional Ethical Committee of Government Medical College of Surat, and written informed consent was taken from each participant before the study. This study was conducted in study and control group. In study group, 50 hypertensive patients aged 40–60 years, males and females with the history of previously diagnosed HT >1 year duration, or taking antihypertensive medications with blood pressure (BP) systolic BP (SBP) >140 mmHg, diastolic BP (DBP) >90 mmHg were taken from out-patient clinics of medicine department, New Civil Hospital Surat. In control group, 50 normotensives aged 40–60, males and females with SBP <140 mmHg, DBP <90 mmHg were taken, who were matching socioeconomically with study group. Any systemic disease other than HT, such as diabetes mellitus, tuberculosis, rheumatoid arthritis, osteoarthritis, and systemic lupus erythematosus, patients using medical treatment affecting WBC count such as hematopoietic disorders, treatment with chemotherapy, any present illness, acute infection in past 6 months (typhoid, malaria, pharyngitis, etc.), and acute coronary syndrome history of using glucocorticoid therapy within past 3 months, history of heart failure, chronic renal disease, hepatic disease, cerebrovascular disease were excluded from the study.[17]

The physiological parameters such as height, weight, pulse rate, and BP along with detailed history were taken. BP was measured in sitting position after 5 min rest through auscultatory method using sphygmomanometer (mercury manometer) in both the arms. Higher of the two readings was taken and if the SBP and DBP were in different categories; the higher of 2 was classified. General and systemic examinations were done thoroughly. 3 mL of venous blood samples were collected in ethylenediaminetetraacetic acid vacutte from median cubital vein under aseptic precautions and analysed by ABX MICRO6 60 hematology-autoanalyzer in hematology laboratory, Department of Pathology, New Civil Hospital Surat.

Pulse pressure (PP), mean arterial pressure (MAP), and EMR were calculated. The data between two groups were compared using unpaired t-test. P < 0.05 was considered as statistically significant and P < 0.01 considered as statistically highly significant. In the study group, Correlation between BP and total WBC count, eosinophil, monocyte, and EMR were found by calculating Pearson’s correlation factor (r). All statistical analysis were done in SPSS software version 17.

RESULTS

Table 1 shows the comparison of age, height, weight, BMI between two groups were not statistically significant (P > 0.05). Both groups were comparable to each other. Table 2 shows the SBP, DBP, PP, and MAP in study and control groups. Table 3 shows that total WBC count, eosinophil count, and monocyte count are high in study group (hypertensives) as compare to control group (normotensives) which is statistically significant (P < 0.05), whereas EMR is statistically not significant (P > 0.05) even its high in study group. Table 4 shows statistically not significant (P > 0.05) positive correlation between WBC count, Eosinophil count, Monocyte count and SBP, DBP, PP, MAP. In addition, EMR also shows statistically not significant (P > 0.05) positive

### Table 1: Comparison of age, height and weight between study and control groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study group Mean±SD</th>
<th>Control group Mean±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>48.88±7.18</td>
<td>47.84±7.04</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Height</td>
<td>154.16±7.64</td>
<td>153.8±8.77</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Weight</td>
<td>67.78±10.58</td>
<td>65.34±6.72</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

### Table 2: SBP, DBP, PP, MAP in study and control groups

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study group Mean±SD</th>
<th>Control group Mean±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>146.24±6.53</td>
<td>119.16±3.43</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>DBP</td>
<td>91.56±1.95</td>
<td>78.4±2.09</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PP</td>
<td>54.68±5.21</td>
<td>40.76±2.61</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>MAP</td>
<td>109.79±3.27</td>
<td>91.98±2.31</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PP: Pulse pressure; MAP: Mean arterial pressure

### Table 3: Total WBC count, eosinophil count, monocyte count and EMR in study and control group

<table>
<thead>
<tr>
<th>Variables</th>
<th>Study group Mean±SD</th>
<th>Control group Mean±SD</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total WBC count</td>
<td>10070±1933.20</td>
<td>6558±1255.70</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Eosinophil count</td>
<td>484.38±634.01</td>
<td>226.3±171.98</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Monocyte count</td>
<td>289.44±155.75</td>
<td>181.28±124.63</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EMR</td>
<td>1.74±1.63</td>
<td>1.45±0.96</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

EMR: Eosinophil-monocyte ratio, WBC: White blood cell
correlation with SBP, DBP and MAP, whereas statistically not significant \((P > 0.05)\) negative correlation with PP.

**DISCUSSION**

This study conducted by taking 50 hypertensive patients as a study group and 50 normotensive subjects as a control group. Both groups were comparable to each other as age; height and weight are not statistically significant [Table 1]. As shown in Table 2, SBP, DBP, PP and MAP were significantly higher in study group as compare to control group. Study by Belen et al. reported resistant hypertensive and controlled hypertensive groups have higher SBP and DBP as compare to normotensives.\(^{18}\) Our study found significantly high total WBC count, eosinophil count, and monocyte count in hypertensives but increase in EMR is not significant [Table 3]. Furthermore, we found positive correlation between SBP, DBP, and MAP with total WBC count, Eosinophil count, Monocyte count and EMR [Table 4].

Our study shows high total WBC count [Table 3] which in accordance with the study of Shankar et al. who had shown that elevated WBC count is associated with incident HT, independent of smoking, and other traditional cardiovascular risk factors.\(^{19}\) Similarly, Pusuroglu et al. also reported increased leukocyte count in hypertensives and suggested leukocytes may increase blood viscosity, and thus affect peripheral vascular resistance, which may also be a factor in the development of HT.\(^{20}\) Along with increases total WBC count, we found Eosinophil count and Monocyte count were significantly higher in study group. Study by Kuzeytemiz et al. found higher eosinophil count in patients of nondipper HT, and they concluded the measurement of eosinophil count may be used to indicate increased risk of HT-related adverse cardiovascular events as it plays an important role in vasoconstriction and thrombosis.\(^{11}\) Eosinophils help platelets to adhere injured vessel wall and release immunosuppressive cytokines like Interleukin (IL)-10, IL-4, and IL-13, which suggested to modulate the inflammatory response in cardiovascular disease.\(^{15}\) A study by Salvado et al. found higher monocyte count in patients with high MAP and its positive association with SBP and PP. However, we found positive correlation between SBP, DBP, and MAP with total WBC count, Eosinophil count, Monocyte count and EMR [Table 4]. Another study by Zhao et al. observed monocyte activation and monocyte mediated inflammation in HT. Monocyte chemoattractant protein-1/cysteine-cysteine chemokine receptor 2 pathway appears to be involved in the increased inflammatory response observed in HT.\(^{21,22}\) This study shows higher EMR in study group compare to control group but it is not statistically significant [Table 3]. In contrast to our study, Deng et al. have shown lower EMR on admission was associated with higher 1-month and long-term mortality in patients with ST segment elevation myocardial infarction.\(^{15}\)

Although, our study is the first to find relation of EMR with HT but still it should be investigated on a wider scale.

**CONCLUSION**

Eosinophil and monocyte, both play key role in chronic inflammatory conditions such as HT. Eosinophil plays an important role in vasoconstriction and thrombosis. In addition, monocyte activation and release of monocyte chemoattractant protein-1 induces inflammatory changes in HT. Hence, EMR is a simple, useful, and inexpensive marker in early assessment of cardiovascular risk in hypertensive patients.

**REFERENCES**

9. Rohrbach MS, Wheatley CL, Slifman NR, Gleich GJ.

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