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Cardiovascular

COMPARISON OF BLOOD CELL INDICES IN NSTE-ACS AND STE-ACS

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ABSTRACT **INTRODUCTION** Blood cells play an essential role in the formation, extension and propagation of atheromatous plaques. Platelets, inflammatory cells (neutrophils and monocytes), red blood cell indices indicating the thickness of blood are involved in the pathogenesis of ACS. ACS encompasses three levels of pathophysiology. STEMI is caused due to complete blockage of the involved artery; NSTEMI is due to partial blockage. However, the role of inflammatory markers is not exact in causing NSTEMI and STEMI. The current study is an attempt to determine the differences in blood indices between STEMI and NSTEMI.

RESULTS The analysis has returned 93 cases of ACS presenting to the ED. Out of them, 74 patients had STEMI at the time of presentation, and the rest had NSTEMI. No statistically significant difference in the WBC count between the two groups. Patients with STEMI had higher neutrophil counts (average-73.13%) compared to NSTEMI. Eosinophil count and neutrophil/lymphocyte rate are important parameters showing difference. there were no significant differences in terms of RBC, Red cell distribution width, Platelet count and platelet distribution width. Packed cell volume had a significant difference between the two groups.

CONCLUSION The current study concludes that blood indices indicating inflammation like neutrophils, eosinophils, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio are generally raised in patients with ACS. These parameters can be useful in predicting propensity for increased clot formation and thus more severe disease.

KEYWORDS : Blood cell indices, NST-ACS, ST-ACS, neutrophil-lymphocyte ratio

INTRODUCTION:

Blood cells play an essential role in the formation, extension and propagation of atheromatous plaques. Platelets, inflammatory cells (neutrophils and monocytes), red blood cell indices indicating the thickness of blood are involved in the pathogenesis of ACS. Neutrophils might indicate the extent of the inflammatory response and host reaction after plaque rupture [1,3]. Adherence of eosinophils to the clot will help in its extension [4]. Red blood cell distribution width determines the viscosity of the blood and thus spells adverse outcomes and risk of death in patients of acute heart failure [8,9]. Following rupture of an atheromatous plaque, platelets are involved in the formation and propagation of a clot [7,8]. Similarly, platelet indices also can be used as prognostic markers as they are involved in the extension of the clot [7,8].

ACS encompasses three levels of pathophysiology. STEMI is caused due to complete blockage of the involved artery; NSTEMI is due to partial blockage. The chronic inflammatory response has been proven in causing plaque instability and thus rupture. However, the role of inflammatory markers is not exact in causing NSTEMI and STEMI. The current study is an attempt to determine the differences in blood indices between STEMI and NSTEMI.

OBJECTIVES:

To Determine differences in blood cell indices between NSTE-ACS and STE-ACS.

METHODOLOGY (MATERIALS & METHODS):

This study is a time-bound retrospective observational study in the emergency room of a tertiary care hospital. Patients with an admitting diagnosis of ACS during the period Jan 2019 to DEC 2019 were included in the study.

Descriptive statistics were used for all continuous variables. Mean, and the standard deviation is calculated for discrete variables. Chi-square test or student t-test is used as a test of significance.

RESULTS-

The analysis has returned 93 cases of ACS presenting to the ED. Out of them, 74 patients had STEMI at the time of presentation, and the rest had NSTEMI. No statistically significant difference in the WBC count between the two groups. Patients with STEMI had higher neutrophil counts (average-73.13%) compared to NSTEMI group (60.64%) with high statistical significance (P-value - 0.0011). Eosinophils is another parameter which had a very high statistically significant difference. Patients with STEMI had significantly lower levels of eosinophils (1.35 with an SD of 0.18 in STEMI vs 3.08 with an SD of 0.69). Neutrophil/lymphocyte ratio is another parameter which has shown a significant difference.

Similarly, lymphocyte counts were significantly low in the STEMI group (18.52) compared to NSTEMI group (30.79). Other lineages of white blood cells did not show a significant difference. Likewise, there were no significant differences in terms of RBC, Red cell distribution width, Platelet count and platelet distribution width. Packed cell volume had a significant difference between the two groups. STEMI group had a mean PCV of 41.11 and, NSTEMI had a mean value of 36.12.

Platelet-lymphocyte ratio is another highly significant parameter between the two groups with a P-value of 0.006.

Table1. Parameters showing a significant difference between NSTEMI and STEMI

s.no	Parameter	NSTEMI (mean±SD)	STEMI (mean±SD)
1	Total WBC count	12.34±2.8	12.93±3.77
2	Neutrophils	60.65±13.66	73.13±14.15
3	Lymphocytes	30.79±10.89	18.52±11.4
4	Neutrophil-lymphocyte ratio	2.38±1.31	6.60±6.03
5	Eosinophils	3.08±2.94	1.35±1.61
6	Packed cell volume	36.07±11.8	41.11±6.55
7	Platelet count	284.96±112.61	263.4±112.56
8	Platelet-lymphocyte ratio	0.102±0.059	0.232±0.212

DISCUSSION

STEMI and NSTEMI are distinct types of myocardial infarction. STEMI has a complete arterial occlusion, while NSTEMI has a partial occlusion. Inflammatory processes appear to be more pronounced in STEMI compared to NSTEMI. Literature indicates that inflammation plays a significant role in plaque instability, plaque rupture, clot formation and extension[1]. The current study has found high neutrophils, low lymphocytes, high levels of neutrophil-lymphocyte ratio, more significant packed cell volumes and lower eosinophil counts in ST-elevation MI. Total counts, red blood cell counts, and platelet indices did not show any statistically significant differences between the two groups. Leukocytosis has been noted uniformly in both the groups, although it was a bit higher in STEMI (mean-12.93) than NSTEMI (12.34). No statistically significant difference was found. In a study by sabatine et al., leukocytosis was found to be an indicator of increased mortality and more advanced coronary artery

disease. The current study has noticed high levels of neutrophilia, low lymphocyte count and high neutrophil-lymphocyte ratio in STEMI patients compared to NSTEMI patients, which are highly significant statistically. Earlier studies have extensively studied the role of these parameters in the causation of coronary artery disease [2,3,4,5]. The current study proves that and further differentiates between STEMI and NSTEMI. Neutrophilia can be attributed to both as causing more severe forms of myocardial infarction and also as reactionary to the stress of more severe disease. The neutrophil-leukocyte ratio is a more significant predictor of severity of disease rather than neutrophils or lymphocytes individually. Hence patients with STEMI will have higher NLR than those with NSTEMI. A fact which the current study proves [4,5].

Eosinophils are another lineage of cells which reflect the extent of thrombosis. Earlier studies have documented the role of hypereosinophilia as a predisposing factor for thrombosis. After clot formation, eosinophils will adhere to the clot leading to peripheral eosinophilia. Significantly lower eosinophil count reflected in STEMI patients compared to NSTEMI patients in this study can be attributed to more pronounced clot formation in the STEMI group [6].

Although RBC count, mean corpuscular volume and red cell distribution width are slightly higher in the STEMI group, it is not found to be statistically significant. Packed cell volume showed a more significant difference, being higher in the STEMI group. Studies have shown that raised PCV will lead to a two-fold increase in the risk of cardiovascular disease, peripheral vascular disease and also cerebrovascular disease. The current study shows that raised hematocrit will lead to more severe disease, reflected by its significant raise in patients with STEMI [1,6].

None of the platelet indices [5,6,7] showed any difference between the groups, although platelet counts were slightly higher in NSTEMI group. Platelet counts, mean platelet volume were within normal limits in both the groups. Platelet distribution width was on the higher range in both groups, although marginally higher in NSTEMI (16.9 in NSTEMI vs 16.7 in STEMI). Studies have shown that raised platelet indices are involved in increased cardiovascular mortality. It appears that platelets are involved in the initiation of the clotting process, but does not seem to be involved in clot extension. They are also involved in maintaining the inflammatory response at the site of clot formation by releasing inflammatory mediators.

Platelet-lymphocyte ratio [5] is another highly significant parameter between the two groups. STEMI group had higher levels of PLR with an average of 0.22 compared to 0.102 in NSTEMI group. Platelet-lymphocyte ratio describes a systemic inflammatory response. Earlier studies have that high PLR is associated with recurrent infarction and heart failure. The same findings have been proven in this study. STEMI patients generally appear to have higher levels of systemic inflammation, partly owing to more extensive clots.

Based on the findings of the study, STEMI patients have higher levels of inflammatory markers compared to NSTEMI. This reflects more extensive clot and a significant amount of tissue damage, leading to increased inflammatory markers in the same.

CONCLUSIONS

The current study concludes that blood indices indicating inflammation like neutrophils, eosinophils, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio are generally raised in patients with ACS. Raise of these markers are pronounced in patients with STEMI compared with those of NSTEMI. This could indicate that patients with STEMI have extensive clotting. High hematocrit found in patients with STEMI can indicate its role in forming advanced clots.

Hence these parameters can be utilized in detecting patients prone to have increased clotting and thus more severe disease.

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