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Platelets large cells ratio (P-LCR)-A new platelets indices use as a risk assessment prognostic tool for post myocardial infarction patients

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ABSTRACT

OBJECTIVES & AIMS: To evaluate and identify new prognostic markers suggested in recent studies for coronary heart disease, raised platelets large cells ratio (P-LCR)) has been found to be associated with poor prognosis after recent myocardial infarction. To evaluate the relationship between raised P-LCR and mortality/ morbidity after the initial attack of myocardial infarction (MI). Increased platelets-LCR has been associated with adverse outcomes. We studied the association between raised P-LCR during hospital course with clinical outcomes survival index of patients with acute myocardial infarction (AMI).

MATERIAL & METHODS: Blood was collected in a sterile EDTA containing tube and processed following our established ISO certified hospital based laboratory protocol .A complete blood counting including HB%,PCV, Red cell indices ,platelet count, total white cell count and PDW was done by Automated blood cell counter. Level of Troponin I done by automated mini vidas bioanalyser.

CONCLUSION: We found significant correlation in patients with post MI along with high P-LCR. P-LCR is an inexpensive cost effective and easily available laboratory test,high P-LCR with high troponin I for post MI have poor outcome of patients' it could be used for mortality and morbidity risk assessment and follow up the patients after MI .We found that high P-LCR of raised troponin I pt. shows poor prognosis.Confirmation of MI done by troponin I level of every patients.

Keywords: myocardial infarction, platelets large cells ratio.

INTRODUCTION

Material & Methods

Study area and design- This present study was conducted at the Dayanand Medical College and associated hospital, ludhiana. The study was designed as an observational retrograde with prospective hospital based study over a period of time from January to December 2014.

Ethical consideration- Blood was collected in a sterile EDTA tube and plaint tube and processed following our established laboratory protocol then generate the report of each patient. Take informed consent was obtained from all study participant for use of your blood sample for medical research after doing physician request investigating and generate the report.

Patient's selection criteria-The study target all patients on the basis of clinical signs, symptoms and ECG ST elevation with high troponin I level, history by patients, attendants and data records. We included both emergency and IPD patients with all age groups, male and female both gender for study. Sample size is 100 patients.

Laboratory investigations Blood was collected in a sterile EDTA containing tube and processed following our established laboratory protocol .A complete blood counting including HB%, PCV, Red cell indices, platelet count and total white cell count

and differential was done by automated blood cell counter and peripheral blood smear examination. The cell count indices including RBC, WBC count with differential along with morphological changes further confirmed by manual oil immersion smear study method.

Materials:

Purple vacutainer tube or capillary collector (EDTA) ethylenediaminetetraacetate,Slides and blue capillary tube,Needle or lancet,Vacutainer holder,Alcohol swab,Cotton balls,Absorbent materials,Slide case and heamatological cell counter. and second sample in clot activator tube for serum troponin I by automated bioanalyser.

Procedure:

Specimen is collected into EDTA (purple) vacutainer. (5 or 7ml volume)

Then the run the sample in hematological cell counter and generate P-LCR data.

The P-LCR median was 12 fl with a reference range of 02%-20% for the 5th-95th percentiles, with a confidence interval of 95%. Normal range 02-20fL. Serological troponin I is done by minividas methods

OBERVATION & DISCUSSION

P-LCR	Grade	Survival outcome of patients	Serological troponin I	Sample size N=100
>12 to <14.1 fL	Mild	Good	>100 TO 1000 ng/L	59
>14.2 to <15.2fL	Moderate	Average	>1000 TO 5000 ng/L	28
>15.2 to <16 fL	Sever	Poor	>5000 TO 10000ng/L	09
>16Fl	Marked	Worst	>10000 ng/L	04

Result:

Univariate analysis showed that there were significant associations of high PDW values with, the acute coronary artery disease, mild to marked type toxicity these various morphological changes cause the raised platelets distribution width use as a prognostic tool for survival index outcome of patients. Kruskal-Wallis tests revealed an association of raised P-LCR values with severity survival index patients: $p<0.0001$, survival prognostic index of patients with higher P-LCR values had worst prognoses

Conclusion:

Our study is, to the best of our knowledge, the first to demonstrate an association between P-LCR and serum troponin I risk of incident MI in a general population. The association was consistent when P-LCR was modeled both as continuous and categorical variables, and the risk of recurrent MI by P-LCR correlation with troponin I pattern.Survival of patients is easily found with PLCR and troponin I correlation .There are only a few previous reports on the relation between PDW and troponin I for post MI patients from general populations. A strong association between higher P-LCR and high level of troponin I with post MI poor outcome high mortality was found is our study.[13–14]The risk of MI death increased by 20% for a 1-SD increment of P-LCR

Volume 1, Issue 4; November-December 2018; Page No. 181-184

[14]and was more than 2-3fold higher among participants in the highest quintile compared with the lowest.[13], the risk of post MI mortility PLCR with normal range%.[19] In contrast, P-LCR is associated with Recurrent MI or myocardial mortality in this study. Greater power to detect a significant association between P-LCR and risk of recurrent MI in our study may be the main reason for the apparent discrepant relationship between P-LCR and serum troponin I for post MI. Because P-LCR is a statistical concept, it can be assumed that P-LCR is a marker of other underlying biological mechanisms.

P-LCR is suggested to be a biomarker reflecting a proinflammatory condition. Oxidative stress and inflammation increase P-LCR by impairing iron metabolism, The stronger association between P-LCR and serum troponin I for post MI in our study supports the suggestion that P-LCR reflects inflammation. Others have also speculated that the biological link between P-LCR and post MI mortality may be mediated by systemic inflammation.It has been reported that increased post MI mortality by P-LCR is confined to those with MI .

Platelet indices,like Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) are well studied markers to prognosticate patients. It was hypothesized that in acute coronary heart diseae, there is increased platelet swelling and pseudopodia formation which causes an increase in the Mean Platelet Volume and Platelet Distribution Width. Among the two indices Platelet Distribution Width is found to be a more specific marker for the activation of platelets. White blood cells are a marker of inflammation and it is also well studied in patients with acute coronary syndrome which causes a rise in the inflammatory markers. White blood cell count, a marker of inflammatory response and platelet distribution width, a marker of reactivity of platelets have been studied to have unfavourable outcomes in patients with ST elevation myocardial infarction. The results of our study has shown significant association between platelet distribution width and white blood cell count with ST segment resolution in patients with STEMI thrombolysed with streptokinase. These factors can be used as simple markers for failure of thrombolysis to suggest an alternative and aggressive management protocol for these patients who require further studies in this context.

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Volume 1, Issue 4; November-December 2018; Page No. 181-184

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Volume 1, Issue 4; November-December 2018; Page No. 181-184