



Raised Platelets Distribution Width as a Risk Assessment Prognostic Tool For Post Myocardial Infarction Patients

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Abstract

Objectives & Aims: *The evaluation and identify new prognostic markers suggested in recent studies for coronary heart disease, raised platelets distribution width (PDW) has been found to be associated with poor prognosis after recent myocardial infarction. Evaluate the relationship between raised PDW and mortality morbidity after the initial attack of myocardial infarction (MI). Increased platelets distribution width (PDW) has been associated with adverse outcomes. We studied the association between raised PDW during hospital course with clinical outcomes survival index of patients with after 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 find significantly correlation in patients with post MI along with high PDW. PDW is an inexpensive cost effective and easily available laboratory test, high PDW with high troponin I for post MI have poor outcome of patients' it could be used for mortality with morbidity risk assessment and follow up the patients after MI. we find that high PDW of raised troponin I pt. shows poor prognosis. confirmation of MI done by troponin I level of every patients.*

Keyword: *myocardial infarction, platelets distribution width.*

Material & Methods

Study area and design- This present study was conducted at the Advanced institute of medical sciences and research Bhopal and associated referral hospital Bhopal mp. The study was designed as a observational retrograde with prospective hospital based study over a period of time from 2016.

Ethical consideration- Blood was collected in a sterile EDTA tube and plain 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 attainer. We include 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 all cell count indices including RBC, WBC count with differential along with morphological changes further confirmed by manual oil immersion smear study method. Peripheral smears study was done with field A and B stain and leishman stain.

Red Cells Distribution Width and Peripheral Smear

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 hematological 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 PDW data.

The PDW median was 13.3%, with a reference range of 10.0%-17.9% for the 5th-95th percentiles, with a confidence interval of 95%. Normal range 9-13fL.

Serological troponin I is done by minividas methods

Obervation & Discussion

RDW-CV	Prognosis	Survival outcome of patients	Serological troponin I	Sample size N=100
>13 to <14 fL	Mild	Good	>100 TO 1000 ng/L	66
>14 to <15fL	Moderate	Average	>1000 TO 5000 ng/L	24
>15 to <16 fL	Sever	Poor	>5000 TO 10000ng/L	08
>16fL	Marked	Worst	>10000 ng/L	02

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 PDW values with severity survival index patients: $p < 0.0001$, survival prognostic index of patients with higher PDW values had poorer worst prognoses than those with normal RDW values (Wilcoxon test: $p = 0.002$). multivariate analysis showed higher PDW is a significant prognostic factor ($p = 0.040$).

Conclusionion

Our study is, to the best of our knowledge, the first to demonstrate an association between PDW and serum troponin I risk of incident MI in a general population. The association was consistent when PDW was modeled both as continuous and categorical variables, and the risk of MI by RDW correlation with troponin i pattern. The presence of anemia did not affect the risk estimates. Survival of patients is easily find with PDW 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 PDW and high level of troponin I with post MI poor outcome high mortality was found is our study.¹³⁻¹⁴The risk of MI death increased by 22% for a 1-SD increment of PDW (HR 1.22; 95% CI, 1.14 to 1.31)¹⁴and was more than 2-fold higher among participants in the highest quintile compared with the lowest.¹³, the risk of post MI mortality events increased 39% among patients with P DW of 16% to 17% (HR 1.39; 95% CI, 1.24 to 1.57) compared with patients with RDW with normal range%.¹⁹ In contrast, PDW is associated with MI (HR 1.05; 95% CI, 0.65 to 1.68) or myocardial mortality (HR 1.09; 95% CI, 0.96 to 1.23) in this study.

Greater power to detect a significant association between PDW and risk of MI in our study may be

the main reason for the apparent discrepant relationship between PDW and serum troponin I for post MI .

The mechanism for the observed association between PDW and post MI morbidity and mortality now a day settled. Because PDW is a statistical concept, it can be assumed that PDW is a marker of other underlying biological mechanisms.

PDW is suggested to be a biomarker reflecting a proinflammatory condition. Oxidative stress and inflammation increase PDW by impairing iron metabolism, The stronger association between PDW and serum troponin I for post MI in our study supports the suggestion that PDW reflects inflammation. Others have also speculated that the biological link between PDW and post MI mortality may be mediated by systemic inflammation. It has been reported that increased post MI mortality by PDW is confined to those with anemia. we included hemoglobin in our multivariable model and performed analyses in which anemic participants were excluded. The risk estimates for MI by PDW in our study were not affected by adjustment for hemoglobin or by excluding participants with anemia. This demonstrates that anemia does not explain the strong association between PDW and MI. Furthermore, results from association between extremely high PDW (>16fl).

Platelet distribution width (PDW) Platelet distribution width shows the heterogeneity in the size of platelets and is derived from the platelet indices from an automated analyzer. The reference range in a study done by Mariela Graniro Farias was derived as 13.3 % as median with a reference range of 10% to 17.9%. Platelet distribution width is a marker of platelet reactivity^[30]. When laser technology is used in an analyzer it primarily detects the cross diameter of the cell to derive its volume. Machines using impedance principle focus on the vertical diameter of the cell to assess the cell's size. Whatever may be the technique used, activated platelets will be larger and is not dependent on the technique of analysis^[31].

Role of platelets in coronary artery heart disease Whenever there is a stressful situation, the platelets that are produced are larger in size and they possess a very high potential for thrombus formation since they produce more thromboxane B₂ . During situations of platelet activation both MPV and PDW increase. This change is hypothesized due to the change in platelet shape from that of discoid to spherical shape to attain larger surface area. These changes can be analyzed by the hematology analyzers that work on the impedance principle discussed already. Platelet activation is a very essential step in the production and propagation of the process of atherothrombosis^[32]. The platelet parameters like MPV and PDW are independent risk parameters in Myocardial Infarction (MI) and stroke indicating worse clinical course and mortality^[33]. ST-segment Elevation Myocardial Infarction (STEMI) and failure of thrombolysis is influenced by high PDW values. Also it has been studied that PDW is higher in patients with STEMI rather than stable Coronary Artery Disease (CAD). Rather than just association they also influence the success of thrombolysis in STEMI patients^[34]. There are several studies which analysed the risk of Platelet Distribution Width (PDW) in acute coronary syndrome like ST Elevation Myocardial Infarction. Varasteh-ravan et al.^[30] studied the relationship of platelet distribution width in patients with acute STEMI thrombolysed with streptokinase and found that patients with higher platelet distribution width had more risk of thrombolysis failure measured by ST segment resolution. PDW can be used as an independent marker of risk of thrombolysis failure and short term mortality in patients with STEMI. 4.4. White blood cells Human white blood cell count is normally 4000 to 11000 cells per microlitre. The most predominant of Conclusion 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 disease, 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 which require further studies in this context.

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