



## Raised Red Cell 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 red blood cell distribution width (RDW) has been found to be associated with poor prognosis after resent myocardial infarction. Evaluate the relationship between raised RDW and mortality morbidity after the initial attack of myocardial infarction (MI). Increased red blood cell distribution width (RDW) has been associated with adverse outcomes. We studied the association between raised RDW 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 RDW 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 RDW. RDW is an inexpensive cost effective and easily available laboratory test, high RDW 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 RDW of raised troponin I pt. shows poor prognosis. confirmation of mi done by troponin I level of every patients.*

**Keyword:** *myocardial infarction, Red cell distribution width.*

### Material & Methods

**Study area and design-** This present study was conducted at the cure well hospital pvt Ltd and associated referral hospital Indore mp. The study was designed as a observational retrograde with prospective hospital based study over a period of time from 2016 to 2017 years.

**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 attainder. 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.

### Observation & Discussion

| RDW-CV       | Prognosis | Survival outcome of patients | Serological troponin I | Sample size<br>N=100 |
|--------------|-----------|------------------------------|------------------------|----------------------|
| >25 to <30 % | Mild      | Good                         | >100 TO 1000 ng/L      | 67                   |
| >30 to <36%  | Moderate  | Average                      | >1000 TO 5000 ng/L     | 23                   |
| >36 to <40 % | Sever     | Poor                         | >5000 TO 10000ng/L     | 07                   |
| >40%         | Marked    | Worst                        | >10000 ng/L            | 03                   |

### Result

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

### Procedure

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

Then the run the sample in hematological cell counter and generate RDW data.

Red cell distribution width (RDW) is a red blood cell parameter that measures variability of red cell volume/size (anisocytosis). Depending on the types of hematology analyzer instruments, RDW can be reported statistically as coefficient of variation (CV) and/or standard deviation (SD), RDW-CV and/or RDW-SD, respectively.

RDW-SD (express in fL) is an actual measurement of the width of the RBC size distribution histogram and is measured by calculating the width (in fL) at the 18-20% height level of the RBC size distribution histogram.

RDW-CV (express in %) is calculated from standard deviation and MCV

RDW-CV (%) = 1 standard deviation of RBC volume/MCV x 100%.

- RDW-SD 39-46 fL[1]
- RDW-CV 11.6-14.6% in adult[2]

.Serological troponin I is done by minividas methods

values had poorer worst prognoses than those with normal RDW values (Wilcoxon test:  $p = 0.002$ ). multivariate analysis showed higher RDW is a significant prognostic factor ( $p = 0.040$ ).

### Conclusion

Our study is, to the best of our knowledge, the first to demonstrate an association between RDW and serum troponin I risk of incident MI in a general population. The association was consistent when RDW 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 RDW and troponin I correlation.

There are only a few previous reports on the relation between RDW and troponin I for post MI patients from general populations. A strong association between higher RDW and high level of troponin I with post MI poor outcome high mortality was found in our study.<sup>13-14</sup> The risk of MI death increased by 22% for a 1-SD increment of RDW (HR 1.22; 95% CI, 1.14 to 1.31)<sup>14</sup> and was more than 2-fold higher among participants in the highest quintile compared with the lowest.<sup>13</sup> , the risk of post MI mortality events increased 39% among patients with RDW of 16% to 17% (HR 1.39; 95% CI, 1.24 to 1.57) compared with patients with RDW with normal range%.<sup>19</sup> In contrast, RDW 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 RDW and risk of MI in our study may be the main reason for the apparent discrepant relationship between RDW and serum troponin I for post MI .

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

RDW is suggested to be a biomarker reflecting a proinflammatory condition. Oxidative stress and inflammation increase RDW by impairing iron metabolism, reducing red cell life span, and modulating the response to erythropoietin by the bone marrow.<sup>21-22</sup> The stronger association between RDW and serum troponin I for post MI in our study supports the suggestion that RDW reflects inflammation. Others have also speculated that the biological link between RDW and post MI mortality may be mediated by systemic inflammation.

It has been reported that increased post MI mortality by RDW is confined to those with anemia.<sup>20</sup> To explore the impact of anemia on the relationship between RDW and risk of MI in our study, we included hemoglobin in our multivariable model and performed analyses in which anemic participants were excluded. The risk estimates for MI by RDW 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 RDW and MI. Furthermore, results from association between extremely high RDW (>16.6%) and mortality was particularly strong in those with nonanemic macrocytosis (MCV >96 fL) or microcytosis (MCV <80 fL).<sup>35</sup> We found association between RDW and risk of MI in nonanemic participants with macrocytosis or microcytosis

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