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**Original Research Article**

# Study of the correlation between platelet parameters in the patients with coronary heart disease

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## ABSTRACT

**Background:** The incidence and development of coronary heart disease are ultimately associated with the size and function of platelet. Present study aimed to determine the clinical value of platelet distribution width as an indicator for prediction or risk stratification of coronary heart disease by retrospective analysis of particular population.
**Methods:** This retrospective study covered a total of 150 patients that were included due to a variety of indications for coronary angiography. The control group N-CHD was the patients without coronary heart disease confirmed by coronary angiography examination. The S-CHD group was patients with Stable type coronary heart disease confirmed diagnosis by coronary angiography examination. Study group 2 defined as group ACS were the patients who suffering acute coronary syndrome episode at admission and received coronary angiography and interventional treatment thereafter.
**Results:** The PDW of S-CHD and ACS were 13.85±2.68 and 13.89±1.16, respectively, and there was no significant difference, while the PDW of the N-CHD group was 12.58±2.11, and the values of the first two groups were significantly higher than those of the N-CHD group. In addition, the MPV and P-LCR of S-CHD were significantly higher than those of N-CHD group (11.14±1.17 versus 10.51±0.91 and 32.71±9.99 versus 28.41±7.69), respectively $P<0.01$; but there was no significant difference between S-CHD and ACS.
**Conclusions:** PDW, MPV, and P-LCR are the platelet volume indicators that can reflex the variability of circulation platelets; their increase was highly and positively associated with Coronary heart disease.

**Keywords:** Acute coronary syndrome, Coronary heart disease, Platelet distribution width

## INTRODUCTION

Coronary heart disease (CHD), including Stable angina, acute coronary syndromes (ACS), and acute myocardial infarction (AMI), is a global concern for public health problems, and its increasing incidence has made many developed and developing countries must pay attention to and invest abundance medical resources and public expenditure. According to the statistics of the American Heart Association, the United States each year about six hundred and ten thousand cases of new myocardial infarction and three hundred and twenty-five thousand cases of recurrence of cardiovascular events, about every second one American having coronary event and every minute a person died of adverse cardiovascular events.[1]

Fortunately, in recent years, advances in medical technology and pharmaceutical development, the direct mortality rate of AMI was decreased significantly compared with previous decades, but its complications

and succession of a series of problems is worthier of attention and seeking solutions. However, it is vital to distinguish between high-risk populations, and accurately assess and stratify their risk levels in order to initiate effective evidence-based medical treatment based on the patient's condition. And at the same time, recognizing new prognostic factors or related factors while improving risk stratification, moreover, it is critical for clinicians and scientists to further understand more about the pathophysiological mechanisms of coronary heart disease and to find effective preventive and therapeutic measures.

Platelets morphology and function in the incidence and development of coronary heart disease play a significant role; research suggests that the large platelet volume has a more hemostatic function than the smaller-size platelets volume, as an important factor of coronary heart disease and severe complications of MI.[2,3] The mean platelet volume was used as an indicator of myocardial infarction, recurrent myocardial infarction, or even stent restenosis, and in patients with AMI with elevated platelet volume, the response to thrombolytic therapy was poor and the short-term mortality was significantly higher.[4-7]

The platelet volume distribution width and the mean platelet volume are also a parameter of the complete blood count; it mainly reflects the distribution and dispersion of platelet volume in circulation, which is related to the volume, quantity, and distribution of platelets, previous clinical application was limited, and it is generally used in the diagnosis and treatment of some hematological diseases, such as thrombocytopenia.

Nevertheless, both local and international, there is little research and reports about whether may be a predictive parameter of cardiovascular disease; therefore, the purpose of this study is to investigate the relationship between PDW and coronary heart disease as well as to explore the correlation between them and the related mechanisms.

## METHODS

The change of platelet volume and function are closely connected with the incidence and development of coronary heart disease consequently, the aim of this study is based on coronary heart disease patients with platelet volume distribution width were retrospectively analyzed, discussed as an indicator reflects the heterogeneity of platelet, PDW prediction of coronary heart disease and its value in clinical application.

### *Data source*

This study was conducted in Northern Jiangsu people's Hospital from January 2015 to December 2016, which covered a total of One hundred and fifty patients that were included due to a variety of indications for coronary angiography.

### *Grouping criteria*

The control group (N-CHD group) were the patients without coronary heart disease confirmed by coronary angiographic examination. The case group 1 (S-CHD group) were patients with stable type coronary heart disease confirmed diagnosis by coronary angiography, repeated coronary angiography examination or post-CABG were excluded. The case group 2 (ACS Group): were the patients who suffering acute coronary syndrome episode (both myocardial infarction and unstable angina) at admission, and received coronary angiography and interventional treatment thereafter. In this study, all cases were excluded patients with atrial fibrillation and other Organic heart diseases (including dilated cardiomyopathy, rheumatic heart disease, valvular disease, etc.) on clinical populations.

### *Inclusion criteria*

All patients who were admitted in the Department of Cardiology at Northern Jiangsu people's Hospital during in between January 2015 to December 2016 due to a variety of indications for Coronary angiography, age ≥18 years old, <85 years old, within 24 hours after admission were conducted blood routine test, all individuals have been fluent in Chinese, and they are residents of Yangzhou city - China. Verbal consents have been acquired from all patients before the research procedures.

### *Exclusion criteria*

- Any Clinical data which was not complete (especially routine blood test, echocardiography, biochemical laboratory tests and other indicators of a serious missing).
- Patients a Prior diagnosis of hematological diseases, such as Aplastic anemia.
- Patients with severe liver dysfunction or malnutrition. 4. Chronic renal insufficiency (uremia patients), the need for hemodialysis.
- History of Malignant tumors, Chemotherapy, Radiotherapy and Organ transplantation.
- Patients nearly 1 year, had history G.I bleeding or Hemorrhagic anemia;
- Patients nearly a month, had severe infections;
- Patients nearly three months had surgery operation;
- Patients nearly half a year have received a blood transfusion or blood donors.

### *Data collection*

The results of routine blood and biochemical tests were received within 24 hours after admission. (Sample collection by the professional staff in accordance with the standard practice; and send to the laboratory in accordance with the routine procedures). Heart failure was categorized as (NYHA functional class) I-IV based on symptoms during varying activities. LVEF and LEVD

were examined by an echocardiography, results are taken in one week before or after admission either in our hospital outpatients or inpatients (if the patient had multiple results, we used the data with the date of the closest result).

***Observation parameters***

Platelet volume distribution width (PDW), mean platelet volume (MPV), platelet-large cell ratio (P-LCR), platelet count (Plt); serum creatinine, uric acid (UA), total bilirubin (TB), prealbumin, total cholesterol (TC), and left atrial diameter (LAD).

***Statistical analysis***

SPSS 16.0 statistical software was used to analyze the data, the continuous data were expressed as mean±standard deviation (x±s), and categorical data were expressed as frequency and percentage (%), also, between the two groups of data, continuous data has used the t-test to compare differences between mean. Classification data were utilized Chi-squared test, ($\chi^2$ test) or Fisher's exact probability method to compare the difference between the composition ratios. Analysis of variance (ANOVA) was used to compare the difference of mean values between multiple groups; comparison between the two variables was used linear correlation analysis; screening of independently related factors was used multivariate, stepwise logistic regression analysis. P-value<0.05 was considered statistically significant.

## RESULTS

***Case feature***

A total of 150 patients (Table 1) were enrolled in this study, including 104 patients were males and 46 patients were females with an average age over 60 years. The control group (N-CHD group) were the patients without coronary heart disease confirmed by coronary angiographic examination, with a total of 45 cases, among them 25 patients were males and 20 patients were females.

**Table 1: The clinical data of one hundred and fifty patients.**

| Parameters | Total | N-CHD group | S-CHD group | ACS group | P value |
|---|---|---|---|---|---|
| Number cases (M/F) | 150 (104/46) | 45 (25/20) | 58 (42/16) | 47 (37/10) | |
| Male (n %) | 104 (69) | 24 (16) | 42 (28) | 38 (25) | |
| Female n (%) | 46 (30.7) | 20 (13.3) | 16 (10.6) | 10 (6.7) | 0.031 |
| Age year | 63.03±9.84 | 57.98±7.78 | 62.84±9.14 | 65.19±10.95 | <0.005 |
| Hypertension n (%) | 95 (63.3) | 24 (16) | 44 (29.3) | 27 (18) | 0.016 |
| Diabetes n (%) | 33 (22) | 6 (4) | 14 (9.3) | 13 (8.7) | 0.223 |
| Hyperlipidemia n (%) | 15 (10) | 4 (2.7) | 7 (4.7) | 4 (2.6) | 0.797 |
| Smoking history n (%) | 59 (39.3) | 14 (9.3) | 22 (14.7) | 23 (15.3) | 0.208 |

**Table 2: Baseline characteristics and laboratory findings of one hundred and fifty patients.**

| Parameters | Total | N-CHD Group | S-CHD Group | ACS Group | P value |
|---|---|---|---|---|---|
| Plt (*10^9/L) | 202.43±57.36 | 213.00±50.64 | 194.80±49.23 | 199.5±70.21 | 0.295 |
| MVP (pl) | 10.95±1.01 | 10.51±0.91 | 11.14±1.17 | 11.21±0.95 | 0.001 |
| P-LCR (%) | 31.83±8.55 | 28.41±7.69 | 32.71±9.99 | 34.36±7.99 | 0.004 |
| PDW (%) | 13.44±2.32 | 12.58±2.11 | 13.85±2.68 | 13.89±2.16 | 0.010 |
| WBC(*10^9/L) | 7.9±4.72 | 6.48±1.73 | 7.59±9.32 | 9.61±2.93 | 0.032 |
| NT-ProBNP (pg/ml) | 990.39±1622.62 | 116.17±169.41 | 508.31±1079.16 | 2346.70±3619.01 | <0.017 |
| CTnT (μ g/L) | 0.9382±1.68 | 0.0199±0.19 | 0.0041±0.06 | 2.7907±4.81 | <0.001 |
| hsCRP (mg/L) | 8.83±16.94 | 3.09±7.29 | 3.11± 6.07 | 20.31±37.46 | <0.003 |
| TB (μ mol/L) | 10.36±5.22 | 10.53±7.80 | 9.49±3.36 | 11.08± 4.51 | 0.678 |
| TC (mmol/L) | 4.232±1.09 | 4.229±0.92 | 4.193±1.11 | 4.274±1.24 | 0.844 |
| TG (mmol/L) | 1.718±1.13 | 1.599±0.96 | 1.936±1.12 | 1.621±1.32 | 0.110 |
| HDL (mmol/L) | 1.128±0.81 | 1.199±0.29 | 1.081±0.27 | 1.104±0.25 | 0.035 |
| LDH (mmol/L) | 2.298±1.22 | 2.209±0.75 | 2.268±1.03 | 2.419±1.9 | 0.747 |
| Creatinine (μ mol/L) | 83.78±37.59 | 74.56±18.96 | 84.39±34.85 | 92.4±58.98 | 0.091 |
| UA (mmol/L) | 6.046±1.93 | 5.809±1.63 | 6.249±1.87 | 6.079±2.30 | 0.214 |

Abbreviations in table: Platelet Count(Plt), Mean Platelet volume (MPV), Platelet-Large Cell Ratio（P-LCR, Platelet Volume Distribution Width（PDW), White Blood Cell Count（WBC), Troponin T (CTnT), Hypersensitive C-reactive Protein (HsCRP), Total Bilirubin(TB),Total Cholesterol(TC), Triglyceride (TG), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Creatinine(Cr),Uric Acid (UA).

Case group 1, (S-CHD group) were the patients with Stable-type of Coronary heart disease confirmed diagnosis coronary angiography examination, with a total 58 cases, among them 42 male patients and 16 female patients and case group 2, (ACS group) was the patients who suffering acute coronary syndrome episodes (both unstable angina and myocardial infarction) at admission and received coronary angiography and interventional treatment thereafter. Containing a total of 47 patients, among them 37 male patients and 10 female patients, clinical data of each group are shown (Table 1).

**Table 3: Differences in platelet volume distribution width (PDW) between groups.**

| Group | | P-value | 95% CI | |
|---|---|---|---|---|
| | | | Lower bound | Upper bound |
| NCHD | S-CHD | 0.013 | -2.325 | -0.230 |
| | ACS | 0.005 | -2.273 | -0.299 |
| SCHD | N-CHD | 0.013 | 0.229 | 2.326 |
| | ACS | 1.001 | -1.054 | 1.023 |
| ACS | N-CHD | 0.005 | 0.296 | 2.279 |
| | S-CHD | 1.000 | -1.030 | 1.059 |

Continuous data were expressed as the mean±standard deviation: S-CHD and ACS group on gender have statistically significant differences in the N-CHD group (P<0.01), Male patients were obviously more than female patients; At the same time, the mean age of patients with coronary heart disease, in groups S-CHD and ACS were greater than that in patients with N-CHD group, on average above 62 year-old (P<0.01). In addition, hypertension patients with coronary heart disease were also significantly higher than in patients without Coronary heart disease, with (P<0.05).

**Table 4: Differences in platelet-large cell ratio (P-LCR) between groups.**

| Group | | P-value | 95% CI | |
|---|---|---|---|---|
| | | | Lower bound | Upper bound |
| NCHD | S-CHD | 0.007 | -7.551 | -1.157 |
| | ACS | 0.002 | -9.267 | -2.296 |
| SCHD | N-CHD | 0.007 | 1.159 | 7.551 |
| | ACS | 0.323 | -4.696 | 1.565 |
| ACS | N-CHD | 0.002 | 2.578 | 9.263 |
| | S-CHD | 0.323 | -1.564 | 4.698 |

Continuous data were expressed as mean±standard deviation. The platelet volume distribution width of S-CHD group and ACS group was significantly higher than that of N-CHD group; the differences were 13.85±2.68, 13.89±2.16 and 12.58±2.11 respectively, (P<0.01). In addition, each group in MPV, P-LCR, WBC Count, NT-ProBNP and HsCRP had obvious statistically significant differences (P<0.05) (Table 2).

Through one-way ANOVA, we found that the reflected change of platelet volume index of PDW, MPV and P-LCR in the S-CHD group and ACS group had statistically significant difference than those in the N-CHD group (P<0.01). Among them, the PDW of S-CHD group was significantly higher than that of N-CHD (95% CI of 0.229 to 2.326, P=0.013), but there was no difference from the ACS group (95% CI -1.054 to 1.023, with P=1.001) (Table 3). Differences in platelet-large cell ratio (P-LCR) and mean platelet volume (MPV) between groups is given in Table 4 and 5.

**Table 5: Differences in mean platelet volume (MPV) between groups.**

| Group | | P-value | 95% CI | |
|---|---|---|---|---|
| | | | Lower bound | Upper bound |
| N-CHD | S-CHD | 0.004 | -1.093 | -0.165 |
| | ACS | 0.001 | -1.132 | -0.253 |
| S-CHD | N-CHD | 0.004 | 0.165 | 1.093 |
| | A-CS | 0.984 | -0.541 | 0.414 |
| ACS | NCHD | 0.001 | 0.253 | 1.132 |
| | S-CHD | 0.984 | -0.414 | 0.541 |

PDW were positively correlated with P-LCR, MPV, Albumin, triglyceride (TG) and negatively correlated with platelet count, wherein the highest correlation was found between the P-LCR and MPV, their correlation coefficients respectively were r=0.870 and r=0.957 (Table 6).

**Table 6: Correlation analysis between PDW and Plt, P-LCR, MPV, Alb, TG, and diabetes.**

| Variables | Pearson correlation coefficient (r) | P value |
|---|---|---|
| PLT count | -0.340 | <0.001 |
| P-LCR | 0.870 | <0.001 |
| MPV | 0.957 | <0.001 |
| Albumin | 0.154 | 0.044 |
| Triglyceride (TG) | 0.235 | 0.003 |
| Diabetes | 0.193 | 0.011 |

After adjustment for coronary heart disease-related risk factors such as age and gender, PDW remained independently associated with coronary heart disease (Table 7).

## DISCUSSION

Coronary atherosclerosis is the leading cause of morbidity and mortality among all cardiovascular diseases; consequently, it costs a huge amount of medical and social resources each year and seriously affects the public health and life. Furthermore, with the development of society, the cardiovascular disease in developed and developing countries is the main health issues, thus, how

to accurately distinguish between high-risk groups, and further evaluate the risk stratification, and prognosis of the population is extremely important. Therefore, our study found that PDW, MPV, and P-LCR changes were positively correlated with coronary heart disease, both for patients with stable or unstable coronary heart disease, hence, in these three indicators were significantly higher than non-coronary heart disease patients with significant statistical differences (P<0.01).

**Table 7: Multivariate linear regression analysis between PDW and coronary heart disease-related risk factors.**

| Groups | Variables | Regression coefficient | Wald value | P value | OR value | 95% CI for OR value | |
|---|---|---|---|---|---|---|---|
| | | | | | | Lower | Upper |
| S-CHD | Age | 0.081 | 12.659 | <0.001 | 1.087 | 1.039 | 1.137 |
| | Gender | -1.183 | 6.746 | 0.008 | 0.309 | 0.127 | 0.746 |
| | PDW | 0.294 | 8.042 | 0.006 | 1.341 | 1.091 | 1.640 |
| N-CHD | Age | 0.112 | 19.359 | <0.001 | 1.118 | 1.061 | 1.175 |
| | Gender | -1.675 | 10.981 | 0.001 | 0.187 | 0.071 | 0.507 |
| | PDW | 0.303 | 7.793 | 0.006 | 1.349 | 1.095 | 1.671 |

Taking the N-CHD group as reference.

PDW reflects the platelet heterogeneity in circulation, the size of the platelet distribution and the degree of dispersion; MPV reflects the overall situation of the platelet volume size in the body and the platelet formation in the bone marrow; meanwhile, P-LCR reflects the circulatory system in large volume platelet turnover situation. Therefore, we suggested that the elevated of these three indicators and main mechanism associated with coronary heart disease were the important role of P-LCR in the development of coronary heart disease.

Through a process called atherogenesis, atherosclerosis is formed in the tunica intima zone of the arterial vessel walls**,** primarily the large and medium-sized elastic and muscular arteries.[8,9] Normally, this is a very slowly developing lesion that might take decades to become clinically significant. The "atherosclerotic plaques" protrude into the vessel lumen and obstructs the blood flow. This obstruction is dangerous in itself, but the atherosclerotic plaques also weaken the underlying tunica media, which may lead to plaque rupture and the creation of an acute arterial thrombosis.[10] Such plaque ruptures cause acute myocardial infarctions and strokes.

Intimal smooth muscle cell proliferation, monocytes transform into macrophages invade and begin to attack endothelium, a large number of collagen, proteoglycan, elastic fibers and lipid accumulation of atherosclerotic lesions.[11] The development of atherosclerosis is caused by a variety of factors such as low-density lipoprotein and homocysteine, increased smoking, diabetes and high blood pressure resulting in a large number of free radicals, genetic variation, microbial infection and other common effects caused by chronic inflammatory process.[12,13] At the same time, by the endothelial cells, macrophages, monocytes, T cells, platelets in their interactions released and produced a large number of cytokines, growth factors, chemokines, hormones and enzymes that are involved in this process. Among them, platelets act as the smallest blood cells in the blood circulation; the changes of platelet morphology and function are closely related to the incidence and development of coronary heart disease. during the development of atherosclerosis, platelets adsorb and accumulate on damaged arterial intima, collagen and macrophages. Activated platelets release platelet factor and growth factor-containing cell granules and coagulation factors such as thrombin, together stimulate proliferation and migration of smooth muscle cells and monocytes.[14] After platelet activation can promote the increase in arachidonic acid synthesis, prostaglandins, thromboxane, 5-hydroxytryptamine (5-HT), leukotriene, conversion and release of other active factors, and 5-HT, leukotriene strong vasoconstriction and platelet aggregation, increased coronary artery contraction, vascular endothelial damage, the degree of thrombus formation and proliferation of vascular smooth muscle cells, thereby enhanced the inflammatory reaction of the intima. In addition, after the activation of platelet aggregation in blood vessel walls and to gather other platelets thrombogenesis and expand, once the platelet rupture or thrombosis is to further increase the vascular injury and the occurrence of unstable angina and even acute myocardial infarction. Therefore, the structure, morphology, function and metabolism of platelets are closely related to the formation of thrombosis and the development of atherosclerosis.

During the same period, the pathogenesis of coronary atherosclerosis, platelet activation, increased damage, shortened survival period lead to constant consumption of platelets, thrombocytopenia in the peripheral blood circulation, promote the bone marrow megakaryocyte increasing feedback and splitting to form new platelet that quickly replenish the number of platelets in peripheral blood circulation, newborn platelets are larger in volume than aged platelets, and contain more dense

granules, which can release more clotting material.[4] Peripheral platelets appear to vary in size due to the compensatory proliferation of bone marrow, in the case of uneven distribution, the platelet volume distribution width, the mean platelet volume, and the platelet large cell ratio can reflect the change in different aspects.

Present study had several limitations; first, this study was a retrospective case analysis. Second, there are many influencing factors of coronary heart disease, including age, gender, diabetes mellitus, hyperlipidemia, hypertension, body mass index (BMI), and because of the sample size was relatively small, we could not observe other coronary heart disease or thrombosis factors such as thromboxane B2, Platelet aggregation rate, fibrinogen, blood viscosity and leukotriene into the analysis, therefore, it is inevitable there is a certain bias. On the other hand, our studies were retrospectively analyzed so we were not able to obtain the data of these indexes and the prognosis of the patients. However, we hoped to reduce the effects of bias by increasing sample size and multiple stratification analysis, but the retrospective analysis of the study itself for the causal inference argument strength is low, so we hope that with further large-scale prospective study to further understanding the relationships between PDW, MPV, and P-LCR and coronary heart disease will become clearer with prediction of coronary heart disease and their feasibility in clinical application. Furthermore, the future direction of research should be focused to further understanding of the platelets and coronary heart disease-related pathophysiology mechanism, and to seek the best strategy for prevention and treatment of coronary heart disease

## CONCLUSION

The results of this study revealed, that there was no significant difference in Platelet count between patients with coronary heart disease (case group) and non-coronary heart disease (control group), while the differences of PDW, MPV, and P-LCR were statistically significant. And in the Multivariate analysis, PDW is, even more, an independent factor associated with coronary heart disease. Therefore, we believe that the platelet volume distribution width, mean platelet volume, and platelet-large cell ratio can well reflect the changes of platelets in the body, on the other hand; they have some reference value for the prediction, prevention and risk stratification of patients with coronary heart disease. In addition, the PDW and MVP of the S-CHD group were similar to those of the ACS group, therefore, the PDW and MVP can prompt the occurrence of acute coronary syndrome and acute myocardial infarction; however, the P-LCR of ACS group were higher than that of S-CHD group, but the difference was not statistically significant; nonetheless this may indicate that when there are more large platelets in circulation, the patient will be in a critical condition of adverse cardiovascular events. Thus, for MPV, PDW and P-LCR elevated populations may consider early intervention treatment, to prevent the incidence of adverse events.

Meanwhile, in the correlation analysis between PDW and diabetes, this study also revealed that the change of platelet volume was significantly correlated with DM (P=0.009, r=0.196), and diabetes is a high risk-factor for CHD, thus, the incidence and development of atherosclerosis in diabetic patients is associated with the increase of platelet-large cell ratio, increased platelet aggregation, and the release of related factors in diabetes mellitus.

Finally, the routine blood test is a current application widely used clinical test indicators, which is simple, convenient and inexpensive, so the use of PDW, MPV, and P-LCR as risk assessment indicators have been widely developed and their advantages can be generally accepted by patients.

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