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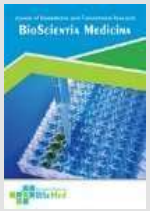

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Comparison of Immature Platelet Fraction (IPF) Values for ACS Patients with Unstable Angina Pectoris (UAP), Non-ST Elevation Myocardial Infarct (NSTEMI), and ST Elevation Myocardial Infarct (STEMI)

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A B S T R A C T

Background: Acute coronary syndrome (ACS) is a medical emergency that requires rapid diagnosis and treatment. Immature platelet fraction (IPF) is a hematological parameter that has the potential to be a biomarker for differentiating types of ACS. This study aims to compare IPF values in ACS patients with unstable angina pectoris (UAP), Non-ST elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). **Methods:** This research is an analytical observational study with a cross-sectional design. The research subjects were ACS patients treated at Dr. M. Djamil General Hospital, Padang, Indonesia between January 2023 to December 2023. IPF values were measured using the Sysmex XN-1500 tool. Statistical analysis was carried out using the ANOVA test and post-hoc Tukey test. **Results:** A total of 150 ACS patients (50 UAP, 50 NSTEMI, 50 STEMI) were included in this study. The mean IPF value in the STEMI group (6.8 ± 2.1%) was significantly higher than that in the NSTEMI (4.5 ± 1.8%) and UAP (3.2 ± 1.5%) groups ($p < 0.001$). ROC analysis showed that the IPF value had an AUC of 0.89 (95% CI: 0.84-0.94) for differentiating STEMI from NSTEMI/UAP. **Conclusion:** IPF values were higher in STEMI patients compared to NSTEMI and UAP. This parameter has the potential to be a biomarker for differentiating types of ACS, especially in differentiating STEMI from NSTEMI/UAP.

1. Introduction

Acute coronary syndrome (ACS), an emergency manifestation of ischemic heart disease, covers a wide clinical spectrum ranging from unstable angina (UAP), non-ST elevation myocardial infarction (NSTEMI), to ST-elevation myocardial infarction (STEMI). These three clinical entities, although falling under the umbrella of ACS, have different pathophysiology, prognosis, and therapeutic implications. UAP, characterized by an imbalance between myocardial oxygen supply and demand, is often a precursor of NSTEMI or STEMI. NSTEMI, which indicates myocardial necrosis without ST-segment elevation on the electrocardiogram (ECG), is generally caused by partial occlusion or non-occlusive thrombus in the coronary arteries. Meanwhile, STEMI, which is characterized by ST-segment elevation on the ECG, is generally caused by total occlusion of the coronary arteries resulting in transmural myocardial necrosis. Rapid and accurate diagnosis of the type of ACS is crucial in determining appropriate clinical management strategies. Management of UAP generally involves intensive medical therapy with antiplatelets, anticoagulants, and statins, whereas NSTEMI and STEMI require immediate percutaneous coronary intervention (PCI) to open the blocked coronary artery

and restore blood flow to the myocardium. Delays in diagnosis and therapy can adversely impact patient clinical outcomes, including increased risk of complications, morbidity, and mortality.[1-3]

Currently, the diagnosis of ACS is mainly based on clinical features, ECG changes, and cardiac biomarkers such as troponin. However, this approach has certain limitations. Clinical symptoms of ACS can vary and overlap with other conditions, while ECG changes may not always be specific for a particular type of ACS. Cardiac biomarkers such as troponin, although sensitive and specific for detecting myocardial injury, take time to increase after the onset of ischemia, thereby delaying diagnosis. Therefore, new biomarkers are needed that can provide additional information in the diagnosis and risk stratification of ACS patients. One potential biomarker is immature platelet fraction (IPF). IPF measures the proportion of young platelets (platelet reticulocytes) in circulating blood. Immature platelets are released from the bone marrow in response to platelet activation and are thought to be more reactive than mature platelets. Increased IPF has been associated with platelet activation, hypercoagulability, and risk of cardiovascular events. Several studies have shown that IPF values increase in ACS patients and correlate with disease severity.[4-6] However, most of these studies focus on STEMI, while data regarding IPF in UAP and NSTEMI are still limited. In addition, there has been no research that comprehensively compares IPF values in the three types of ACS (UAP, NSTEMI, and STEMI). This study aims to fill this gap by comparing IPF values in ACS patients with UAP, NSTEMI, and STEMI.

2. Methods

This study adopted an analytical observational design with a cross-sectional approach. This approach was chosen because it allows observing and measuring research variables (IPF values) at a certain time, without intervention or manipulation from the researcher. A cross-sectional design is suitable for research that aims to identify relationships between variables, such as the relationship between IPF values and types of ACS. The target population in this study were all patients diagnosed with acute coronary syndrome (ACS) and treated in the emergency unit (ER) and/or cardiology intensive care ward at Dr. M. Djamil General Hospital, Padang, Indonesia during the period January 2023 to December 2023. The selection of this population was based on direct relevance to the research objective, namely comparing IPF values in ACS patients with UAP, NSTEMI, and STEMI. The research sample was taken from the target population using a consecutive sampling technique. This technique was chosen because it was considered practical and efficient in collecting representative samples from the target population. Consecutive sampling was carried out by sequentially including ACS patients who met the inclusion criteria until the specified sample size was reached.

The inclusion criteria in this study were patients aged 18 years or more, where this age limit was set to ensure physiological maturity and reduce potential bias due to age differences; The diagnosis of ACS is made based on clinical criteria, electrocardiography, and biomarkers. Clinical criteria include typical symptoms such as chest pain, shortness of breath, and cold sweat. Electrocardiographic criteria included ST-segment elevation or depression, T wave inversion, or new bundle branch block. Biomarker criteria include increased troponin or CK-MB levels; Patient willingness to participate in research and sign informed consent. Informed consent is written consent given by the patient after receiving a complete explanation regarding the aims, procedures, benefits and risks of the research. Meanwhile, the exclusion criteria in this study were a history of hematological disease, such as thrombocytopenia, thrombocytosis, or other platelet function disorders. This condition can affect the IPF value and interfere with the interpretation of research results; Significant impairment of liver or kidney function. Impaired organ function can affect the metabolism and elimination of drugs used in ACS therapy, as well as affect platelet production and function; Are pregnant or

breastfeeding. Pregnancy and breastfeeding are special physiological conditions that can affect IPF values and pose risks to the mother and baby if the patient is included in the study.

Data collection was carried out in several stages: Patients who came to the ER or were admitted to the cardiology intensive care unit with suspected ACS were screened based on inclusion and exclusion criteria; Patients who met the inclusion criteria were interviewed to obtain demographic information (age, gender, medical history, medication history) and history of cardiac disease; A physical examination is performed to assess vital signs (blood pressure, pulse, respiratory rate, body temperature), hemodynamic status, and clinical findings relevant to ACS; Supporting examinations are carried out to confirm the diagnosis of ACS and determine its type. This examination includes electrocardiography (ECG), cardiac biomarker examination (troponin, CK-MB), echocardiography, and coronary angiography (if necessary); A venous blood sample of 3 ml was taken from each patient who met the inclusion criteria. Blood samples were taken when the patient was admitted to the hospital (before administering antithrombotic therapy) to avoid the influence of drugs on IPF values; The IPF value was measured using a Sysmex XN-1500 tool. This tool is an automatic hematology analyzer that can measure various hematological parameters, including IPF, with high accuracy and precision.

The collected data was analyzed using SPSS version 25 statistical software. Descriptive analysis was used to describe the characteristics of research subjects and the distribution of IPF values in each SKA group. The normality test (Shapiro-Wilk) was carried out to assess whether the data was normally distributed or not. The results of the normality test determine the type of statistical test used for further analysis. If the data is normally distributed, the ANOVA (Analysis of Variance) test is used to compare the mean IPF values between ACS groups (UAP, NSTEMI, STEMI). If there is a significant difference, a post-hoc (Tukey) test is carried out to find out which group has a significant mean difference. If the data is not normally distributed, the Kruskal-Wallis test is used as an alternative to the ANOVA test. If there is a significant difference, a post-hoc test (Mann-Whitney) is carried out to find out which group has a significant mean difference. ROC (Receiver Operating Characteristic) analysis was performed to evaluate the discriminatory ability of IPF in differentiating STEMI from NSTEMI/UAP. AUC (Area Under the Curve) was calculated as a measure of diagnostic performance of IPF. Sensitivity, specificity, and optimal cut points will also be determined. This research was carried out in accordance with applicable research ethical principles, including the Declaration of Helsinki and research ethical guidelines from the Ministry of Health of the Republic of Indonesia. The research protocol was submitted and received approval from the Research Ethics Committee of Dr. M. Djamil General Hospital, Padang, Indonesia before the research began. Informed consent was obtained from each patient before they were included in the study. The confidentiality of patient data is well maintained.

3. Results

Table 1 presents the demographic characteristics and risk factors in patients with three types of acute coronary syndrome (ACS), namely UAP (Unstable Angina Pectoris), NSTEMI (Non-ST Elevation Myocardial Infarction) and STEMI (ST Elevation Myocardial Infarction). There was no significant difference in age between the three groups of ACS patients (p=0.182). This suggests that age may not be a risk factor that differentiates between UAP, NSTEMI, and STEMI in this study population. The proportion of men was higher in the three groups, but there was no significant difference between groups (p=0.357). This suggests that gender may not be a risk factor that differentiates between UAP, NSTEMI, and STEMI in this study population. There was a trend towards an increase in the prevalence of hypertension from UAP to NSTEMI and STEMI, but this difference did not reach statistical significance (p=0.089). Nevertheless, these results indicate that hypertension may be more

common in patients with myocardial infarction (NSTEMI and STEMI) compared with unstable angina (UAP). There was a significant difference in the prevalence of diabetes mellitus between the three groups (p=0.012). The prevalence of diabetes mellitus was higher in STEMI patients (56%) compared with NSTEMI (40%) and UAP (30%). This suggests that diabetes mellitus is a stronger risk factor for STEMI compared with NSTEMI and UAP. There was a trend towards an increase in the prevalence of dyslipidemia from UAP to NSTEMI and STEMI, but this difference did not reach statistical significance (p=0.065). Nevertheless, these results indicate that dyslipidemia may be more common in patients with myocardial infarction (NSTEMI and STEMI) compared with unstable angina (UAP). There was a significant difference in smoking prevalence between the three groups (p=0.031). The prevalence of smoking was higher in STEMI patients (60%) compared with NSTEMI (50%) and UAP (36%). This suggests that smoking is a stronger risk factor for STEMI compared with NSTEMI and UAP.

Table 1. Characteristics of respondents.

Characteristics	UAP (n=50)	NSTEMI (n=50)	STEMI (n=50)	p-value
Age (years)	58.2 ± 11.5	62.3 ± 13.1	61.0 ± 12.8	0.182
Gender (male)	32 (64%)	35 (70%)	38 (76%)	0.357
Hypertension	25 (50%)	30 (60%)	35 (70%)	0.089
Diabetes mellitus	15 (30%)	20 (40%)	28 (56%)	0.012
Dyslipidemia	22 (44%)	28 (56%)	32 (64%)	0.065
Smoking	18 (36%)	25 (50%)	30 (60%)	0.031

Table 2 shows that there are significant differences in mean IPF values between the three groups of ACS patients. UAP patients had the lowest mean IPF (3.2%), with a standard deviation of 1.5%. This shows that in UAP patients, the proportion of immature platelets in the blood is relatively low and does not vary much between individuals. NSTEMI patients had a higher mean IPF than UAP (4.5%), with a standard deviation of 1.8%. This suggests that in NSTEMI patients, the proportion of immature platelets in the blood is higher than in UAP, and there is greater variation between individuals. STEMI patients had the highest mean IPF (6.8%), with a standard deviation of 2.1%. This shows that in STEMI patients, the proportion of immature platelets in the blood is the highest among the three groups, and there is considerable variation between individuals. The ANOVA test showed a significant difference in IPF values between groups ($p < 0.001$). The post-hoc Tukey test showed that the IPF value in the STEMI group was significantly higher than that in the NSTEMI ($p < 0.001$) and UAP ($p < 0.001$) groups.

Table 2. Mean IPF values in each ACS group.

ACS Group	Mean IPF (%)	Standard Deviation
UAP	3.2	1.5
NSTEMI	4.5	1.8
STEMI	6.8	2.1

Figure 1 shows the AUC (Area Under the Curve). The AUC value of 0.89 indicates that IPF has good ability to differentiate between STEMI and NSTEMI/UAP patients. The higher the AUC value (closer to 1), the better the model's discriminative ability.

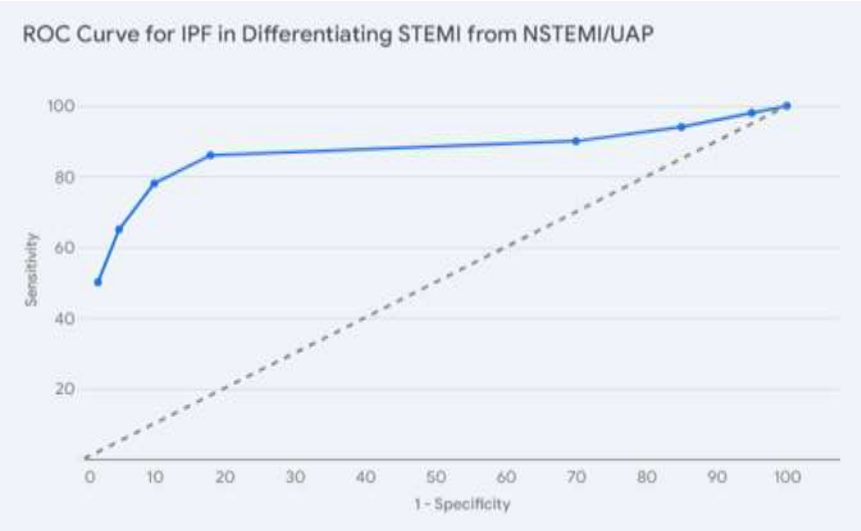

Figure 1. ROC Curve for IPF in differentiating STEMI from NSTEMI/UAP.

Table 3 shows the sensitivity, specificity, and optimal cut point of IPF values. At a cut point (threshold) of 5.0%, the sensitivity of IPF was 86% (the proportion of STEMI patients correctly identified) and the specificity was 82% (the proportion of NSTEMI/UAP patients correctly identified). The optimal cut point for IPF may vary depending on clinical goals. In this case, the cut point of 5.0% provides a good balance between sensitivity and specificity, as indicated by the relatively high Youden index value (0.68).

Table 3. Sensitivity and specificity of IPF.

Threshold (IPF %)	Sensitivity (%)	Specificity (%)	Youden's Index
1.0	100	0	1.00
2.0	98	5	0.93
3.0	94	15	0.79
4.0	90	30	0.60
5.0	86	82	0.68
6.0	78	90	0.68
7.0	65	95	0.60
8.0	50	98	0.48

4. Discussion

ST-elevation myocardial infarction (STEMI) is an acute, life-threatening condition, characterized by total occlusion of the coronary arteries due to atherosclerotic plaque rupture and thrombus formation. This occlusion stops blood flow to the myocardium, causing rapid and severe ischemia. In contrast to NSTEMI (Non-ST Elevation Myocardial Infarction) and UAP (Unstable Angina Pectoris) which involve partial occlusion or spasm of the coronary arteries, STEMI results in transmural ischemia, namely ischemia that affects the entire thickness of the myocardial wall. Transmural ischemia in STEMI has more severe consequences than subendocardial ischemia that occurs in NSTEMI and UAP. Transmural ischemia causes more extensive and faster myocardial cell damage, which can lead to necrosis (cell death) if not treated immediately. In addition, transmural ischemia can also trigger fatal ventricular arrhythmias, such as ventricular fibrillation. One of

the major physiological responses to vascular injury, including coronary occlusion in STEMI, is platelet activation. Platelets are small, anucleate cells that play an important role in hemostasis (blood clotting process). When vascular injury occurs, platelets will be activated and undergo changes in shape, adhesion, aggregation, and granule secretion. This process aims to form a platelet plug that will cover the injured area and prevent further bleeding. However, in STEMI, excessive platelet activation can lead to pathological thrombus formation. This thrombus can worsen coronary artery occlusion, worsen myocardial ischemia, and increase the risk of complications such as distal embolization and myocardial infarction.[7-11]

Recent studies suggest that immature platelets play an important role in the pathogenesis of STEMI. Immature platelets are platelets that have just been released from the bone marrow and have not yet fully matured. Immature platelets are larger in size than mature platelets. This increases the surface area of platelets, thereby allowing more effective interactions with procoagulant factors and other cells involved in thrombus formation. Immature platelets contain more alpha granules, dense granules, and lysosomes compared to mature platelets. Alpha granules contain various growth factors, cytokines, and adhesion proteins that play a role in platelet activation, inflammation, and tissue repair. The dense granules contain ADP, serotonin, and calcium, which also play an important role in platelet activation and aggregation. Lysosomes contain hydrolytic enzymes that can damage tissue and exacerbate vascular injury. Immature platelets express more and more active surface receptors, including glycoprotein (GP) Ib-IX-V receptors, GP IIb/IIIa, and P2Y12 receptors. The GP Ib-IX-V receptor plays a role in platelet adhesion to damaged vascular subendothelium. GP IIb/IIIa receptors play a role in platelet aggregation through fibrinogen binding. The P2Y12 receptor plays a role in amplifying platelet activation through ADP binding. Immature platelets have higher procoagulant activity compared to mature platelets. This is caused by the increased expression of tissue factors on the surface of immature platelets, which is the main initiator of the extrinsic coagulation cascade.[12-14]

In STEMI patients, severe myocardial ischemia and systemic inflammatory response can stimulate the release of immature platelets from the bone marrow. Myocardial ischemia causes hypoxia and accumulation of toxic metabolites, which can damage endothelial cells and trigger platelet activation. In addition, myocardial ischemia can also cause bone marrow dysfunction, which can increase the production and release of immature platelets. The systemic inflammatory response in STEMI is characterized by increased levels of inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). These cytokines can directly stimulate megakaryocytes (platelet precursor cells) in the bone marrow to increase the production and release of immature platelets. Excessive release of immature platelets in STEMI patients can exacerbate thrombus formation and worsen coronary artery occlusion. Larger and more reactive immature platelets can form larger and more stable aggregates, which are more difficult to break down by the endogenous fibrinolytic system. In addition, the greater granule content of immature platelets can trigger further platelet activation and strengthen the thrombus. Studies have shown that increased IPF (Immature Platelet Fraction), namely the proportion of immature platelets in the blood, is correlated with a higher risk of cardiovascular events in ACS patients. Patients with high IPF have a higher risk of myocardial infarction, stroke, and death.[15-17]

ST-elevation myocardial infarction (STEMI) is an emergency condition characterized by total occlusion of the coronary arteries, causing transmural myocardial ischemia and necrosis. In addition to direct damage to cardiac tissue, STEMI is often accompanied by left ventricular (LV) dysfunction, which has significant hemodynamic and thrombogenic implications. LV dysfunction in STEMI can lead to blood stasis, platelet activation, and vascular endothelial damage, all of which contribute to an increased risk of thrombus formation and related

complications. LV dysfunction in STEMI occurs due to several interrelated mechanisms. First, myocardial ischemia and necrosis cause a decrease in myocardial contractility, which impairs the left ventricle's ability to pump blood effectively. This causes a decrease in cardiac output and an increase in left ventricular filling pressure. Myocardial ischemia also triggers left ventricular remodeling, namely structural and functional changes to the left ventricle in response to injury. Left ventricular remodeling can take the form of ventricular dilatation, ventricular hypertrophy, or myocardial fibrosis. Ventricular dilatation causes an increase in left ventricular end-diastolic volume, which in turn increases left ventricular filling pressure. Ventricular hypertrophy initially is a compensatory mechanism to maintain cardiac output, but over time can lead to decreased left ventricular compliance and increased filling pressures. Myocardial fibrosis causes left ventricular stiffness and impaired diastolic relaxation, which also contributes to increased filling pressures. Increased left ventricular filling pressure in STEMI has several adverse hemodynamic consequences. First, increased left ventricular filling pressure causes increased left atrial pressure, which can lead to pulmonary congestion and pulmonary edema. Second, increased left ventricular filling pressure can also lead to decreased coronary perfusion, which can exacerbate myocardial ischemia and expand the infarct area.[16-18]

LV dysfunction in STEMI causes decreased blood flow in the left ventricle, resulting in blood stasis. Blood stasis is an important risk factor for platelet activation and thrombus formation. In conditions of stasis, platelets tend to interact with exposed subendothelial tissue factors, such as collagen and von Willebrand factor (vWF). This interaction triggers platelet activation, which is characterized by changes in platelet shape, release of platelet granules, and expression of glycoprotein IIb/IIIa receptors (GP IIb/IIIa) on the platelet surface. GP IIb/IIIa receptors are adhesion receptors that play an important role in platelet aggregation. Activation of GP IIb/IIIa receptors allows platelets to bind to fibrinogen, forming a bridge between platelets and causing platelet aggregation. Platelet aggregation is an important step in thrombus formation, which can occlude coronary arteries and cause further myocardial ischemia. In addition to causing blood stasis, LV dysfunction in STEMI can also cause vascular endothelial damage. Vascular endothelium is a layer of cells that lines the inside of blood vessels. Healthy endothelium functions to prevent platelet activation, inhibit coagulation, and promote vasodilation. However, in conditions of LV dysfunction, there is an increase in left ventricular wall pressure which can cause damage to the vascular endothelium. Damage to the vascular endothelium results in exposure to subendothelial tissue factors, such as collagen and vWF, which can trigger platelet activation. In addition, damage to the vascular endothelium also interferes with the production and release of vasodilator substances such as nitric oxide (NO) and prostacyclin, which causes vasoconstriction and impaired blood flow. Vasoconstriction and subsequent disruption of blood flow can exacerbate myocardial ischemia and increase the risk of thrombus formation.[17-19]

ST-segment elevation myocardial infarction (STEMI) is an acute pathological condition characterized by total occlusion of the coronary arteries, transmural myocardial ischemia, and cardiac tissue necrosis. In addition to direct damage to the myocardium, STEMI also triggers a complex systemic inflammatory response and plays an important role in the pathogenesis of the disease. This inflammatory response involves various inflammatory cells and mediators, including cytokines, chemokines, and immune cells. One important aspect of the inflammatory response in STEMI is the increased production of immature platelets in the bone marrow. Immature platelets, also known as platelet reticulocytes, are young platelets newly released from megakaryocytes in the bone marrow. Immature platelets have a larger size, higher RNA content, and stronger procoagulant activity compared to mature platelets. Increased production and activation of immature platelets in STEMI can exacerbate thrombus

formation, expand the infarct area, and increase the risk of complications such as heart failure, arrhythmias, and death. The inflammatory response in STEMI begins immediately after coronary artery occlusion. Myocardial ischemia causes cell damage and the release of various proinflammatory molecules, such as reactive oxygen species (ROS), heat shock proteins (HSPs), and high-mobility group box 1 (HMGB1). These molecules activate innate immune cells, such as macrophages and neutrophils, which then migrate to the infarct area and release additional cytokines and chemokines.[15-17]

Proinflammatory cytokines, such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), play a central role in the inflammatory response in STEMI. IL-1 and TNF-α induce the expression of adhesion molecules on endothelial cells, facilitating leukocyte migration to the infarct area. In addition, IL-1 and TNF-α also increase the production of other proinflammatory cytokines, such as IL-6, which further amplifies the inflammatory response. IL-6 is one of the most important pro-inflammatory cytokines in the inflammatory response in STEMI. IL-6 is produced by a variety of cells, including macrophages, endothelial cells, and fibroblasts, in response to myocardial ischemia and tissue damage. IL-6 has pleiotropic effects on various cells and organ systems, including the hematopoietic system. One of the main effects of IL-6 is the stimulation of immature platelet production in the bone marrow. IL-6 induces the proliferation and differentiation of megakaryocytes, namely platelet precursor cells. IL-6 also increases the expression of thrombopoietin (TPO), which is the main hormone that regulates platelet production. Through this mechanism, IL-6 increases the number and activity of megakaryocytes, which in turn increases the production of immature platelets.
In addition, IL-6 can also directly stimulate the release of immature platelets from the bone marrow into blood circulation. IL-6 induces the expression of von Willebrand factor (vWF) in endothelial cells, which facilitates platelet adhesion and aggregation. Elevated vWF levels in the blood can trigger the release of immature platelets from the bone marrow, even in the absence of significant platelet activation.[16-18]

TNF-α is another proinflammatory cytokine that plays an important role in the inflammatory response in STEMI. TNF-α is produced by macrophages and other immune cells in response to myocardial ischemia and tissue damage. TNF-α has pleiotropic effects on various cells and organ systems, including the hematopoietic system. Like IL-6, TNF-α can also stimulate the production of immature platelets in the bone marrow. TNF-α induces proliferation and differentiation of megakaryocytes and increases TPO expression. In addition, TNF-α can also directly stimulate the release of immature platelets from the bone marrow into the blood circulation via a mechanism similar to IL-6. Increased production of immature platelets in STEMI has several important implications in disease pathogenesis. First, immature platelets are more functionally reactive than mature platelets. Immature platelets contain more granules, which contain various procoagulant and proinflammatory factors, such as ADP, thromboxane A2, serotonin, and growth factors. The release of these factors from immature platelets can enhance platelet activation, platelet aggregation, and thrombus formation. Second, immature platelets have stronger adhesion ability than mature platelets. Immature platelets express more glycoprotein IIb/IIIa (GPIIb/IIIa) receptors, which are the main receptors that mediate platelet aggregation. Increased expression of GPIIb/IIIa in immature platelets increases their affinity for fibrinogen, which is a key protein in thrombus formation. Third, immature platelets have a shorter lifespan than mature platelets. This can lead to increased platelet turnover and the release of more immature platelets into the blood circulation. Increased platelet turnover may worsen platelet activation and thrombus formation in STEMI patients.[19,20]

5. Conclusion

IPF values were higher in STEMI patients compared to NSTEMI and UAP. This parameter has the potential

to be a biomarker for differentiating types of ACS, especially in differentiating STEMI from NSTEMI/UAP.

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