Original Article



Assessment of plasma level of D-dimer, platelets, and MPV in myocardial infarction patients

Albara Ahmed¹, Abrar Azhari Dafaalla², Hisham Ali Waggiallah^{3*}

¹Department of Hematology, Medical Laboratory Science Program, Alfajr College of Science and Technology, Khartoum, Sudan. ²Department of Hematology and Immunohematology, Faculty of Medical Laboratory Sciences, National University, Khartoum, Sudan. ³Department of Medical Laboratory Science, College of Applied Medical Science, Prince Sattam Pin Abdulaziz University, Alkharj, KSA.

Correspondence: Hisham Ali Waggiallah, Department of Medical Laboratory Science, College of Applied Medical Science, Prince Sattam Pin Abdulaziz University, Alkharj, KSA. hishamwagg30@hotmail.com

ABSTRACT

Myocardial infarction disease is a leading cause of morbidity and mortality. D-dimer was previously only utilized as a marker in venous thromboembolism and aortic dissection, and types of research on its significance in MI diagnosis have been undertaken. This study aims to assess D- dimer plasma level &platelets and MPV among myocardial infarction patients. Fifty Sudanese patients with myocardial infarction were enrolled as the case group, and fifty healthy individuals of various ages were included as the control group. D- dimer was measured using an immunodetection technique in both groups, whereas platelet counts and MPV were measured using a cell counter. (automated hematology analyzer Sysmex KX-21N). When patient and control samples were analyzed, it was observed that MPV and D-dimer had a highly significant relationship (P-value 0.001), whereas platelet count has a significant correlation (P-value 0.001). (P-value 0.05). Also, there is a significant association between patient age groups, platelet count, and D dimers. The D-dimer, platelet count, and MPV were considered as risk factors and diagnostic values for MI.

Keywords: D-dimer, Platelets, MPV, Myocardial infarction

Introduction

In the Western world, myocardial infarction (MI) is the major cause of death [1]. Platelets have an important part in the growth of coronary plaques and the thrombotic blockage of coronary arteries, which leads to ischemia and MI [2-4]. Furthermore, microembolization and platelet aggregation within the myocardium's damaged microcirculation during late ischemia and reperfusion (IR) cause additional tissue injury [2]. Because D-dimer is the major breakdown product of cross-linked fibrin, it acts as an effective marker of continuing coagulation with

Access this article online	
Website: www.japer.in	E-ISSN: 2249-3379

How to cite this article: Ahmed A, Dafaalla AA, Waggiallah HA. Assessment of plasma level of D-dimer, platelets, and MPV in myocardial infarction patients. J Adv Pharm Educ Res. 2021;11(4):55-9. https://doi.org/10.51847/rXvmRSgrTe

fibrinolysis. D-dimer levels have also been demonstrated to have a strong correlation with later coronary artery occurrences [5-7]. Platelet activation is critical for the onset of acute coronary syndromes. Platelet size has been linked to platelet function. Large platelets are thought to be more metabolically and enzymatically active than smaller ones [8]. High mean platelet volume (MPV) is linked to the degree of sluggish coronary flow [9]. MPV levels are higher in patients with unstable angina pectoris and MI. Higher MPV following acute ischemic cerebrovascular events is related to a poorer result and a higher risk of recurrent stroke. It has also been identified as an independent risk factor for MI, but not for the prevalence of coronary artery disease. MPV has been identified as a robust, independent predictor of poor angiographic reperfusion and 6month death [8]. Platelet size has also been involved in the development of restenosis following successful coronary angioplasty [8]. As a result, the purpose of this study is to assess the levels of D-dimer, platelets, and MPV in Sudanese patients with myocardial infarction.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Materials and Methods

Study design

This hospital-based case-control study was undertaken at Ahmed Gasim Hospital in Khartoum, Sudan, between December 2020 and January 2021. One hundred Sudanese volunteers participated in this study, 50 of whom were MI patients while the others appeared to be healthy as controls.

Inclusion criteria

Patients with MI in Khartoum state who were on regular followup were included in this study.

Exclusion criteria

Patients with other diseases rather than MI such as Bleeding disorder, malignancy, patients with PE, patients with DVT, patients with chronic liver disease, and patients with recent surgery were excluded

Data collection

The demographic data were acquired from individuals using a pre-prepared structural questionnaire; the questionnaire is an open-ended questionnaire performed by an interviewer.

Sampling

Each individual had 3 mL of venous blood drawn, with 2.7mL collected in 3.8 % tri-sodium citrate (9:1 vol/vol) for D-dimer assay. After centrifuging the samples for 15 minutes at 2500 rpm, the plasma is delivered to the laboratory in ice bags and maintained at 25 $^{\circ}$ C until evaluated within 24 hours of collection To collect CBC samples, blood was drawn into a tube containing an anticoagulant (usually EDTA) to prevent natural coagulation.

Principle of D dimer

The sandwich immunodetection method is used in the test; the detector antibody in buffer binds to antigens in the sample, generating antigen-antibody complexes that move to the nitrocellulose matrix and are collected by the other immobilized antibody on the test strip. The more antigens in the sample, the more antigen-antibody complexes produced, resulting in a brighter fluorescence signal on the detector antibody, It is processed by a piece of equipment for the Ichroma test to determine D-dimer concentration in a sample [10].

Principle of Sysmex KX-21N for CBC analysis

The Sysmex KX-21N is a quantitative automated hematology analyzer that can determine 17 hematological parameters in vitro. The numerical and/or morphologic findings of CBC can help in the diagnosis of such clinical conditions. The KX-21N uses electrical resistance detection to count and size red blood cells (RBC) and platelets (PLT). Using cumulative pulse height detection, hematocrit (HCT) is calculated as the ratio of total RBC volume to whole blood. HGB is transformed to methemoglobin and photometrically read at 555 nm. White blood cells (WBC) are evaluated using direct current and classified into three groups using Particle Distribution Analysis (PDA). The WBC histogram that results is divided into lymphocyte, neutrophil, and mixed cell populations. Monocytes, basophils, and eosinophils make up the mixed cell population [11].

Statistical analysis

The data were analyzed with SPSS program version 23.0. Initially, all information was gathered through a data master sheet and coded into variables. The results were presented using descriptive and inferential statistics such as the Independent T-test, one sample T-test, One Way ANOVA (Analysis of variances) test, Pearson Correlation test, and ROC Curve. A statistically significant p-value of less than 0.05 was evaluated.

Results and Discussion

This study included fifty patients who had all suffered a myocardial infarction. **Figure 1** shows that the average age of the participants ranged from 40 to 49 years. Males made up 62% of all patients, while females comprised 38% of all participants, as seen in **Figure 2**. **Figure 3** shows that the treatment duration of 7-12 months accounted for the greatest number 27 %.

As patient and control samples were examined, it was observed that MPV and D- dimer had a highly significant (P-value 0.001) relationship, whereas platelet count has a significant (P-value 0.05) relationship, as shown in **Table 1**. As seen in the **Table 2**, there was no significant relationship between treatment time and platelet count, MPV, or D dimer. Furthermore, as shown in the **Table 3**, there was no significant link between patient gender and platelet count, MPV, and D-dimers in the test and control samples. The **Table 4** indicates the substantial link between different age groups, platelet count, and D -dimers in patients.

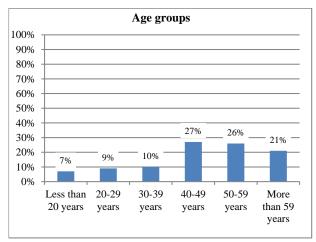


Figure 1. Shows the age distribution

.

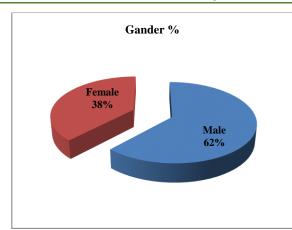


Figure 2. Illustrates the gender distribution

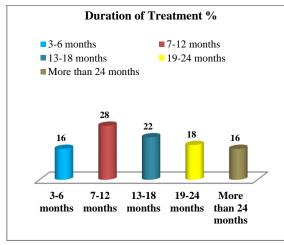


Figure 3. displays the percentage of treatment duration

Table 1. Comparison of D dimer, platelets count, and MPV in case (n=50) and control (n=50) individuals						
Variables Groups Mean±SD P-value						
D dimer	Case	471±157	0.001**			
	Control	160±60	0.001**			
Platelets count	Case	268±106	0.023*			
	Control	324±137	0.025			
MPV	Case	8.8 ± 1.5	0.001.55			
	Control	8.0± 1.0	0.001**			

*P≤0.05, ** P≤0.001

Table 2. Correlation between duration of treatment andD dimer, platelets count, and MPV in the case group.

Variables	Duration of treatment	No	Mean± SD	P-value
	3-6 months	8	533±152	
	7-12 months	14	473±152	
D dimer	13-18 months	11	460±173	0.485
	19-24 months	9	498±109	
	>24 months	8	393±193	
	3-6 months	8	329±171	
Platelets count	7-12 months	14	257±89	
	13-18 months	11	264±111	0.502
	19-24 months	9	260±43	

	> 24 months	8	239±96	
	3-6 months	8	9.2±1.8	
	7-12 months	14	8.6±0.6	
MPV	13-18 months	11	8.4±1.8	0.678
	19-24 months	9	9.2±2.0	
	>24 months	8	9±1.1	

Table 3. Association between Gender and D dimer, platelets	5
count, and MPV in both group case and control	

	Variables	Gender	No	Mean±	P-value
	D dimer	Male	31	482±170	0.531
	Dunner	Female	19	453±135	0.551
		Male	31	261±81	
Case	Platelets count	Female	19	278±140	0.603
	MPV	Male	31	8.8±1.4	0.764
		Female	19	8.9±1.6	
Control	D Dimer	Male	31	167±66	0.335
		Female	19	150±49	
	Platelets count	Male	31	308±139	
		Female	19	352±133	0.276
	MPV	Male	31	8±0.9	0.968
		Female	19	8±1.2	0.968

Table 4. Correlation between age groups and D dimer,platelets count, and MPV in case and control.

Va	riables	Age groups	No	Mean± SD	P value
		40-49 years	16	428±164	
	D dimer	50-59 years	20	498±157	0.398
		>59 years	14	483±150	
		40-49 years	16	305±134	
Control	Platelets count	50-59 years	20	257±85	0.206
		>59 years	14	240±90	
		40-49 years	16	8.8±1.7	
	MPV	50-59 years	20	9±1.6	0.731
		>59 years	14	9±1	
		< 20 years	7	99±43	
		20-29 years	9	146±29	
	D dimer	30-39 years	10	171±36	
		40-49 years	11	196±49	0.018*
		50-59 years	6	147±71	
Case		>59 years	7	179±94	
		< 20 years	7	399±147	
		20-29 years	9	418±197	
	Platelets count	30-39 years	10	285±85	0.038*
		40-49 years	11	273±68	
		50-59 years	6	237±74	

Journal of Advanced Pharmacy Education & Research | Oct-Dec 2022 | Vol 12 | Issue 4

	>59 years	7	214±59	
	< 20 years	7	7.9±1.0	
	20-29 years	9	7.8±0.9	
MPV	30-39 years	10	7.8±1.5	0.860
	40-49 years	11	8±0.8	
	50-59 years	6	8.5±0.9	
	>59 years	7	8±1	
*P< 0.05				

*P≤0.05

The major findings of our study show that platelet count, MPV, and D- dimer are strong predictors of clinical course in MI patients. We discovered a link between D dimer concentration, platelet count, and MPV and the risk of MI. According to the findings of this study, the age group with the most common manifestation of the condition is between the ages of 40 and 49. According to the findings of this study, the concentrations of Ddimer and MPV in the serum of patients with MI are significantly higher when compared to control samples, while platelet count appears to be significantly lower in patients.

D-dimer, a thrombotic burden marker that reflects fibrin turnover secondary to plaque rupture at any arterial location, and plasmin-mediated fibrin breakdown, another sensitive marker of continuing thrombosis, may be directly connected to an inflammatory vascular state [12, 13]. The levels of D-dimer, platelets count, and MPV was not altered by the duration or shortness of the treatment time in this study, indicating that the treatment period is a non-influential factor in the criteria used to evaluate MI, as was the patient's gender.

D-dimer has been linked to an increased risk of recurrent thrombotic events, all-cause mortality, and cardiovascular disease risk, especially in patients with vascular disease or coronary heart disease [14]. D-dimer was also linked to the incidence of heart failure and all-cause death in individuals with MI in a previous study [15, 16]. The current study discovered that the concentration of D-dimer in patients with MI grows steadily with age, whereas platelet count declines dramatically with age.

Platelets play an important part in the etiology of atherosclerotic problems, aiding in thrombus formation or apposition following plaque rupture. Platelets hyperactivity and local platelet activation following rupture of arteriosclerotic plaque in coronary arteries have been proposed to play a causative role in prothrombotic events leading to MI [17]. Because large platelets are more active than tiny platelets and have a larger thrombotic potential due to high thromboxane A2 concentrations, platelet size has been believed to indicate platelet degree of activity. Platelets have a critical role in accelerating the creation and spread of intracoronary thrombus, resulting in acute thrombotic episodes [17].

MPV, which is regarded as a key marker of cardiovascular illness, indicates the degree of activation of platelets to some extent [18]. It can also be employed in the risk prediction, diagnosis, and prognosis assessment of cardiovascular illnesses [19, 20]. According to a recent study, MPV increased dramatically in AMI patients, and the rise in MPV was associated with the long-term prognosis of AMI patients to some extent [21]. A recent study found that MPV was considerably higher in individuals with AMI and SCAD. The increase in MPV is most likely related to the strength of the body's inflammatory reaction [18].

Conclusion

We can conclude that our key findings reveal that platelet count, MPV, and D- dimer are all good predictors of clinical course in MI patients. It predicts risk andas traditional risk factors and biomarkers can guide prognosis and management decisions.

Acknowledgments: This Publication was supported by the Deanship of Scientific Research at Prince Sattam bin Abdulaziz University.

Conflict of interest: None

Financial support: None

Ethics statement: Ethical approval was obtained from the research committee at National University-Sudan for this research, written consent was obtained from participants in this study before sample collection after a thorough explanation of the research purpose, and the results of this research were retained for participants and their health leaders.

References

- Heusch G. Myocardial ischaemia–reperfusion injury and cardioprotection in perspective. Nat Rev Cardiol. 2020;17(12):773-89.
- Schanze N, Bode C, Duerschmied D. Platelet contributions to Myocardial Ischemia/Reperfusion injury. Front Immunol. 2019;10:1260.
- Mohamed WA, Badr NM, Fouad BE, Abd Alaal ME. Efficacy of intermittent pneumatic compression on blood flow in patient with varicose veins. Arch Pharm Pract. 2020;11(2):149-53.
- Nobandegani AS, Motamedifar M. Antibiotic sensitivity profile of the bacterial isolates from the blood samples of the patients in different wards of a major referral hospital, Shiraz, Iran 2015-2016. Pharmacophore. 2019;10(2):30-6.
- Bergh TH, Steen K, Lindau T, Soldal LA, Bernardshaw SV, Lunde L, et al. Costs analysis and comparison of usefulness of acute MRI and 2 weeks of cast immobilization for clinically suspected scaphoid fractures. Acta Orthop. 2015;86(3):303-9.
- Fathil MF, Arshad MM, Gopinath SC, Hashim U, Adzhri R, Ayub RM, et al. Diagnostics on acute myocardial infarction: Cardiac troponin biomarkers. Biosens Bioelectron. 2015;70:209-20.
- 7. Reihani H, Sepehri Shamloo A, Keshmiri A. Diagnostic value of D-dimer in acute myocardial infarction among

patients with suspected acute coronary syndrome. Cardiol Res. 2018;9(1):17-21.

- Amraotkar AR, Song DD, Otero D, Trainor PJ, Ismai I, Kothari V, et al. Platelet Count and mean platelet volume at the time of and after acute myocardial infarction. Clin Appl Thromb Hemost. 2017; 23(8):1052-9.
- Isik T, Ayhan E, Uyarel H, Ergelen M, Tanboga EH, Kurt M, et al. Increased mean platelet volume associated with extent of slow coronary flow. Cardiol J. 2012;19(4):355-62.
- 10. Ruivo S, Azevedo AM, Prazeres DMF. Colorimetric detection of D-dimer in a paper-based immunodetection device. Anal Biochem. 2017;538:5-12.
- 11. Tiruneh T, Kiros T, Getu S. Hematological reference intervals among full-term newborns in Ethiopia: a crosssectional study. BMC Pediatr. 2020;20(1):417.
- Yoshihisa A, Sato Y, Kimishima Y, Ichijo Y, Yokokawa T, Misaka T, et al. Soluble fibrin monomer complex is associated with cardio- and cerebrovascular events in patients with heart failure. Int J Cardiol Heart Vasc. 2020;32:100697.
- Weitz JI, Fredenburgh JC, Eikelboom JW. A test in context: D-dimer. J Am Coll Cardiol. 2017;70(19):2411-20.
- Di Castelnuovo A, de Curtis A, Costanzo S, Persichillo M, Olivieri M, Zito F, et al. Association of D-dimer levels with all-cause mortality in a healthy adult population: findings from the MOLI-SANI study. Haematologica. 2013;98(9):1476-80.

- Zhang X, Wang S, Liu J, Wang Y, Cai H, Wang D, et al. D-dimer and the incidence of heart failure and mortality after acute myocardial infarction. Heart. 2021;107(3):237-44.
- Zhang X, Wang S, Sun L, Fang S, Yu B. Prognostic value of D-dimer in acute myocardial infarction complicated by heart failure with preserved ejection fraction. ESC Heart Fail. 2020;7(6):4118-25.
- Alvitigala BY, Azra MA, Kottahachchi DU, Jayasekera MM, Wijesinghe RA. A study of association between platelet volume indices and ST elevation myocardial infarction. Int J Cardiol Heart Vasc. 2018;21:7-10.
- Ding L, Sun L, Wang F, Zhu L, Zhang T, Hua F. Clinical Significance of Platelet Volume and Other Platelet Parameters in Acute Myocardial Infarction and Stable Coronary Artery Disease. Arq Bras Cardiol. 2019;112(6):715-9.
- Bae MH, Lee JH, Yang DH, Park HS, Cho Y, Chae SC. White blood cell, hemoglobin, and platelet distribution width as short-term prognostic markers in patients with acute myocardial infarction. J Korean Med Sci. 2014;29(4):519-26.
- Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, et al. Mean platelet volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. J Thromb Haemost. 2010;8(1):148-56.
- Ranjith MP, DivyaRaj R, Mathew D, George B, Krishnan MN. Mean platelet volume and cardiovascular outcomes in acute myocardial infarction. Heart Asia. 2016;8(1):16-20.