The Use of Mean Platelet Volume (MPV) in Differentiating Between Hyper Destructive Versus Hypo Productive Thrombocytopenia

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Abstract

Objective: To establish the use of the mean platelet volume (MPV) as a tool in distinguishing between the hyper destructive versus hypo productive causes of thrombocytopenia.

Methods: This study was conducted at pathology department, Khawaja Muhammad Safdar Medical College, Sialkot (KMSMCS) from July 2020 to December 2020. Ethical approval was obtained from ethical committee of KMSMCS. 180 patients having platelet count less than 100x109/L were enrolled. Patients were categorized into two groups;

A. Patients having thrombocytopenia due to increase destruction of platelets

B. Patients having thrombocytopenia due to reduced production of platelets

Patients were selected from the CBC print generated by the automated haematology analyzer. Platelet count was manually verified by examining peripheral blood smear (PBS) on glass slides. MPV was recorded from the haematology analyzer print.

Results: 180 patients having platelet count less than $100x10^9$/L were enrolled. 87 were males and 93 were females. The male to female ratio was 1: 1.07. Age of the patients were between 0 - 88 years. Patients were further categorized into three groups on the basis of age. Group A (0 - 30 years), B (31 – 60 years) and C (61 -90 years). Group B wasthe most common age group having 108 patients followed by group A which had 42.

Conclusion: MPV is a platelet parameter which is readily available, authentic and can be relied upon to help as regards the basic cause of thrombocytopenia. It is also more convenient for the patient as compared to the bone marrow examination.

Keywords: Mean platelet volume (MPV) • Hyper destructive • Hypo productive • Thrombocytopenia

Introduction

The complete blood count (CBC) is a reflection of the hemopoietic activity showing the exact counts of the white blood cells and their differential, red blood cells and hemoglobin and platelets with their indices [1]. Platelets are derived from megakaryocytesand circulate in blood for 7-10 days [2]. Thrombocytopenia (platelet counts less than 150,000/µl) is routinely found in many disorders of the hemopoietic system [3]. Platelet levels below normal values confirm thrombocytopenia but they do not give a clue the underlying mechanism [4]. Advances in science and the use of automatic blood cell analyzers have led us to easily measure various platelet parameters. Platelet indices like the mean platelet volume (MPV), platelet distribution width (PDW), and platelet large cell ratio (P-LCR), may provide valuable clues [5]. Since immature platelets are large in size, a large number of these results in a higher MPV. The MPV is a part of the routine CBC with no extra added costs. Hence it is available along with the platelet count without any further testing required [6]. MPV is calculated as a measure of platelet volume expressed in femtoliter (fL).Normal range of MPV is reported to be between 7.2 and 11.7 fL. High MPV with ongoing thrombocytopenia represents peripheral destruction. Low MPV indicates underproduction/bone marrow suppression. MPV is in inverse relation to the platelet count [7,8]. Hence it is invaluable in differentiating between the hyper-destructive versus hypo-productive thrombocytopenia in a simple and reliable manner while being cost effective [9]. Additionally,it helps us to predict whether the thrombocytopenia will recover on its own thereby assisting in the clinical intervention of whether a platelet transfusion is needed or not [10-12]. Malarial infection also causes changes in platelet parameters hence the malarial parasite should also be ruled out[12]. Despite the numerous efforts that have been made as regards the clinical co relation of the platelet parameters, their definite link in terms of both diagnosis and prognosis, is yet to be fully proven [13,14]. This study is an attempt in adding to that knowledge and research.

Methodology

Centre of study

Department of Pathology, Khawaja Muhammad Safdar Medical College, Sialkot.

Period: July 2020 to December 2020

Inclusion Criteria: All thrombocytopenic patients having platelet count less than $100x10^9$/L

Exclusion Criteria: Patients who have received platelet transfusion 7 days back Patients who are not willing to participate in the study

Materials and Methods

This study was conducted at pathology department, Khawaja Muhammad Safdar Medical College, Sialkot (KMSMCS) from July 2020 to December 2020. Ethical approval was obtained from ethical committee of KMSMCS. 180 patients having platelet count less than $100x10^9$/L were enrolled.

Patients were selected from the CBC print generated by the automated haematology analyzer. Platelet count was manually verified by examining peripheral blood smear (PBS) on glass slides. MPV was recorded from the haematology analyzer print. Falsely high MPV due to EDTA induced platelet swelling was avoided by running each sample as soon as it was collected. Investigations required identifying the cause of thrombocytopenia such as bone marrow examination, viral serology, drug history, vitamin B12 and red cell folate levels were recorded. Slides were also examined for malarial parasite in suspected cases.

Patients were categorized into two groups;

A. Patients having thrombocytopenia due to increase destruction of platelets

B. Patients having thrombocytopenia due to reduce production of platelets

Group A (n = 167) included cases of hypersplenism (n = 75), immune thrombocytopenic purpura (ITP) (n = 31), chronic kidney disease (n =18) megaloblastic anaemia (n = 13), infection (n = 11), malaria (n = 8), neonatal thrombocytopenia (n = 4), gestational thrombocytopenia (n = 2) dengue (n = 1), drug induced thrombocytopenia (n = 1), hepatitis C virus (HCV) infection (n = 1), hepatitis B virus (HBV) infection (n = 1) and hepatocellular carcinoma (HCC) (n = 1).

Group B (n = 13) included cases of aplastic anaemia (n = 10) and acute leukaemia (n =3).

Bone marrow examination was performed in all patients of group B (n = 13). Bone marrow findings confirmed the diagnosis.

Results

180 patients having platelet count less than 100x109/L were enrolled. 87 were males and 93 were females. The male to female ratio was 1: 1.07. Age of the patients were between 0 - 88 years.

Patients were categorized into two groups;

A. Patients having thrombocytopenia due to increase destruction of platelets

B. Patients having thrombocytopenia due to reduce production of platelets

Group A (n = 167) included cases of hypersplenism (n = 75), immune thrombocytopenic purpura (ITP) (n = 31), chronic kidney disease (n =18) megaloblastic anaemia (n = 13), infection (n = 11), malaria (n = 8), neonatal thrombocytopenia (n = 4), gestational thrombocytopenia (n = 2) dengue (n = 1), drug induced thrombocytopenia (n = 1), hepatitis C virus (HCV) infection (n = 1), hepatitis B virus (HBV) infection (n = 1) and hepatocellular carcinoma (HCC) (n = 1) (Table 1).

Group B (n = 13) included cases of aplastic anaemia (n = 10) and acute leukaemia (n =3) (Table 2).

In group A mean MPV was 11.03 ± 1.38 and mean platelet count was 66.52(Table 3). In group B mean MPV was 8.53 ± 0.92 and mean platelet count was 51.77 (Table 4).

Table 1. Patients in Group A.

Diagnosis	Patients
Hypersplenism	75
Immune thrombocytopenic purpura (ITP)	31
Chronic Kidney Disease	18
Megaloblastic anaemia	13
Infection	11
Malaria	08
Neonatal Thrombocytopenia	04
Gestational thrombocytopenia	02
Dengue	01
Drug induced thrombocytopenia	01
HCV infection	01
HBV infection	01
Hepatocellular carcinoma (HCC)	01
Total	167

Thrombocytopenia due to increase destruction of platelets

Table 2. Patients in Group B.

Diagnosis	Patients
Aplastic Anaemia	10
Acute Leukaemia	03
Total	13

Table 3. Mean platelet volume in Group A.

Diagnosis	Patients	Mean Platelet count x109/L	MPV fL, ±SD
Hypersplenism	75	68.92	10.74 ± 1.55
Immune thrombocytopenic purpura (ITP)	31	62.39	11.86 ± 1.62
Chronic Kidney Disease	18	76.72	10.49± 1.19
Megaloblastic anaemia	13	47.92	10.89± 1.69
Infection	11	71.36	12.23± 1.04
Malaria	08	61.75	10.6± 0.96
Neonatal Thrombocytopenia	04	66.25	10.23± 0.95
Gestational thrombocytopenia	02	75.50	11.20± 0.14
Dengue	01	64	9.90
Drug induced thrombocytopenia	01	30	10
HCV infection	01	55	11.8
HBV infection	01	77	11.6
Hepatocellular carcinoma (HCC)	01	81	12.2
Total	167	66.52	11.03 ± 1.38

Table 4. Mean platelet volume in Group B.

Diagnosis	Patients	Mean Platelet count x109/L	MPV fL, ±SD
Aplastic Anaemia	10	58.80	8.62 ± 0.94
Acute Leukaemia	03	28.33	8.23 ± 0.95
Total	13	**51.77**	**8.53 ± 0.92**

Discussion

A study at Chiang Mai University Hospital, confirmed that since immature platelets are larger in size as compared to mature ones, the MPV measured using an automated hematology analyzer in cases of peripheral destruction is usually higher than in cases involving BM defects [15]. The findings of another similar study supported this hypothesis. In the validation cohort, where the causes of thrombocytopenia were confirmed by BM studies, the mean MPV in the over-destruction cohort was 10.4 fL compared to 7.2 fL in the underproductive BM group. The mean MPV in the over destruction cohort was close to a previous study (9.8 fL) [16].

A study conducted in Ethopia showed that the MPV and P-LCR can better predict ITP in the initial stage and when the baseline investigations are being done [17].

Thrombocytopenia due to reduce production of platelets A2-year observational study which included 100 patients with thrombocytopenia was done in a single tertiary care centre. Results were noted for all patients in regards to their diagnoses, platelet counts and the MPV. The peripheral blood films were also examined for large platelets. The group with hyperdestructive causes demonstrated a total of 53 cases out of 79 having an MPV value more than 10.5 fl. Despite the fact the MPV was not significantly high in the remaining cases; the presence of large platelets was recorded on the peripheral film. The group with hypo-productive causes had 21 cases, none of which demonstrated a high MPV [18].

In a study by Kumari et al ,the mean MPV in the hypoproduction categorywas10.6 ± 0.12 while that in the hyperdestruction catergorywas 11.4 ± 0.89 with a P-value of < 0.0001which is extremely significant [19].

Another study from India demonstrated a high MPV(10.46 fl) in the group with hyperdestructive thrombocytopenia in comparison with the hypoproduction group (8.7 fl) and the group showing abnormal pooling of platelets(8.15fl) and with the control group (9.5fl) [20].

The platelet indices are vital in differentiating between themechanism of thrombocytopenia be it, decreased production or increased destruction. These platelet parameters are easily available and cost effective for the evaluation of thrombocytopenia [21].

In another study, MPV (11.51±1.97)was higher in the category with increased destruction as compared to decreased poduction(8.34 ± 2) [22]. Silimar findings were observed in a study by Mirza Asif Baig in pediatric tertiary care hospital [23].

In a study by Rajalakhshami RB et al, the mean values of all the platelet parameters were higher in the category with increased destruction [PDW(16.6fL), MPV(12.1fL), P-LCR(42.3%)] as compared to the category with decreased production[PDW(11.8fL), MPV(10.9fL), P-LCR(31.5%) [24]. the results were significant with a p value of < 0.05.

Conclusion

Our study is in accordance with other studies showing that the MPV is a reliable indicator in differentiating between the hyperdestructive and hypoproductive causes of thrombocytopenia.

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