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Mean platelet volume: Should we really eye on?

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Abstract
Introduction: Mean platelet volume (MPV) is a hematology analyzer generated measurement of the average size of platelets found in peripheral blood. Thrombocytopenia could be because of accelerated destruction, impaired production or abnormal pooling in spleen. Analyses of MPV values play an important role in assessing the mechanism for TCP.
Material and Methods: 800 blood samples of patients with platelet count <1,50,000 ul and 800 control group samples with normal platelet count >1,50,000 ul were studied. Hematological analysis was done by Sysmex KX-21. Platelet count and MPV of all the cases and control group were noted. The cases were grouped into various categories of A, B or C depending on mechanism of decrease in platelets. Data was analyzed and tested for statistical significance using one-way ANOVA and post hoc testing using Tukey's test.
Results: Majority of the cases in the present study were in group A (75.3%), followed by group B (23.9%) and group C (0.7%). A higher value of MPV (10.15±1.30 fl) was found in group A compared to other categories. Significant results of MPV were seen in group A and group B category.
Conclusion: Mean platelet volume is an easily obtained platelet parameter which is helpful in delineating the various causes of TCPs. Hence an eye on the analyser readings of MPV helps in better targeted therapies.

Keywords: Mean platelet volume, thrombocytopenia, platelet count

Introduction

Mean platelet volume (MPV) is a hematology analyzer generated measurement of the average size of platelets found in peripheral blood ranging in values from 7.5- 11.5 fl normally. MPV predominantly helps in predicting the changes in rate of production of platelet. Higher MPV values are found in cases with accelerated destruction and lower MPV values in impaired production. When the number of platelets falls below normal levels <1,50,000 ul, the condition is termed as thrombocytopenia (TCP) [1-8]. TCP are the major group of hematological emergencies seen day to day. The antecedents for this decrease in platelets can be broadly accredited either to their accelerated destruction, impaired production or abnormal pooling in spleen [1-5]. Accelerated destructive TCPs are due to extramedullary platelet destruction with normal or increased bone marrow productions. Impaired productive TCPs result from decreased bone marrow production either due to primary or secondary bone marrow diseases. Splenic pooling are the various causes leading on to splenic sequestration of platelets. This information regarding the etiologies of TCP is important for the better targeted therapies. Bone marrow examination is the valuable tool which aids in distinguishing the various causes of TCPs. But the procedure is very much invasive and time consuming to be used in every case. Studied have revealed that analyses of MPV values plays an important role in delineating the heterogenous causes of TCP [1-8]. Hence the present study was undertaken to know the variations and correlations of MPV in various causes of TCPs.

Material and Methods

A six months prospective study was carried out from march 2016 to august 2016 in the department of pathology, Belagavi institute of medical sciences, Belagavi. 800 blood samples of patients with platelet count <1,50,000 ul and 800 control group samples with normal platelet count >1,50,000 ul were studied. Blood sample was collected in EDTA vacutainers and analysis was done by Sysmex KX-21 automated hematology analyzer within 2-6 hours of collection and peripheral smears were done and stained with leishman stain to

assess the blood cells morphology. From the analyzers results, platelet count and MPV of all the cases and control group were noted. Prior ethical clearance for the study and informed consent from the patients were obtained. The cases that had sufficient clinicohematological work up were included in the study, grouped into various categories A, B or C depending on mechanism of TCP and data was analyzed.

Statistical analysis

The platelet count and MPV were calculated for each group and presented along with the mean and standard deviation. Comparison of the platelet count and MPV between the different groups as well as the control was carried out with the help of standard t-tests. The results were tabulated and analyzed. A more detailed multiple comparisons were carried out using a one-way ANOVA and the post hoc testing was done using Tukey's test to know the significance between the groups with the p values. The p- value <0.05 was considered significant. All the statistical analyses were carried out using SPSS version 24(IBN, Chicago, USA).

Results

800 cases of TCP were grouped according to the most predominant mechanism similar to a study done by Reddy S *et al*. [10] The majority of the patients were males, making up 518 (64.7%) of the population and the remaining 282 (35.2%) were females. Ages of the patients ranged from 1 day (neonatal cases) to 92 years. Most of the individuals were between 21 and 30 years of age (21%).

Cases were grouped depending on mechanism of thrombocytopenia, Group A- Accelerated platelet destruction, Group B- Impaired platelet production and Group C- Abnormal platelet pooling. Majority of the cases in the present study were of group A (75.3%), followed by group B (23.9%) and group C (0.7%). Infections (63.1%) and anemias (72.2%) were the commonest category in group A and B respectively. In group C, all cases were of splenomegaly (0.7%). (Table 1)

Table 1: Distribution of cases in various categories

Cases	Group A Accelerated destruction	Group B Impaired production	Group C Abnormal pooling	Total
Infections	380 (63.1%)	-	-	380 (47.5%)
Neonatal	87(14.4%)	-	-	87(10.8%)
Cardiac	58 (9.6%)	-	-	58 (7.2%)
Pregnancy	34 (5.6%)	-	-	34 (4.2%)
Renal	24(3.9%)	-	-	24 (3%)
Liver	8 (1.3%)	-	-	8 (1%)
Sepsis	8(1.3%)	-	-	8 (1%)
ITP	2(0.3%)	-	-	2(0.2%)
Burns	2 (0.3%)	-	-	2 (0.2%)
Anemias	-	138(72.2%)	-	138(17.2%)
Leukemia	-	17(8.9%)	-	17(2.1%)
Pancytopenia	-	29(15.2%)	-	29(3.6%)
Postchemotherapy	-	6(3.1%)	-	6(0.7%)
Aplastic anemia	-	1(0.5%)	-	1(0.1%)
Splenomegaly	-	-	6 (100%)	6(0.7%)
Total	603 (75.3%)	191 (23.9%)	6 (0.7%)	800 (100%)

Based on these categories, the mean and standard deviation of the platelet count and MPV of each group was obtained, and is given in Table 2. From this table, it could be seen that that accelerated destruction and abnormal pooling group showed the higher values MPV and impaired production group had lower values.

Table 2: Platelet count and MPV according to each group

Group	Number	Platelet count	MPV (fL)
Accelerated destruction of platelets	603	90641±30761.15	10.15±1.30
Impaired production	191	83483±29500.71	8.52±1.69
Abnormal pooling	6	110750±28087.66	10.6±1.25
Control	800	259160±67846.8	10.15±1.29

The platelet count and MPV of the individually diagnosed cases within each broad group were examined. These results can be seen in Table 3. In group A, lowest PC was seen in burns cases (33000±32526.91), MPV was highest in ITP cases (11.55±0.35 fl) and least in pregnancy associated thrombocytopenia (9.78±1.57). In group B, MPV was highest in leukemia cases (9.60±0.57fl) and least in anemias (7.98±1.43fl). In group C, there were only the cases of splenomegaly with MPV 10.6±1.25 fl

Table 3: Platelet count and MPV in Group A, B and C categories

Group A	Category	Number (%)	Platelet count	MPV (fl)
	Infections	380 (63.1%)	92456±30153.48	10.26±1.34
	Neonatal	87(14.4%)	87522±31246.99	10.09±1.29
	Cardiac	58 (9.6%)	91943±29172.18	10.16±1.19
	Pregnancy	34 (5.6%)	94303±26640.53	9.78±1.57
	Renal	24(3.9%)	106909±21886.96	10.34±1.09
	Liver	8 (1.3%)	98500±29990.47	9.9±0.76
	Sepsis	8(1.3%)	87500±26592.16	9.11±0.96
	ITP	2(0.3%)	47500±45961.94	11.55±0.35
	Burns	2 (0.3%)	33000±32526.91	10.70±0.57
Group B	Anemia	138(72.2%)	84781±30261.39	8.44±1.71
	Leukemia	17(8.9%)	77000±32526.91	9.60±0.57
	Pancytopenia	29(15.2%)	94733±29504.16	8.50±1.62
	Postchemotherapy	6(3.1%)	75895±22536.13	8.60±0.57
	Aplastic anemia	1(0.5%)	76667	9.13
Group C	Splenomegaly	6 (100%)	110750±28087.66	10.6±1.25

The significances of platelet count and MPV were compared with control group by using an ANOVA and Tukey's test was used as a post hoc test (Table 4). The tabulation showed that platelet count was significant in all the three groups A, B, C when compared with control group. MPV was found significant in group A and B with control group with p value <0.05; but not significant in group C versus control group.

Table 4: Statistical comparison of platelet count and MPV of each group with control group.

Platelet Parameter	Group A Vs Control	Group B Vs Control	Group C Vs Control
Platelet count	t = -40.4	t = -38.092	t = -10.179
	p<0.05	p<0.05	p = 0.001
MPV	t = 10.694	t = -10.297	t = 0.717
	p<0.05	p<0.05	p = 0.524

For Table 4, p<0.05 is significant.

Discussion

Platelets are essential elements in clotting process required for the primary stoppage of bleeding after an injury. Platelets take their origin from megakaryocytes in the bone marrow. Decrease in platelets in peripheral blood below normal values is termed as TCP [1-5]. Studies have postulated the causes for this decrease in platelets being either to their accelerated destruction, impaired production or abnormal pooling in spleen. Also analysing MPV values help in assessing the mechanism for TCP [1-8]. Usually peripheral blood smears can be used to know the platelet number, size, distribution and morphology under microscopy [1-4]. Being in the digital age, automated cell counters give better information of these platelet parameters as platelet count and MPV. MPV is the measurement of the average size of platelets which helps in knowing the changes in rate of platelet production. Accelerated production leads to larger sized platelet and impaired production leads to smaller and aged platelets leading on to higher and lower MPV values respectively. Hence the MPV values can be used to make inference about platelet production or destruction [5-10].

The majority of the patients in the present study were males accounting to 518 (64.7%) and rest 282 (35.3%) were females. Ages of the patients ranged from 1 day (neonatal cases) to 92 years similar to other studies.[9-15] Patients were categorized into three groups according to the postulated mechanism of TCPs [10]. Majority of the cases in the present were of group A (75.3%), followed by group B (23.9%) and group C (0.7%) similar to the study done by Reddy RS *et al.* [8], Parveen S *et al.* [15], Katti T *et al.* [16] and Numbenjapon T *et al.* [17] in contrast to study by Khaleel KJ *et al.* [18] and Liqaa M *et al.* [19] showed group B being the majority. (Table 5)

Table 5: The distribution of TCP cases in various groups in comparison with other similar studies

Group	Reddy RS *et al.* [10]	Parveen S *et al.* [15]	Katti T *et al.* [16]	Numbenjapon T *et al.* [17]	Khaleel KJ *et al.* [18]	Liqaa M *et al.* [19]	Present study
A	80%	78.3%	66%	62.74%	12.9%	37.5%	75.3%
B	12%	21.7%	15%	37.26%	87%	44.2%	23.9%
C	12%	-	19%	-	-	18.2%	0.7%

In the present study group A included infections, neonatal TCPs, cardiac cases, pregnancy induced TCPs, renal and liver diseases, sepsis, burns and immune thrombocytopenias (ITP) accounting to 75.3% of which infections were the major entity similar to other studies [10-16]. In contrast, studies done by Numbenjapon T *et al.* [17] and Khaleel KJ *et al.* [18] found ITP being the majority of cases in group A. In the present study highest MPV values in group A category were seen in ITP cases similar to the other studies [10-24]. This increase in MPV is considered to be due to immune destruction of platelets leading on to accelerated production of larger sized platelets [10-24]. Group C category composed of congestive splenomegaly cases suggesting abnormal platelet pooling [1-7]. In the present study this category

attributed to the least number of cases 0.7% similar to other studies. This group also had higher MPV similar to group A possibly due to splenic pooling leading on to increased production of larger platelets [10-24].

Group B category included disorders associated with TCPs due to impaired platelet production from bone marrow. Our study showed 23.9% of cases in this group which included anemias, leukemias, post chemotherapy cases and aplastic anemia of which anemias were the predominant cases similar to other studies [10-17]. Conflicting results were seen in a study by Numbenjapon T *et al.* [17] who found leukemias and Myelodysplastic syndrome and a study by Khaleel KJ *et al*. [18] found postchemotherapy cases being the majority cases in group B. Compared to group A; group B showed lesser MPV values similar to the other studies. These decreased values are considered to be due to compensatory production of smaller aged platelets [10-18].

MPV values in group A, B and C were 10.15±1.30 fl, 8.52±1.69 fl and 10.6±1.25 fl respectively. Similar ranges of values were also seen in other studies [10, 15, 19, 20]. (Table 6). MPV values were statistically analysed and showed significant higher values in accelerated destruction group compared to impaired production group similar to the other studies [10-22]. These results exemplify the fact that in accelerated destruction group, bone marrow tries to compensate for the destructed platelets by actively producing larger younger platelets of varying sizes.

Table 6: MPV values in various groups among various studies

Studies	Reddy RS *et al.* [10]	Liqaa M *et al.* [19]	Parveen S *et al.* [15]	Gulati I. [20]	Present study
MPV-Group A	10.59±1.24	10.39±1.5	12.3±0.9	10.85 ± 1.63 fl	10.15±1.30
MPV-Group B	8.37±0.96	9.35±1.19	10.17±1.3	6.95 ± 0.74 fl	8.52±1.69
MPV-Group C	10.41±1.36	8.59±0.8	-	-	10.6±1.25

Thrombocytopenia being the commonest emergency dealt in everyday and platelet transfusion is the first choice of treatment [1-6]. But this initial treatment may not be helpful in certain causes of TCPs. Hence the challenging issue is to find out the cause in the shortest time available and to treat the patients accordingly. However it is not possible to gather all the necessary information and also an invasive bone marrow procedure to be done in short time to diagnose the etiology for TCPs [23-29]. Various other studies and also the present study reveals that MPV values to some extent can delineate the causes of TCPs. An eye on the routine analyser readings of MPV helps in better targeted therapies and preventive strategies [23-31].

Conclusion

MPV values showed significant higher values in accelerated destruction group compared to impaired production group. MPV is an easily obtained platelet parameter which is helpful in delineating the various causes of TCPs which inturn helps in the better targeted therapies. Hence MPV should be included in the diagnostic armamentarium together with other investigations for the evaluation of thrombocytopenia.

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