

Mean Platelet Volume in the Normal State and in Various Clinical Disorders

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Mean platelet volume (MPV) and platelet distribution width (PDW) were measured, using a Coulter counter (Model S-plus), in newborns, normal children, and healthy adults and in various clinical conditions. MPV was significantly increased in the patients with idiopathic thrombocytopenic purpura (ITP) and iron deficiency anemia (IDA), whereas in those with aplastic anemia and leukemia it was normal. The MPV of the patients with ITP decreased as the platelet count increased, and it became normal when the patients' platelet counts reached the normal range. In acute poststreptococcal glomerulonephritis (APSGN), renal failure and cyanotic congenital heart disease, the MPV was significantly increased. In the pregnant women with preeclampsia, the MPV showed a significantly higher value than in normal spontaneous vaginal delivery (NSVD), spontaneous premature rupture of the membranes (SPRM) and abortion. In the adults, with rheumatic heart disease, angina pectoris myocardial infarction and diabetes mellitus the MPV was significantly increased over that of the control group.

Key Words: Mean platelet volume (MPV), platelet distribution width (PWD).

Recent major advances in laboratory medicine have resulted from the employment of volumetric and morphometric cellular characterization in the diagnosis and management of disease.

With the introduction of the Coulter counter (Model S-plus), new hematologic parameters have become available to the clinician.

One of those is the mean platelet volume (MPV), which is comparable to the mean corpuscular volume (MCV) of red blood cells.

Determinations of platelet size are traditionally made by microscopic measurements of platelet diameters, a method which is not readily available in routine daily practice. The Coulter counter, however, provides an MPV on each whole blood sample that is processed, which makes possible the study of platelet size in a great variety of clinical conditions.

This study was designed to establish a reference value for MPV in normal Korean individuals to facilitate an evaluation of its significance in various clinical conditions.

MATERIALS AND METHODS

Fifty-four newborns, who were delivered at Severance Hospital, and 190 children who visited the Well baby clinic between January 1985 and March 1985 were studied as a normal child control group, with their parents informed consents. One hundred and sixty-seven members of the Severance Hospital staff who were found to be normal on routine physical examination, urinalysis, routine CBC, liver function test and chest X-ray were studied as a normal adult control group. The patients who were admitted to Severance Hospital between January 1984 and December 1984 were observed as a patient group.

All analyses were performed on blood specimens anticoagulated with K3 EDTA that were sent to the hematology laboratory for complete blood cell counts by the Coulter counter (Model S-plus III).

The Coulter counter (Model S-plus) incorporates analysis of erythrocyte and platelet volume distributions into the standard complete blood cell count. The counter's program postulates that all particles with volumes between 2 and 20 fl are platelets, and particles with volumes larger than 20 fl are erythrocytes. From diluted whole blood, the distribution of particle volume is measured, distribution histograms are generated for the two classes of cells, and an analysis

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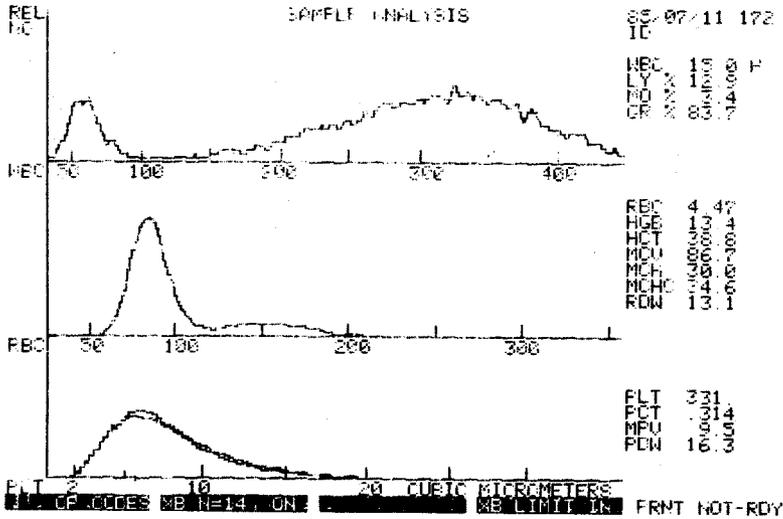


Fig. 1. WBC, RBC, and platelet histograms and counts.

MPV: Mean platelet volume

PDW: Platelet distribution width

is made. On the basis of this analysis of particle volume, the machine automatically prints out whole-blood platelet count, mean platelet volume, and an index of erythrocyte volume distribution, as well as the complete blood cell count generated by previous particle counters (leukocytes, erythrocytes, hemoglobin, hematocrit, and the erythrocyte indices) (Fig. 1).

RESULTS

The normal value of MPV in 54 newborns was 8.21 ± 0.65 fl. Infants and children whose ages ranged from 1 month to 48 months, had a mean MPV of 7.60 ± 1.26 fl. The mean MPV of 167 healthy adults was 8.11 ± 0.85 fl (Table 1). The mean MPV of the

Table 1. Platelet counts, MPV, and PDW in control group

Age	N	Platelet Count ($\times 10^3/\text{ul}$) (Mean \pm SD)	MPV (fl) (Mean \pm SD)	PDW (Mean \pm SD)
Newborn	54	292 \pm 56.9	8.21 \pm 0.65	17.03 \pm 0.71
Infant and child, mos	197	399 \pm 117.8	7.60 \pm 1.26	15.00 \pm 3.67
1	24	451 \pm 148.4	7.88 \pm 1.80	15.46 \pm 3.34
3	30	442 \pm 151.6	7.25 \pm 1.62	15.52 \pm 3.01
6	31	383 \pm 90.5	7.82 \pm 0.85	15.95 \pm 0.63
8	22	401 \pm 90.6	7.72 \pm 0.75	14.60 \pm 4.76
15	30	400 \pm 135.7	7.36 \pm 0.84	15.19 \pm 2.92
18	32	387 \pm 85.7	7.67 \pm 0.84	14.67 \pm 3.92
48	28	345 \pm 82.9	7.77 \pm 0.89	13.93 \pm 4.94
Adult	167	290 \pm 147.0	8.11 \pm 0.85	15.66 \pm 0.52

MPV: Mean platelet volume

PDW: Platelet distribution width

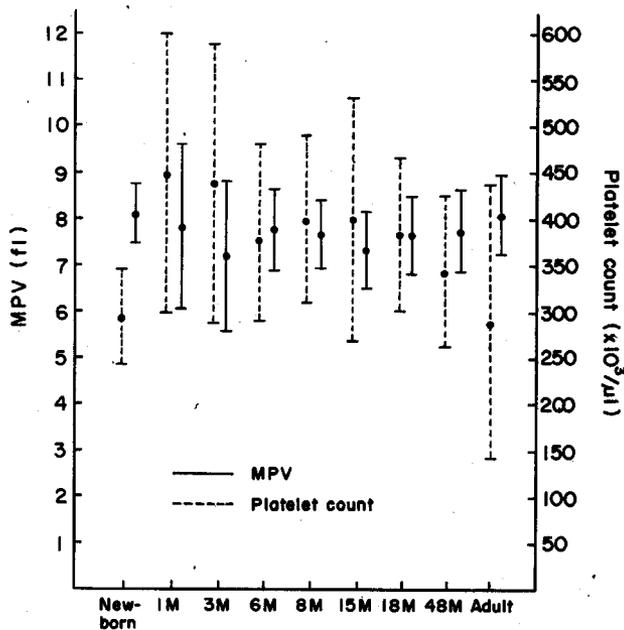


Fig. 2. MPV and platelet count in control group.

neonate and adult groups were significantly increased compared to that of the infant and child group ($p < .05$). In the infant and child group, the MPV ranged from 7.25 fl to 7.88 fl, but there were no significant differences in values, by age (Fig. 2).

In table 2 and figure 3 are shown MPV and PDW in children with various hematologic disorders. In acute ITP (20 cases), the mean MPV was increased

to 9.76 ± 1.18 fl, which was higher than that of the control group ($p < .01$). In chronic ITP (11 cases), the mean MPV was 11.12 ± 1.48 fl, which was significantly higher than that in either the control group or the acute ITP group ($p < .05$). In IDA (48 cases), the mean MPV was elevated. In aplastic anemia (13 cases), although it was the same for thrombocytopenia as for ITP, the mean MPV was not increased. The mean MPV in ALL (55

Table 2. Platelet counts, MPV, and PDW in children with various hematologic disorders

Disease	N	Platelet Count ($\times 10^3/\mu\text{l}$) (Mean \pm SD)	MPV (fl) (Mean \pm SD)	PDW (Mean \pm SD)
ITP				
acute	20	57 ± 26.1	$9.76 \pm 1.18^*$	$17.73 \pm 1.27^*$
chronic	11	67 ± 27.9	$11.12 \pm 1.48^*$	$18.08 \pm 1.07^{**}$
Aplastic anemia	13	31 ± 26.7	7.23 ± 0.80	16.17 ± 0.71
IDA	48	499 ± 171.5	$8.84 \pm 1.34^*$	$16.52 \pm 1.01^{**}$
ALL	55	291 ± 198.3	7.47 ± 1.16	$17.75 \pm 1.81^*$
AML	24	107 ± 70.6	7.48 ± 0.89	$17.92 \pm 2.05^*$

* $p < .01$ ** $p < .05$

ITP : Idiopathic thrombocytopenic purpura

IDA : Iron deficiency anemia

ALL : Acute lymphocytic leukemia

AML : Acute myelocytic leukemia.

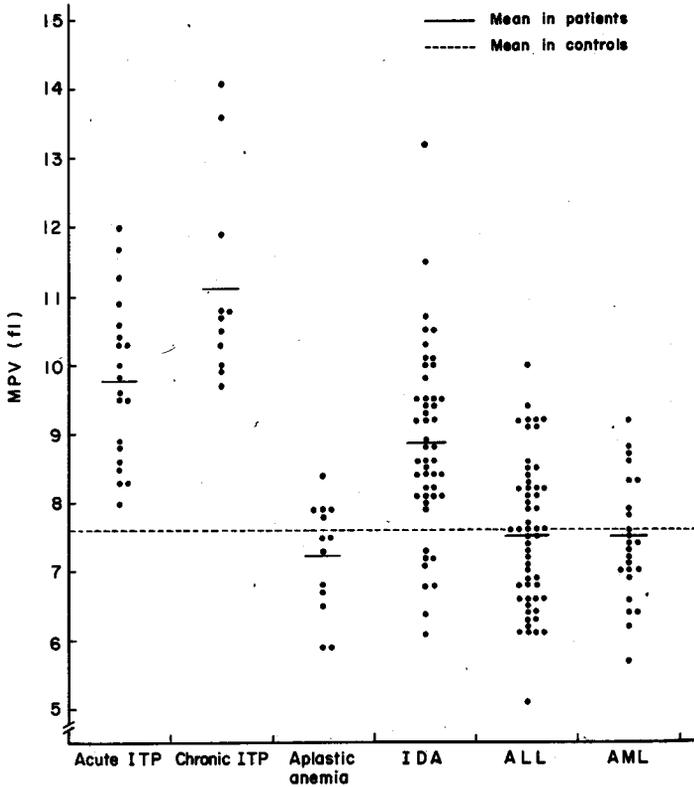


Fig. 3. MPV in children with various hematologic disorders.

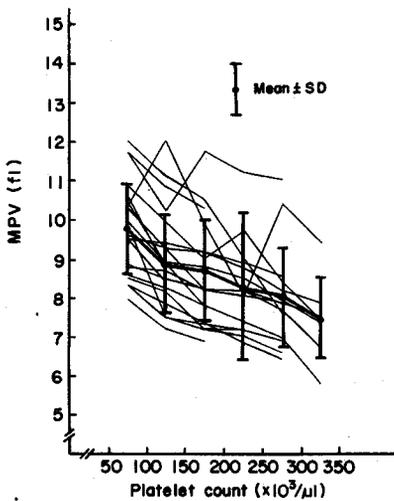


Fig. 4. Serial change of MPV according to platelet count in acute ITP.

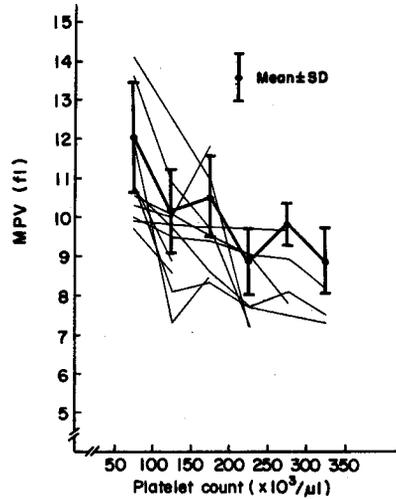


Fig. 5. Serial change of MPV according to platelet count in chronic ITP.

cases) and AML (24 cases) was not changed as compared to the control group.

In acute ITP, when the platelet count increased with treatment, the MPV decreased simultaneously.

When the patient's platelet counts returned to normal range, the MPVs did also (Fig. 4). In chronic ITP, the same tendency was noted (Fig. 5).

In APSGN (27 cases) and renal failure (11 cases),

Table 3. Platelet counts, MPV, and PDW in children with various clinical conditions

Disease	N	Platelet Count ($\times 10^3/\mu\text{l}$) (Mean \pm SD)	MPV (fl) (Mean \pm SD)	PDW (Mean \pm SD)
APSGN	27	326 \pm 113.6	8.51 \pm 0.79*	15.55 \pm 0.68
Nephrotic syndrome	11	443 \pm 157.5	8.05 \pm 1.11	15.82 \pm 0.65
Renal failure	11	308 \pm 172.6	8.84 \pm 2.29**	17.27 \pm 1.19
H-S purpura	26	438 \pm 166.9	7.98 \pm 0.82	15.56 \pm 0.49
CHD				
cyanotic	27	304 \pm 87.8	8.32 \pm 0.92**	16.62 \pm 1.74
acyanotic	29	325 \pm 107.2	7.59 \pm 0.91	15.61 \pm 0.88
Kawasaki disease	59	642 \pm 190.7	7.82 \pm 0.98	15.72 \pm 1.03

*p<.01 **p<.05

APSGN: Acute poststreptococcal glomerulonephritis

H-S purpura: Henoch-Schönlein purpura

CHD: Congenital heart disease

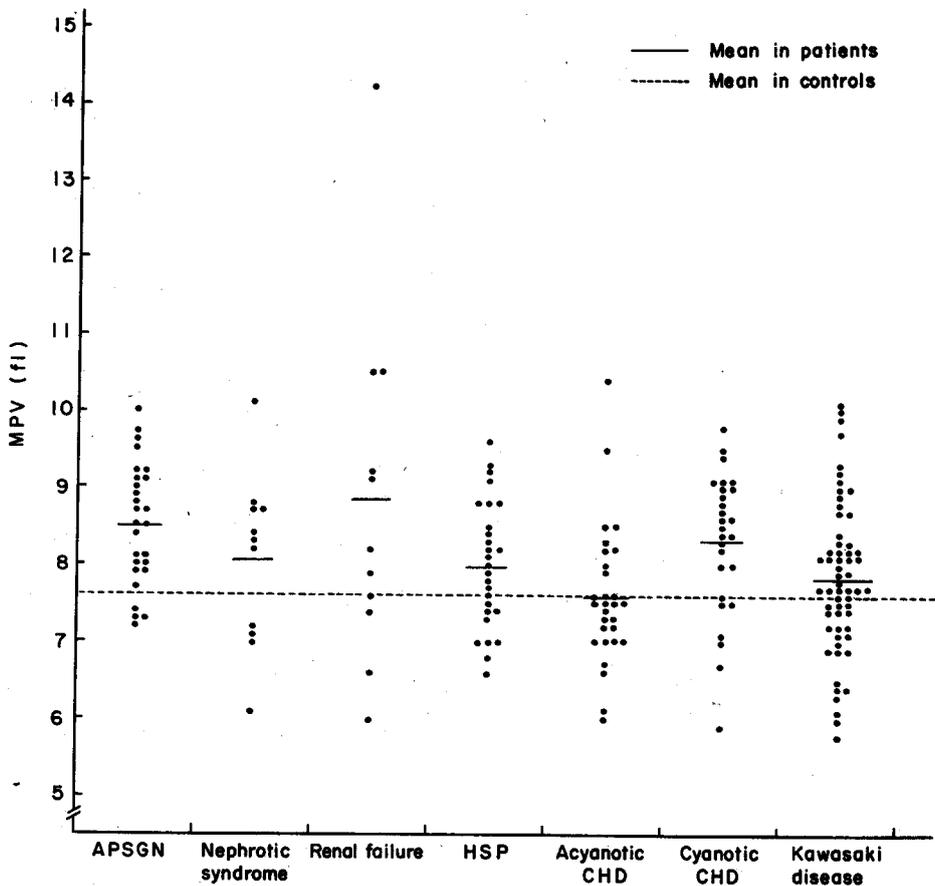


Fig. 6. MPV in children with various clinical conditions.

Table 4. Platelet counts, MPV, and PDW in pregnancy-related conditions

Disease	N	Platelet Count ($\times 10^3/\text{ul}$) (Mean \pm SD)	MPV (fl) (Mean \pm SD)	PDW (Mean \pm SD)
NSVD	75	284 \pm 102.2	8.45 \pm 1.21	16.67 \pm 1.18*
Abortion	37	281 \pm 107.6	8.76 \pm 0.88**	16.31 \pm 1.53*
SPRM	40	279 \pm 79.6	8.96 \pm 1.23**	16.37 \pm 1.31*
Preeclampsia	51	290 \pm 128.9	9.66 \pm 1.73*	16.87 \pm 1.41*

p<.001 **p<.01

NSVD: Normal spontaneous vaginal delivery

SPRM: Spontaneous premature rupture of the membranes

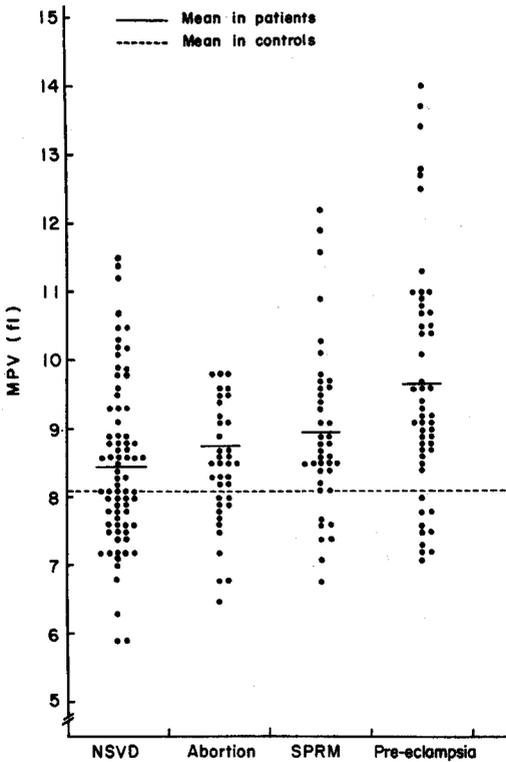


Fig. 7. MPV in pregnancy – related conditions.

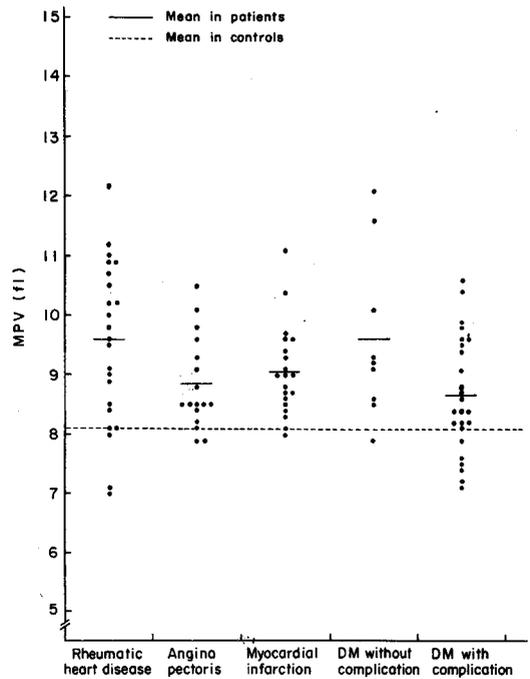


Fig. 8. MPV in adults with various clinical conditions.

the mean MPVs were significantly increase ($p<.01$) (Table 3 and Fig. 6).

In cyanotic congenital heart disease, the mean MPV was increased over that of the control group, and in the acyanotic congenital heart disease group ($p<.05$) (Table 3 and Fig. 6).

In the pregnancy-related conditions, the MPV was as follows: normal in normal spontaneous vaginal delivery, but increased in abortion, spontaneous premature rupture of the membranes and in

preeclampsia (Table 4). Comparing the MPV in preeclampsia with that in other pregnancy-related conditions concerned (NSVD, SPRM, and abortion), the MPV in preeclampsia was significantly higher (Fig. 7).

In the adults with rheumatic heart disease (19 cases), the MPV was 9.59 \pm 1.40 fl; angina pectoris (17 cases), 8.84 \pm 0.78 fl; myocardial infarction (20 cases), 9.06 \pm 0.76 fl; and diabetes mellitus (34 cases), 8.91 \pm 1.17 fl. In other words, the MPV in these was

Table 5. Platelet counts, MPV, and PDW in adults with various clinical conditions

Disease	N	Platelet Count ($\times 10^3/\text{ul}$) (Mean \pm SD)	MPV (fl) (Mean \pm SD)	PDW (Mean \pm SD)
Rheumatic heart disease	19	217 \pm 59.2	9.59 \pm 1.40*	16.13 \pm 0.89**
Angina pectoris	17	287 \pm 60.3	8.84 \pm 0.78**	15.89 \pm 0.38**
Myocardial infarction	20	313 \pm 66.3	9.06 \pm 0.76**	15.88 \pm 0.68
DM	34	268 \pm 86.5	8.91 \pm 1.17*	16.31 \pm 0.54*
(with complications)	9	252 \pm 55.1	9.60 \pm 1.42*	16.15 \pm 0.42*
(with complications)	25	274 \pm 95.6	8.66 \pm 0.98**	16.37 \pm 0.58**

* $p < .001$ ** $p < .05$

DM: Diabetes mellitus

greater than the MPV in the control group (Table 5 and Fig. 3).

DISCUSSION

In the past, the difference in platelet size has been observed on peripheral smear or wet preparation, but an accurate platelet volume could not be obtained. Minister *et al.* (1967) tried to represent platelet volume by the density gradient centrifugation method, but this method has not been utilized in clinical medicine because of the technical difficulties involved.

Recently, an automated blood analyzer, the Coulter counter (Model S-plus), has been introduced. It has made it possible to determine platelet size from a routine specimen of blood collected for a cell count. Platelet size, as measured by this counter, is expressed as mean platelet volume (MPV).

A high MPV indicates the presence of generally larger platelets and, when the MPV is low, platelets are generally smaller.

Studies of circulating platelets, however, indicate that these cells are heterogeneous in size; density; and metabolic, functional, and biochemical properties (McDonald *et al.* 1964; Booyse *et al.* 1968; Karpatkin 1969; Nakell *et al.* 1970; Minster *et al.* 1971; Berg 1972).

Platelet size is heterogeneous even in normal persons; its heterogeneity is, however, increased in patients with idiopathic thrombocytopenic purpura, sepsis, DIC, myocardial infarction and diabetes mellitus (Garg *et al.* 1971; Karpatkin *et al.* 1971; Paulus 1975; Mustard *et al.* 1977; Roper *et al.* 1977; Zeigler *et al.* 1978).

Often seen are the patients with aplastic anemia whose hematologic abnormalities are mild, for example, the patients have remarkable thrombocytopenia, but the intensity of anemia and leukopenia have remained slight for many months. In such a situation, there are difficulties in differentiating between a diagnosis of chronic ITP or mild aplastic anemia, or an early stage of aplastic anemia, without resorting to invasive studies. Furthermore, the differential diagnosis between acute ITP and chronic ITP in childhood at the first visit is not always easy if no history of a preceding infection that could have caused the thrombocytopenia can be found or if the time of onset of bleeding episodes is obscure.

Diagnosis has often been made retrospectively for such patients, as it has for acute or chronic type ITP. Platelet survival studies and immunological studies to discriminate between these and thrombocytopenia but doing these tests is a time-consuming procedure. Therefore, we tried to investigate the usefulness of MPV as a means of early differential diagnosis of these different kinds of thrombocytopenias.

As shown in Table 2 and Figure 3, striking contrast was found in the platelet volume among these three diseases. For example, the differences found in the MPV of the patients with aplastic anemia and chronic ITP were apparent in even early stage, and it was shown that the chronic and acute idiopathic thrombocytopenic purpuras themselves could easily be distinguished from each other by an analysis of the MPV. On the other hand, no differences were found in the MPV among the central thrombocytopenic diseases, such as aplastic anemia, ALL, and AML as those diseases were seen in children which was as they are usually seen.

The platelets of aplastic anemia were relatively small. It is probable that the number of old platelets may increase in the peripheral circulation in this disease, or these platelets may represent a platelet population which is abnormal and aberrant due to defective thrombopoiesis.

In acute leukemia, the mode of the platelet volume was low, and MPV also tended to be low. MPV and PDW remained unchanged from what they were in the pretreatment phase. This fact may show that platelet thrombopoiesis, or function, in acute leukemia does not always normalize completely (Tomita *et al.* 1980).

Comparing the MPV with various pregnancy-related conditions, the MPV in preeclampsia showed significantly higher value than in NSVD, SPRM, and abortion. This finding is almost the same as previously reported (Yoon *et al.* 1984).

The prevalence of an increased MPV in diabetes mellitus, with or without complications, presumably indicates an increased rate of platelet turnover.

To our surprise, there was less increase in MPV in the group with myocardial infarction the cases of which were possibly more were than the cases of rheumatic heart disease.

Our study suggested the usefulness of measuring the MPV in clinical disorders in which an increased rate of platelet turnover is suspected.

CONCLUSION

From these studies, the MPV is significant as a screening test in determination of platelet status with platelet counts. Especially in thrombocytopenia, the MPV was increased in peripheral thrombocytopenia such as ITP, but not changed in central thrombocytopenia such as aplastic anemia, ALL, and AML. So we can discriminate between the two types of thrombocytopenia by the MPV and suppose that the MPV may represent a good indicator of thrombopoiesis as well as reticulocytes in erythropoiesis.

In acute ITP and chronic ITP, the MPV is significantly different. We believe that the ITP, provides a good weapon in early differential diagnosis of acute and chronic ITP, nevertheless, further study is needed.

In cyanotic congenital heart disease, there was no difference in platelet counts compare to acyanotic

congenital heart disease, but the MPV was significantly increased. From these, we assume the increased MPV plays a role in vascular complication such as thrombosis.

The MPV in preeclampsia showed a significantly higher value, so it can be used as a parameter in the diagnosis of preeclampsia.

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