

Mean platelet volume and plateletcrit in the acute ischemic stroke

Taşkın DUMAN¹, Ferhan KÖMÜRCÜ¹, Şeref KÖMÜRCÜ², Sabahat GÜRÇAY¹

¹Dept. of Neurology, Ankara Numune Hospital,

²Dept. of Internal Diseases Gülhane Military Hospital, Ankara, TURKEY

Trombocyte activation has a major role in the development of ischemic cerebrovascular disease. Mean Platelet Volume (MPV) is related with the unmetabolized secretion of the trombocytes. An increase in MPV will cause increased trombocyte activity, agrégation and serotonin secretion. The aim of this study was to investigate MPV and plateletcrit values in the acute phase of ischemic stroke. This study aimed to investigate the MPV and plateletcrit in the acute ischemic stroke and to examine their changes in the acute period. We performed this study on 33 patients who previously had no stroke or who had a cerebral infarct in their tomography which was taken after 48 hours when a stroke began. The evaluations in the 1st, 3rd and 10th day of the stroke and the changes of the obtained data with respect to the days were examined and compared with the data of the contrôle group. At the end of the data evaluation in terms of the days, an increase in MPV was found in the 3rd day ($p<0.001$). The MPV change in the 3rd day was more significant in ex-patients with respect to non ex-patients ($p<0.05$). There was no difference between the 1st and 3rd day, the 1st and 10th day in the plateletcrit data ($p<0.05$). A negative correlation was determined between the platelet number and MPV in the 1st ($p<0.001$) and 10th ($p<0.001$) day. The correlation between the trombocyte number and plateletcrit was positive in the 1st, 3rd and 10th ($p<0.001$). It can be thought that the MPV increase determined in the acute stroke may be dependent on platelet activation that is related with the stroke. [Turk J Med Res 1994; 12(6) 249-252]

Key Words: Stroke, Platelets

Increased platelet activity has a major role in the cardiac and cerebrovascular diseases. During the evaluation of trombocyte function, in different periods of acute ischemic stroke, trombocyte agrégation, circulating trombocyte aggregates and marker proteins have been used as clinical parameters in previous reports. Moreover there are some reports that indicate that activation has not only have a major role in the development of ischemic stroke but its genesis as well. Because of the relation between the volume and the degree of platelet activation, MPV must be considered in the ischemic cerebral diseases. Automated measurements of the trombocyte parameters will be helpful in the evaluation of physiopathological and clinical results after the cerebral infarct. This study is devoted for the investigation of MPV and plateletcrit values in the acute phase of ischemic stroke.

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Correspondence: Taşkın DUMAN

Ankara Numune Hospital
Clinic of Neurology,
Ankara, TURKEY

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MATERIALS AND METHODS

In this study 33 adult (42<age<92; 67.12±10.86 years) patients (17 female-16 male) were investigated. They had thromboembolic cerebrovascular disease according to CT and clinical findings. The patients who previously had infection, malign and hematological disorder or stroke and myocard infarct were all excluded. Moreover those who had therapy which had an effect on the trombocyte function were not investigated as well.

Antecubital blood samples were obtained on the 1st, 3rd, and 10th day of the stroke. They were all collected in a styrene tubes having EDTA as an anticoagulant. Sereno System 9000 Counters was used for the MPV, trombocyte and hematocrite counting. Also, plateletcrit was calculated. Two specimens which belong to the first day were hemolyzed so they were excluded. Therefore, 31 dates were available for the 1st day. The data about the 10th day was only 19 because 6 patients died and 8 samples were not available for the tests. The dates which belong to the alive patients and the others were compared.

The control group of this study was consisted of 26 healthy subjects (15 male, 11 female, 41<age<76).

Student t test, correlation analysis and paired t-test were used for the statistical evaluations.

RESULTS

The average results of the patient and control group were given in Table 1. The results of the study and control group were also summarized in Table 2 and 3 respectively.

The lowest average platelet count were observed on the 3rd day. The average platelet counts on the 1st, 3rd and 10th day were lower than the control group. Finally the results related to the 3rd day was statistically significant ($p < 0.05$).

Increased MPV value was observed on the 3rd day ($p < 0.001$) but the average MPV decreased on the

10 day and the difference was disappeared ($p > 0.05$). Although the MPV values of the 1, 3 and 10th day were lower with respect to control group, the difference was statistically insignificant. During the study period the 6 patients who died had a significant different MPV value between the 1st and 3rd days (-0.7 ± 0.66 ex group, -0.21 ± 0.37 in the alive group $p < 0.05$).

The plateletcrit values in the 1-3 and 1-10 did not have a significant differentiation ($p > 0.05$). The 3rd day parameter was lower than the control group ($p < 0.05$). Significant negative correlation was observed between the platelet count and MPV values; 1st day ($r = -0.4516$, $p < 0.01$), 3rd day ($r = -0.5232$, $p < 0.001$) and 10th day ($r = -0.7025$, $p < 0.001$). A positive correlation was existed between the platelet counts and plateletcrit; 1st day

Table 1. Platelet, MPV and plateletcrit averages of patient and control groups.

	1st day (average)	3rd day (average)	10th day (average)	Controle group (average)
Trombocitex $10^3/mm^3$	255.90+88.02	233.55+76.85	247.63+71.94	280.12+99.26
MPVfl	8.27+0.90	8.52+0.93	8.28+1.22	8.62+1.13
ptc%	0.21+0.06	0.20+0.06	0.20+0.04	0.24+0.09

Table 2. The plateletcrit, hematocrit, platelet and MPV values in the 1 st, 3rd and 10th day of the patient group.

Patients Number	Clns	Prog- nose	Plateletcrit			Hematocrit			Trombocyte			MPV		
			1.day	3.day	10.day	1.day	3. day	10.day	1.day	3.day	10.day	1.day	3.day	10.day
1	71	M	0.2014	0.1340	0.2426	37.1	34.5	31.8	255	205	337	7.9	8.0	7.2
2	66	M	0.1513	0.2365	46.3	48.9	44.2	189	161	285	9.3	9.4	8.3	
3	69	F	0.1424	0.1710	0.2261	39.4	36.2	39.7	178	225	266	8.0	7.6	8.9
4	56	M	0.2023	0.1759	-	38.6	39.1	-	238	212	-	8.5	8.3	-
5	75	F	-	0.1779	0.1512	-	47.2	38.0	-	217	189	-	8.2	8.0
6	42	F	0.3123	0.3323	-	41.8	35.4	-	351	391	-	8.9	8.5	-
7	61	M	ex 0.1776	0.1637	-	47.9	49.8	-	209	184	-	8.5	8.9	-
8	48	M	0.1932	0.1883	-	46.6	43.3	-	230	219	-	8.4	8.6	-
9	55	M	ex 0.3191	0.2400	-	37.2	41.0	-	541	381	-	5.9	6.3	-
10	63	M	-	0.1897	-	-	37.9	-	-	275	-	-	6.9	-
11	76	F	ex 0.1405	0.2610	-	36.8	43.1	-	273	348	-	6.6	7.5	-
12	67	M	0.2268	0.1711	0.1310	46.1	50.0	36.2	210	157	113	10.8	10.9	11.6
13	64	M	0.1170	0.1284	-	37.7	39.5	-	150	146	-	7.8	8.8	-
14	75	F	ex 0.2108	0.1426	-	40.1	46.7	-	251	164	-	8.4	8.7	-
15	53	M	ex 0.3712	0.2440	-	26.0	31.1	-	476	249	-	7.8	9.8	-
16	56	F	0.2614	0.2987	-	40.7	40.1	-	344	383	-	7.6	7.8	-
17	70	M	0.1363	0.1740	0.1836	40.7	45.1	47.0	192	229	287	7.1	7.6	6.4
18	92	F	0.1989	0.1833	0.2172	36.5	35.0	38.0	203	178	224	9.8	10.3	9.7
19	65	F	0.2123	0.2888	-	28.8	33.2	-	259	332	-	8.2	8.7	-
20	85	F	0.1966	0.1740	0.2371	46.2	45.6	48.1	221	200	279	8.9	8.7	8.5
21	66	M	0.2312	0.1365	0.2697	47.8	48.0	49.8	282	157	329	8.2	8.7	8.2
22	79	M	0.1634	0.1161	0.1080	52.4	49.6	49.3	190	132	135	8.6	8.8	8.0
23	63	F	0.2304	0.1952	0.1495	42.4	41.1	47.7	281	227	178	8.2	8.6	8.4
24	67	F	0.1601	0.1450	0.1420	42.0	45.9	47.1	176	145	134	9.1	10.0	10.6
25	55	M	0.1864	0.1880	0.1984	43.1	44.7	44.8	233	235	242	8.0	8.0	8.2
26	67	M	0.1986	0.1869	0.2117	44.4	44.4	47.2	258	246	290	7.7	7.6	7.3
27	70	M	0.1109	0.1223	-	46.1	49.5	-	129	133	-	8.6	9.2	-
28	85	F	0.3024	0.1921	0.1754	36.2	33.7	31.1	378	226	274	8.0	8.5	8.2
29	61	F	0.2265	0.3444	0.2310	30.7	35.7	33.0	249	387	304	9.1	8.9	7.6
30	70	F	ex 0.1969	0.1610	-	37.0	39.6	-	229	181	8.6	8.9	-	
31	66	F	0.2480	0.2261	0.1871	40.8	41.6	35.4	310	266	231	8.0	8.5	8.1
32	77	F	0.1853	0.2187	0.2304	38.8	41.5	39.8	226	270	320	8.2	8.1	7.2
33	80	F	0.2143	0.1918	0.1810	40.0	38.0	38.1	282	246	348	7.6	7.8	7.4

Table 3. The values of plateletcrit, hematocrit, platelet and MPV in control groups

Row no.	Age	Sex	Plateletcrit %	Htc %	TrombocitexlO ³ mm ³	MPV (fl)
1	48	E	0.3549	42.3	408	8.7
2	45	E	0.2141	46.3	166	12.9
3	43	E	0.2936	48.4	367	8.0
4	76	E	0.4655	38.5	529	8.8
5	62	K	0.4428	47.0	492	9.0
6	52	K	0.1942	42.0	234	8.3
7	57	E	0.1672	55.0	204	8.2
8	65	K	0.3302	47.3	384	8.6
9	41	K	0.2157	37.1	232	9.3
10	45	E	0.2666	53.3	303	8.8
11	70	E	0.2303	48.8	245	9.4
12	61	K	0.2335	41.3	229	10.2
13	55	E	0.2180	44.5	232	9.4
14	48	E	0.1859	44.4	224	8.3
15	44	E	0.2693	51.2	402	6.7
16	44	E	0.2448	59.5	255	9.6
17	46	K	0.2571	44.2	299	8.6
18	49	E	0.2807	49.9	319	8.8
19	41	E	0.2803	48.1	326	8.6
20	46	K	0.2358	42.6	268	8.8
21	52	K	0.1396	39.7	179	7.8
22	56	K	0.1861	37.5	214	8.7
23	70	K	0.1550	37.1	204	7.6
24	52	K	0.1396	39.7	179	7.8
25	50	K	0.1886	39.3	245	7.7
26	65	E	0.1035	40.4	138	7.2

($r=0.9230$, $p<0.001$), 3rd day; $r=0.928$, $p<0.001$) and 10th day ($r=0.8357$, $p<0.001$). However a significant variation was not observed in the hematocrite.

DISCUSSION

The volume of the platelet and its heterogeneity is related with its degree of stimulation (1). Moreover there are some reports which indicate that MPV can be detected before the fragmentation of megacaryocites to the platelet and, also by aging the dimensions of the platelet don't change (2). The number of platelet and MPV are under independent hormonal control mechanisms (3). By evaluating the platelet activation with the conventional tests, the large platelet were detected to be more active (4,5). The big platelet aggregate more rapidly as a result of the influence of ADP and collagens (6) and they can produce prothrombic factors more (7,8). Also the serotonin secretion is different (9,10). Moreover the secretion of arachidonic acid from the platelet are related with MPV (8).

Studies dealing with the evaluation of the platelet functions; thrombocyte aggregation, the circulating platelet aggregates or marker proteins proved that the platelet were hyperactive during the acute phase of the stroke (11-14). Some of the studies suggested that platelet aggregation took place, after 2-3 days of the stroke (15-16). Tohgi et al have reported that the MPV, platelet and plateletcrit values were decreased with

respect to the controls (17). D'Erasmo reported the decreased platelet number but increased MPV values. MPV values were seemed to be normal on the 4-5th day while platelet were 9th day. Their difference were statistically significant. But for plateletcrit the situation was rather different. It has started to increase on the 4-5th day and then remained to be constant (18), but this difference was insignificant. In the morphological evaluation of the thrombopoietic function, the relation between the platelet number and MPV was in accordance (19) while the thrombocyte number and its volume had negative correlation (18-23). Our finding support this item.

The platelet account of the patient group was lower than the control group and they had a significant difference on the third day in our study. While MPV values were lower than the control and had an insignificant difference. An increase in MPV was observed on the 3th day. It declined and on the tenth day they were in the same place as in the beginning. The plateletcrit was lower than the control, this phenomenon could be explained by the variation in the platelet count and MPV values on the 3th day. The difference in our finding and previous studies could be due to the timing of the biological material. In one of the invitro studies, dogs with reduced cerebral blood flow were subjected and the observed platelet accumulation was in the lesions with focal ischemia (24). As a result of

this study it could be concluded that the increase of MPV in acute stroke was related with the platelet activation during stroke.

Akut iskemik strokta platelekrit ve mean trombosiz volümü

İskemik serebrovasküler hastalığın gelişiminde trombosit aktivasyonunun önemli rol oynadığı bilinmektedir. Trombositlerin, metabolize olmamış arakidonik asit salınım yeteneği, mean platelet volume (MPV) ile ilgilidir. MPV arttıkça trombositlerin aktivitesinin arttığı, özellikle agregasyon yeteneklerinin ve serotonin salınımının fazlalaştığı bilinmektedir. Trombosit fonksiyonlarının bir göstergesi olarak kabul edilen MPV'nin iskemik strokun akut dönemindeki değişiminin bilinmesinin bu dönemdeki trombosit aktivitesini değerlendirmede yararlı olacağı düşünülebilir. Bu çalışma akut iskemik strokta MPV ve platelekriti değerlendirmek ve bunların akut dönemdeki değişimini incelemek amacıyla yapıldı. Çalışmada daha önce strok geçirmemiş olan ve strok başlangıcından itibaren 48 saat sonra çekilen tomografide serebral infarkt tespit edilen 33 hasta değerlendirildi. Strokun 1., 3. ve 10. günlerinde yapılan değerlendirmeler ile elde edilen verilerin günlere göre değişimi incelendi ve kontrol grubuna ait verilerle karşılaştırıldı. Hasta grubunun verilerinin günlere göre değerlendirilmesi sonucunda MPV'de 7.güne göre 3.günde yükselme bulundu ($p<0.001$). Ölen hastalarda 3.gündeki MPV değişimi ölmeyenlere göre daha belirgindi ($p<0.05$). Platelekrit değerlerinde 7.gün ile 10.gün arasında fark yoktu ($p>0.05$). Trombosit sayısı ile MPV arasında 1.günde ($p<0.01$) 3.günde ($p<0.001$) ve 10.günde ($p<0.001$) negatif korelasyon vardı. Trombosit sayısı ile platelekrit arasında 1. gün, 3.gün ve 10.günde pozitif korelasyon vardı ($p<0.001$). Akut strokta belirlenen MPV artışının strokla ilgili trombosit aktivasyonuna bağlı olduğu düşünülebilir.

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