Mean Platelet Volume may be Reflects the Disease Activity of Ulcerative Colitis

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ABSTRACT

Aim: To determine the relation between the disease activity of ulcerative colitis and mean platelet volume (MPV) and to evaluate the clinical usage of a more simple and easier determinant.

Method: Complete blood count, C-reactive protein (CRP), erythrocyte sedimentation ratio (ESR), serum albumine and prothrombine time were measured in a total of 41 ulcerative colitis patients. The clinic, pathologic, colonoscopic disease activities of ulcerative colitis and the localisation of colonic involvement were designated. Truloeve-Witts score was used for the clinic activity of ulcerative colitis.

Result: Eleven patients were in remission (26.8%) and 30 had active disease (73.2%). The mean values were; age: 44.58±15.08 year (20 women, 21 men), CRP: 36.80 ± 32.90 mg/L, ESR: 52.29 ± 31.23 mm/h, albumin: 3.43 ± 0.65 gr/dl, platelets: 400780 ± 161196 K/mm3, MPV: 7.41±1.04 fentoliter (fl), prothrombine time: 13.32 ± 1.05 second, respectively. By the correlation analysis there was a negative significant relation between CRP and MPV (p<0.05). The mean MPV values of the 11 patient in remission were 8.62 ± 1.15 fl. The mean MPV values of the 30 patients who had active disease were 6.97 ± 0.53 fl. The patients having active ulcerative colitis had lower MPV values when compared with the patients who had inactive disease (in remission) (p<0.001). There were negative significant relations between MPV and clinical, pathological aand colonoscopic activity indices (p<0.001). However there was no relation between the extent of the disease involvement and MPV.

Conclusion: MPV can reflect the disease activity of ulcerative colitis.

Key words: Ulcerative colitis, mean platelet volume, C-reactive protein

Ortalama Trombosit Hacmi Ülseratif Kolit Hastalık Aktivitesini Yansıtabilir

Amaç: Ülseratif kolit (ÜK) hastalık aktivitesi ile ortalam trombosit hacmi (OTH) arasındaki ilişkiyi belirlemek ve daha basit ve kolay elde edilebilir bir belirteçin klinik kullanımını değerlendirmektir.

Metod: Toplam 41 ÜK'li hastada tam kan sayımı, C-reactive protein (CRP), eritrosit sedimantasyon hızı, serum albumini ve protrombin zamanı ölçüldü. Bütün hastalarda ÜK klinik, patolojik, kolonoskopik hastalık aktiviteleri ve ÜK kolonik tutulum lokalizasyonları belirlendi. ÜK aktivitesi Truloeve-Wihts skoruna göre yapıldı.

Bulgular: ÜK'li hastanın 11 tanesi remisyonda (%26,8) ve 30 hastada aktif hastalık vardı (%73,2). Hastaların ortalama değerleri sırası ile, yaş: 44,58±15,08 yıl (20'si kadın 21'i erkek), C-reactive protein: 36,80±32,90 mg/L, ESR: 52,29±31,23 mm/saat, Albumin: 3,43±0,65 gr/dl, Trombosit: 400780,5±161196,2 K/mm3 Ortalama trombasit hacmi (OTH): 7,41±1,04 fentolitre (fl), PTZ: 13,32±1,05 saniye idi. Korelasyon analizinde CRP ile OTH arasında negatif yönde anlamlı ilişki vardı (p<0,05). Remisyonda olan 11 hastanın ortalama OTH'si 8,62±1,15 fl idi. Aktif hastalığı olan 30 hastanın ortalama OTH'si 6,97±0,53 fl idi. Aktif ülseratif koliti olan hastaların OTH değerleri, inaktif hastalığı (remisyonda) olan hastaların değerleri karşılaştırıldığında belirgin olarak daha düşük bulundu (p<0,001). OTH ile klinik, patolojik ve kolonoskopik aktivite indeksleri arasında negatif anlamlı ilişki vardı (p<0,001). Ancak OTH ile ülseratif kolit hastalığının tutulum yaygılığı arasında ilişki yoktu.

Sonuç: OTH , ülseratif kolit hastalık aktivitesini yansıtabilir.

Anahtar kelimeler: Ülseratif kolit, ortalama trombosit hacmi, C-reactive protein

INTRODUCTION

Inflammatory bowel disease(IBD) is a systematic inflammatory disease which can not only affect gastrointestinal system but also affect our all organs and aetiology of which is not clearly known. Many studies show that platelets also play role in IBD physiopathology (1,2). Platelets have low capacity for protein synthesis. Interestingly they show all functions of the cell though they don't include nucleus and DNA (3). Mean platelet volume (MPV) the measurement of which had some difficulties in the before years can be measured today very easily with automatic devices. It is seen that in the situations in which thrombopoiesis increases, depending on the increase in young platelets transferred into the circulation from the bone marrow, MPV also increases (4). It is seen that in serious thrombocytopenia the low MPV becomes in correlation with bleeding episodes susceptibly (5). As expected, big platelets including denser granules carry much more biochemical, functional and metabolic abilities. The increase in MPV namely big voluminous in peripheral blood are related with megakaryocyte number in the bone marrow as well (4). In normal, platelets are heterogeneous when they are free from megakaryocytes and since all of them are the same, they are not big and dense and also they can be small and less dense (4). If there is a reason that stimulates platelet production in the bone marrow, MPV not only increases but also platelet distribution width increases. This situation is used in discrimination and diagnosis of many diseases. For example, MPV and platelet distribution width bear importance in the discrimination of myeloproliferative diseases, essential thrombocytosis

and reactive thrombocytoses (6,7). Many studies on clinical benefit of MPV were made on many subjects such as splenectomy, microcytic anemia, serebral infarct (8-10), thrombocytopenia (11,12), thrombocytoses (6,7), congenital platelet diseases, sepsis, chronic obstructive lungal diseases (13), chronic venous failure (14), high MPV in hyperthyroidism (15), low MPV in hyperthyroidism (16), hypertension and preeclampsia in pregnanacy, organic solvent toxicity.

On the other hand it was shown that in the increased platelet number in the blood had direct proportion to the disease tension in IBD (17,18). This situation can be speculated in this way with that its reason can't be known absolutely: In IBD, platelets have been increasing in peripheral blood as a reaction to systemic inflammatory response. Also, the increased platelet number is responsible for systemic thromboembolism seen in IBD and for intestinal micro-infarcts (19-23). In many studies it was documented that platelets increase systemic inflammatory response by secreting inflammatory mediators in IBD (18-24). While it is observed platelet number in IBD is affected, it is shown MPV decreases (25-27). Contrary to this, MPV increase in peripheral blood has direct proportion to platelet function (28). The increase in MPV, being related to the increase in the disease activity in some clinical states, was shown in various clinical situations such as preeclampsia, myocardial infarct and unstable angina pectoris (9,10,14).

Our aim in this study is to determine the interaction between the disease activity of ulcerative colitis patients in our own population (Turkish patients) and MPV, and to

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	Patients	
	(n:41)	
I.phase	2 (4.9%)	
II.phase	12 (29.3%)	
III.phas	e 27 (65.9%)	

Table 1. Ulcerative colitis pathological activity	
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evaluate a very simple parameter which can show the disease activity and then to interpret its use in clinical practise.

MATERIALS AND METHODS

The total 41 ulcerative colitis patient monitored in gastroenterology clinic were taken into the study. In all patients, diagnosis was given with radiological, endoscopic and histological study. None of patients had medicinal use (e.g. anticoagulations, aspirin, contraceptive and nonsteroidal anti-inflammatory medicines) which can make platelet and coagulation abnormality in the blood of 8 weeks. Also, as exemption criteria, those patients were not taken to the study: ones having abnormal liver and renal function test, ones having myeloproliferative disease, ones having malignancies and ones having any haematological disease which can be affect platelets. In all patients, total blood counting, C-reactive protein (CRP), erythrocyte sedimentation speed, serum albumin and prothrombin time were measured. In all patients, ulcerative colitis clinical, colonoscopic disease activities and ulcerative colitis tied localizations were determined. Ulcerative colitis clinical activity was made according to Truloeve-Wihts score. 11 of 41 ulcerative colitis patients as ulcerative proctitis 10 (24.4%), distal colitis 9 (22.0%), left colitis 6 (14.6%), common colitis 3 (7.3 %), pancolitis 13 (31.7 %) were in remission (26.8 %) and 30 of them had active disease (73.2 %). For platelet number and MPV measurement: platelet number (per mm3) and MPV (fentoliter (fl)), blood samples were taken from fore hand into EDTA tubes by applying a minimal stasis from antecubital vein.

Table 3. Ulcerative colitis clinical activity(according to Truloeve-Wihts score)

	MPV
Disease in remission (n:11)	8.6±1.2 fl (6.3-10.2)
Active disease (n:30)	6.97±0.53 fl (5.8-8.7)

Table 2. Ulcerative	colitis	colonoscopic	activity grade
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	Patients (n:41)
I.grade	2 (4.88%)
II.grade	12 (29.27%)
III.grade	15 (36.88%)
IV.grade	12 (29.27%)

Blood samples were treated with automatic standard hemogram device.

Statistical Analysis

RESULTS

Mean values of patients were age: 44.58±15.08 years (20 women, 21 male), C-reactive protein: 36.80±32.90 mg/L, ESR: 52.29±31.23 mm/hour Albumin: 3.43±0.65 gr/dl Platelet: 400780.5±161196.2 K/mm3 MPV: 7.41±1.04 fL (normal=7.2-11.1) PTZ: 13.32±1.05 seconds. In the correlation analysis there was a negative directional meaningful relation between CRP and MPV (p<0.05). Mean MPV of 11 patients being in remission was 8.62±1.15 (median: 8.62 min: 6.29 max:10.20). MPV values of patients having active ulceratice colitis, when compared with values of patients having anactive (in remission), were found quite lower (p<0.001). There was a negative meaningful relation between MPV and clinical (r:-0.608; p<0.001), pathological (r:-0.523; p=0.001) and colonoscopic activity indexes (r:-0.441; p=0.005).

However there was not any meaningful relation between MPV and ulcerative colitis capturing prevalence and other observed parameters. Moreover there was an inverse proportional relation between MPV and the increase in platelet number and sedimentation but direct proportional with the decrease in albumin. Some patients information and results are shown in Tables 1, 2, 3.

DISCUSSION

In IBD although in the one side bleeding occurs, in the other side if we think that there are microthromboses being clinically apparent or being in sub-clinical vascular system, platelets play vital role in these types of patients. MPV, which is supposed to be able to reflect platelet function and disease clinical activity, can be measured very easily and guickly through automatic hemogram measurement devices. As also observed in our study, we determined that in active ulcerative colitis disease, MPV decreased in statistically significance level according to patients in remission. In addition, there was a negative significance relation between clinical, pathological and colonoscopic activity indexes of MPV and ulcerative colitis disease (p<0.001). However as expected in any systemic inflammatory response, in our ulcerative colitic clinically active patients, we found serum albumin, which is known as negative acute phase reactant, as low as well as the increase in sedimentation, platelet number and CRP. In our study there was a inverse proportional relation between MPV and the increase in platelet number and sedimentation and direct proportional relation with the decrease in albumin. Our this result shows that MPV is a finding not only coming across us with clinical activity index of ulcerative colitis disease but also reflecting pathological and colonoscopic activity indexes and which can be got so easily and with low cost. On the other hand, it is known that platelets having small MPV have lower functional capacity than platelets having big MPV (32). In our study that results similar to a few studies made before about MPV to have reduced apparently in our patient population having IBD shows that our this finding is suitable with the literature (25,26,35). In ulcerative colitis patients platelets increase with the increase in thrombopoiesis in the one side, in this situation it is expected MPV be big but MPV decreases in these patients. Although the reason of this dilemma is clear, the existence of so much and different agents making effect on different phases of thrombopoiesis and forming inflammatory process can be responsible. It was reported that there were some diseases, except IBD, in that MPV decreased. These are states such as haemodialysis application and macrocytic anaemia occurring after acute blood loss and after deficit of vitamin B12 and folic acid (33,34). According to a claimed mechanism, within haemodialysis, after disorder of platelet aggregation, platelet micro-aggregations can occur and decreasing MPV can

the consumption of big volumed and activated platelets in IBD in intestinal vascular system or their taking place in intestinal microvascular system in the shape of microthromboses can cause low MPV. In the other side, as known, an inflammatory process like IBD and also gastrointestinal bleeding increase thrombosis and platelet number in the blood increases. Big platelets or increases MPV is watched in this blood as expected in consequence of platelet increase. Here the key guestion must be that. Ok, then while MPV increasing in active haemorrhagic IBD is being expected, why does MPV significantly reduce? This question has not an apparent answer according to our information today but the existence of a defect in regulation of thrombopoiesis in the bone marrow comes across us as the most possible comment. MPV and platelet activation can follow different patterns in various diseases. As a result, the present study shows that MPV can reflect

contribute to this state (33). Similarly in the literature we see a comment for low MPV in IBD in that way (21):

ulcerative colitis disease activity since active ulcerative colitis patients have got low MPV values and the disease is related with clinical, pathological and colonoscopic indexes. MPV is very sensitive indicator in exhibiting ulcerative colitis disease activity. In clinical practise, it can be possible to use MPV and so to understand the disease clinical activity without spending additional effort and cost. A relation can be available between low MPV and IBD's inflammatory process. In the process passing from the bone marrow to the formation of platelet volume, mechanisms regulating new platelet volume are not known very clearly. There is need to explain mechanism regulating platelet volume through studies which will be made in the future.

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