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Evaluation of mean platelet volume as a predictor of gastric disorders

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ABSTRACT

Aim: Gastric disorders present in a wide range, including malignant and benign diseases of the upper gastrointestinal system. Increased MPV levels are associated with inflammation. The goal of this study was to determine the diagnostic importance of MPV in gastric disorders and evaluate level of MPV in patients that underwent upper gastrointestinal endoscopy.

Materials and Methods: Patients who had undergone endoscopy and shown to have a gastric disorder with blood count performed were included in the study. Only one surgeon performed all of the endoscopies. MPV value, platelet count, hemoglobin and white blood cell count levels were analyzed.

Results: 116 patients were included in the study. Mean age of all patients was 47,8±16,4. Mean value of MPV was determined to be 7,79±1,21, within the range of 5,85 to 12,5 fL. There was no significant correlation between diagnoses and MPV levels (P>0,05). Additionally, there was no significant difference in MPV levels in histopathological diagnoses groups (p>0,05). There was a highly negative correlation between platelet count and MPV levels in a Scatter Plot correlation graph (r=0,083).

Conclusions: MPV is a frequently used hematological parameter that indicates platelet function and activity affected by inflammation. It was hypothesized that changes in MPV levels could be associated with gastric disorders. Statistical analysis of the data revealed there was no association between MPV and gastric disorders. It is suggested that MPV is not a suitable marker to determine gastric disorders, however further larger studies can be useful to determine the importance of MPV such a context.

Key words: *Mean platelet volume, gastric disorders, gastritis, helicobacter pylori*

Introduction

Gastric disorders are present in a wide variety, including malignant and benign diseases of the upper gastrointestinal system. Most patients are symptomatic. One of the most common symptoms is dyspepsia. Other symptoms are epigastric pain, vomiting, nausea, heartburn, abdominal distension, weight loss, acid reflux or regurgitation, lack of appetite, and indigestion. Gastritis is the most common gastric disease diagnosed in symptomatic patients [1]. Gastric and duodenal ulceration is also commonly seen in patients having undergone endoscopy. Almost all cases of esophagitis are diagnosed with gastroesophageal reflux disease or hiatal hernia, the remaining cases related to drugs, infection diseases, radiotherapy, autoimmune disease, and allergies [2]. *Helicobacter pylori* is one of the primary

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causes of chronic gastritis. A range of diseases, from acute gastritis and gastric cancer to mucosa associated lenfoid tissue (MALT) lymphoma, are related to *Helicobacter pylori* infection as evidenced in recent studies [3-5]. *Helicobacter pylori* infection incidence is reported at between 23% to 65% in Turkey [6]. The main platelet volume (MPV) is one of the most important indicators of platelet activity. MPV is usually measured during routine blood count, and there is a positive relationship between MPV and platelet activity. Increased MPV levels are associated with inflammation [7]. The previous literature has shown that increased MPV levels have been established in serious diseases like myocardial infarction, diabetes mellitus, cerebral stroke and rheumatoid arthritis [8]. With increasing MPV, platelets have larger granules and thus more enzymatic and metabolic activity. This in turn enhances the thrombotic function of platelets brings about a higher risk of thrombosis [9]. The aim of this study was to determine the diagnostic importance of MPV in gastric disorders and evaluate the level of MPV in patients who had undergone upper gastrointestinal endoscopy. With this, the distribution of hematological parameters of such patients is put forth.

Materials and Methods

Data of the patients who were admitted to the General Surgery Department with upper gastrointestinal complaints were retrospectively examined from the Hospital Information System. 116 patients who underwent endoscopy and had a gastric disorder and blood count performed were included in the study. The study itself was approved by the Ethical Committee of the Sivrihisar State Hospital (2015/4). Patients recruited to the study were those determined to have anemia and thrombocytopenia, a diagnosis of malignancy, or a history of disease, including cardiac failure, chronic renal failure, liver failure, rheumatoid arthritis, systemic lupus erythematosus, diabetes mellitus, and coronary artery disease. Anemia criteria was Hb<11 g/dl for males and Hb<10 g/dl for females. 150.000 / µL was accepted as the thrombocytopenia lower limit. Only one surgeon performed all endoscopies and biopsies were taken from the antrum when in doubt of gastritis and malignancy using Olympus Actera CV-150 Processors and GIF-Q150 Endoscope (Olympus Corporation; Tokyo,Japan). Histopathological examination of biopsy specimens was made by a single blind pathologist and results were classified according to the Sydney System [10]. The Giemsa staining method was used to determine *Helicobacter pylori* presence. Blood samples were collected into tubes and included EDTA. A hemogram test was performed with the CELL-DYN 3700 Hemogram device (Abbott Laboratories. Abbott Park, Illinois). Normal values for MPV were between 7 and 11 fL. At this point, MPV values, platelet counts, and hemoglobin and white blood cell levels of all patients were analyzed. Correlation of MPV and endoscopic diagnoses was also analyzed. SPSS 15.0 (IBM Corporation, USA) statistical software was employed for statistical analysis of the data. The Kolmogorov-Simonov test and histogram was utilized for determining the distribution of the parameters. A Pearson p test was used to assess the correlation of parametric parameters with MPV. Spearman's correlation test was used for non-parametric parameters. One-way analysis of variance (ANOVA) was employed to analyze the patient group and histopathological diagnosis group. Continuous value results are given as mean±SD. A p value <0,05 was accepted as significant. The scatter plot correlation method was utilized for graphically analyzing correlation of continuous values. Further, power analysis with Web Power Free online statistical power analysis software (Zhang, Z., & Yuan, K.-H. (2015). WebPower: Statistical power analysis online. Retrieved from http://webpower.psychstat.org) was used because of the limitation of the number of patients.

Results

116 patients were included in the study. The mean age of all patients was 47,8±16,4. 62,1% of patients were female (mean age 48,5±17,4) and 37,9% were male (46,8±14,8), (Table 1). All patients underwent endoscopy procedures and biopsies were taken from 76,7% (n=89). Only four patients were evaluated as normal after endoscopy. The most frequent diagnosis was gastritis at 87,7% in 101 patients. Esophagitis and hiatal hernia followed with 44,83% and 26,72% (Table 2), respectively. The most frequent histophatological diagnosis was chronic gastritis, appearing in 58,6% of 89 patients from whom a biopsy was taken. *Helicobacter pylori* presence was affirmed with positive Giemsa

staining in 36 patients. Detailed results of diagnoses and histopathological results are listed in Table 3. Mean values of MPV were determined to be 7,79±1,21 within the range of 5,85 to 12,5 fL. The mean value of platelet counts were 247500±54550 /µL. The MPV levels in patients with gastritis diagnosed through endoscopy was 7,79 ± 1,24 fL, 8,00 ± 1,36 fL in patients with hiatal hernia, 8,01 ± 1,32 fL in patients with esophagitis, 7,68 ± 1,28 fL in patients with duodenitis, 7,21 ± 0,72 fL in patients with duodenal ulcer, 8,27 ± 1,10 fL in patients where bile reflux was seen, 8,21 ± 1,28 fL in patients with GERD diagnosed, and 7,84 ± 0,63 fL in patients evaluated as normal (Table 3). There was no significant correlation between diagnoses and MPV levels (P>0,05). Distribution of the diagnoses and histopathological results as well as the correlation results are listed in Table 2. Patient group platelet count mean levels are listed; 250740 ± 56720 /µL in gastritis, 235450 ± 48930 /µL in hiatal hernia, 243960 ± 56210 /µL in esophagitis, 250620 ± 59770 /µL in duodenitis, 259000 ± 73750 /µL in duodenal ulcer , 234620 ± 46480 /µL in bile reflux, 225180 ± 48290 /µL in GERD and 22080 ± 12120 /µL in normal patients (Table 3). Examination of hemoglobin and WBC showed no significant difference according to endoscopic diagnoses. The mean levels of hemoglobin and WBC are also listed in Table 3. Histopathological examination of biopsies revealed chronic gastritis was the most common with a mean MPV level of 7,70 ± 1,10 fL The mean level of MPV was determined as 7,82 ± 1,17 fL in chronic inflammation, 7,48 ± 0,98 fL in lenfoid aggregates, 7,37 ± 0,91fL in intestinal metaplasia, 7,31 ± 0,48 fL in gastric atrophy, and 7,71 ± 1,06 fL in the *Helicobacter pylori* positive patient group. There was not any significant differences in MPV levels in the histopathological diagnoses groups (p>0,05). The mean level of platelet count was determined to be 250400 ± 57140 /µL in chronic inflammation, 251590 ± 59490/µL in lenfoid aggregates, 246380 ± 60370/µL in intestinal metaplasia, 219440 ± 53770/µL in gastric atrophy, and 250060 ± 64050 /µL in the *Helicobacter pylori* posi-

Table 1. Mean values and standard derivations of the parameters of this study.

		Mean	± SD	P
MPV (fL)		7,79	1,21	0,660
PLT (x10³/µL)		247,50	54,55	0,731
Hb (g/dl)		14,61	1,53	0,392
WBC (x10³/µL)		7,68	2,02	0,232
Age	Total	47,88	16,44	
	Male	46,86	14,85	
	Female	48,50	17,41	

Table 2. Correlations of parameters with MPV.

Parameters		n	%	p
Diagnose	Gastritis	101	87,07	0,92
	Hiatal Hernia	31	26,72	0,26
	Esophagitis	52	44,83	0,07
	Duodenitis	29	25,00	0,60
	Duedonal Ulcer	3	2,59	0,41
	Bile Reflux	8	6,90	0,24
	GERD	11	9,48	0,21
	Normal	4	3,45	0,92
Pathologies	Chronic Gastritis	68	58,6	0,39
	Chronic Inflammation	46	39,7	0,83
	Lenfoid Aggregate	24	20,7	0,16
	Intestinal Metaplasia	9	7,76	0,29
	Gastric atrophy	9	7,76	0,22
	H. Pylori (+)	36	31	0,67

Table 3. Distribution of MPV, platelets, hemoglobin, and white blood cell count levels according to the diagnoses.

Diagnoses	MPV ± SD	Plt ± SD	Hb ± SD	WBC ± SD
Gastritis	7,79 ± 1,24	250,74 ± 56,72	14,63 ± 1,55	7,61 ± 2,00
Hiatal Hernia	8,00 ± 1,36	235,45 ± 48,93	15,00 ± 1,53	7,23 ± 1,79
Esophagitis	8,01 ± 1,32	243,96 ± 56,21	15,01 ± 1,56	7,69 ± 2,23
Duodenitis	7,68 ± 1,28	250,62 ± 59,77	14,86 ± 1,36	8,04 ± 2,19
Duedonal Ulcer	7,21 ± 0,72	259 ± 73,75	15,77 ± 2,10	6,86 ± 1,55
Bile Reflux	8,27 ± 1,10	234,62 ± 46,48	13,99 ± 1,28	8,67 ± 2,26
GERD	8,21 ± 1,28	225,18 ± 48,29	15,33 ± 1,89	6,87 ± 1,64
Normal	7,84 ± 0,63	220,8 ± 12,12	14,81 ± 0,82	9,33 ± 1,16

Table 4. MPV, platelets, hemoglobin, and white blood cell count levels according to histopathological examination.

Pathology	MPV ± SD	Plt ± SD	Hb ± SD	WBC ± SD
Chronic Gastritis	7,70 ± 1,10	250,40 ± 57,14	14,61 ± 1,59	7,68 ± 2,07
Chronic Inflammation	7,82 ± 1,17	251,59 ± 59,49	14,71 ± 1,79	7,89 ± 2,06
Lenfoid Aggregate	7,48 ± 0,98	246,38 ± 60,37	14,69 ± 1,62	7,20 ± 1,70
Intestinal Metaplasia	7,37 ± 0,91	219,44 ± 53,77	15,20 ± 1,69	6,92 ± 1,66
Gastric Atrophy	7,31 ± 0,48	251,00 ± 89,64	14,48 ± 2,06	7,08 ± 1,61
H. Pylori (+)	7,71 ± 1,06	250,06 ± 64,05	14,70 ± 1,62	7,71 ± 2,12

tive patient group (Table 4). Distribution of platelet counts were similar in groups of histopathological diagnoses (p>0,05). Similarly, serum hemoglobin levels and WBC followed a comparable distribution (Table 4). There was a highly negative correlation between platelet count and MPV levels in the Scatter Plot correlation graph (r=0,083) (Figure 1). A minimally negative correlation was determined between MPV with WBC and hemoglobin (r=0,28; r=0,027) (Figure 2).

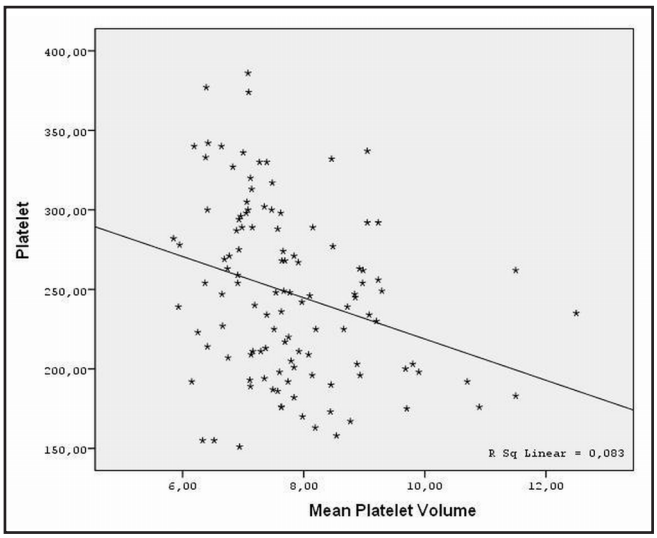

Figure 1. Correlation graphics of platelet and hemoglobin with MPV.

Discussion

MPV is a simple and commonly used hematological parameter that allows insight into platelet function and activity affected by inflammation. Increased MPV levels are established in a number of diseases, such as diabetes mellitus, myocardial infarction, and acute ischemic stroke. In this study, MPV was investigated as a probable marker of chronic inflammation and, in this context, a precursor to chronic gastritis. In addition, the relationship between the other endoscopic diagnoses and MPV was evaluated. Further to the MPV results with hemoglobin, WBC, and platelet count are included in the study. The dataset of endoscopic diagnoses and histopathological results from patients that had a biopsy taken collected. Hemogram results were analyzed and MPV, hemoglobin, WBC, and platelet count data separated. Intestinal inflammations, like gastritis, duodenitis, esophagitis, gastric, duodenal ulcerations, hiatal hernia, GERD, and bile reflux was in-

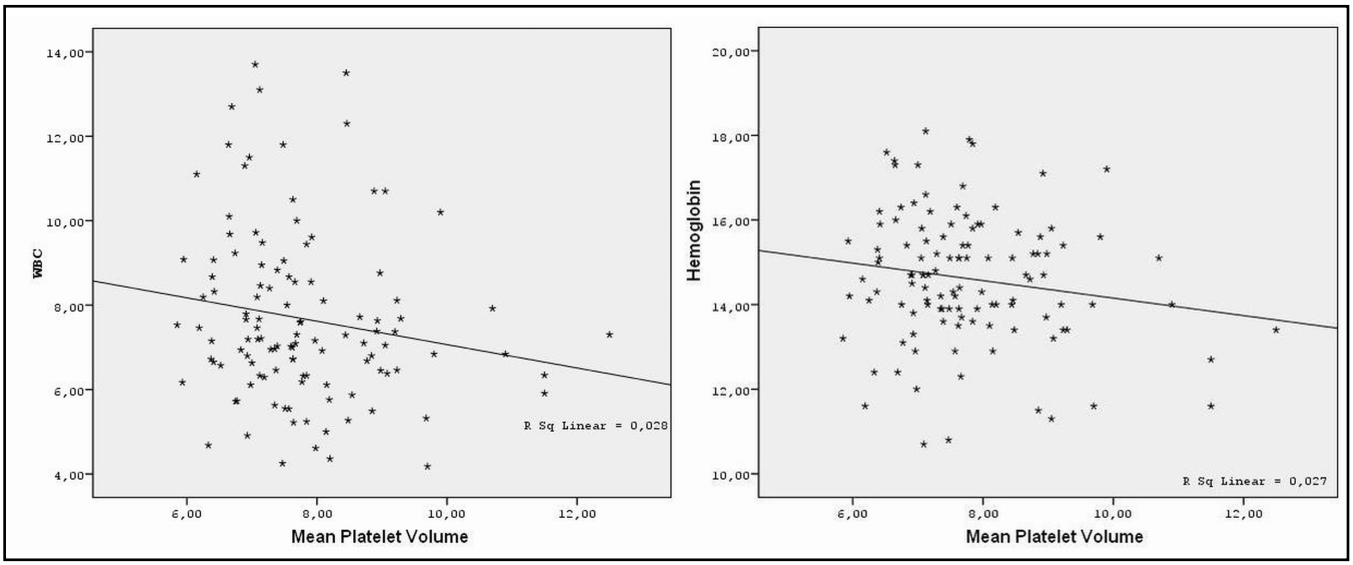

Figure 2. Correlation graphics of WBC and hemoglobin with MPV.

vestigated. The most common diagnosis was chronic gastritis as measured through both endoscopy and histopathology. The results of the biopsies, analyzed according to the Sidney system, were scored according to positivity. Inflammatory gastric disorders have a multifactorial mechanism. One of these factors is the resulting mucosal damage associated with platelets in immune system activation. As a consequence of this immune system activation, inflammation of gastric mucosa begins and leukocyte adhesion and platelet aggregation are suspected as the cause of gastric lesions [11]. Larger platelets have more granules and cytokines, and as a result of superoxide anions and hydroxyl radicals generated from platelets, platelet aggregation and vascular changes occur. Platelet activation develops with the increase of MPV; activated platelets release P-selectin that aggravates inflammation [11, 12]. Previous studies have demonstrated that increased MPV is associated with metabolic syndrome, diabetes mellitus, coronary artery syndrome, hypertension, ischemic stroke, and periphery artery syndrome, all the result of chronic inflammation of vascular structures [13-17]. There are a few studies that have looked at the relationship of MPV and *Helicobacter pylori* gastritis [18, 19]. No study in the literature has assessed the correlation between MPV and gastric disorders. In the present work, the mean value of MPV was found to be 7,79±1,21 fL in normal ranges. There was no relationship between MPV levels and any kind of gastric disorders as diagnosed by upper gastrointestinal endoscopy. Power analysis of the both patient group and the histopathological group showed there there was a limitation because of the small numbers of patients with a power of 80% and a type 1 error rate of 5%. Previous investigations from the literature have looked at the relationship between MPV and *Helicobacter pylori* gastritis present and saw no relationship. Topal et al. found the mean value of MPV to be 8,79±1,43 fL and reported that MPV is not a suitable marker for *Helicobacter pylori* gastritis [18]. Akin to this, Yeniova et al. determined that the mean MPV value of 8,75±0,93 fL might not be an indicator of gastric inflammation [19]. Similar to the previous work, here, a negative correlation between platelet count and MPV levels was found. Analysis of hemoglobin and WBC resulted in no significant difference or correlation. Moreover, there are various studies that have reported the relationship between C reactive protein levels and *Helicobacter pylori* gastritis [20]. As demonstrated in the work presented here, a hemogram is seen as a routine test frequently utilized in practice. It is hypothesized that changes in MPV levels could be associated with gastric disorders. This routine blood test can be used as a practical precursor to gastric disorder diagnosis. In order to support patients, their complaints could be used to decide further lines of inquiry. Statistical analysis of the data indeed revealed there was no correlation between MPV and gastric disorders. Consequently, it is suggested that MPV is not a suitable marker to determine the existence of gastric disorders, however further larger studies can be useful to determine the importance of MPV as related to them.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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