Exp Biomed Res 2018; 1(2):55-59 DOI: 10.30714/j-ebr.2018237497

EXPERIMENTAL BIOMEDICAL RESEARCH
http://www.experimentalbiomedicalresearch.com

Research Article

Mean Platelet Volume to Platelet ratio as a promising marker of hepatosteatosis

Mehmet Ali Kosekli[1], Edip Erkus[2], Mehmet Zahid Kocak[2]

[1] *Abant Izzet Baysal University Hospital, Department of Gastroenterology, Bolu, Turkey*

[2] *Abant Izzet Baysal University Hospital, Department of Internal Medicine, Bolu, Turkey*

ABSTRACT

Aim: Hepatosteatosis confers increased lipid accumulation in the hepatocytes which is associated with inflammation. Hemogram parameters, such as mean platelet volume (MPV) and MPV to platelet ratio (MPR) are proposed as novel inflammatory markers in recent studies. We aimed to compare MPR of subjects with hepatosteatosis to those in healthy controls.

Methods: Patient admissions to our clinic with a diagnosis of hepatosteatosis were retrospectively analyzed MPR values compared to those in healthy controls.

Results: Mean MPR of hepatosteatosis group (0,04 ± 0,01fL/mm^3) was significantly higher than the MPR of control subjects (0,03 ± 0,01 fL/mm^3) (p=0.04). A Pearson's Correlation analyze was revealed significant correlations between MPR and fasting plasma glucose (r=0.26, p=0.004) and between MPR and LDL-cholesterol (r=0.19, p=0.04).

Conclusion: An elevated MPR should alert physicians for hepatosteatosis in otherwise healthy subjects. Therefore, calculation of MPR by automatic hemogram analyzers is advised.

Keywords: Mean platelet volume to platelet ratio; hepatosteatosis; inflammation.

Corresponding Author: *Mehmet Ali Kosekli, M.D.*
Abant Izzet Baysal University Hospital, Department of Gastroenterology, Bolu, Turkey
E-mail: kosekli@gmail.com
Received 2018-02-16, Revision 2018-03-05
Accepted 2018-03-07

Introduction

Hepatosteatosis confers increased lipid accumulation in the hepatocytes. It is one of the clinical spectrums of nonalcoholic fatty liver disease. Despite it is the mildest form of NAFLD, it may progress to steatohepatitis, fibrosis and cirrhosis [1]. Inflammation is involved in the course of hepatosteatosis. An inflammatory marker, interleukin-6, have been proposed to be linked to steatosis [2].

Hemogram parameters are proposed as novel inflammatory markers in recent studies. These parameters include mean platelet volume (MPV), red cell distribution width (RDW) and neutrophil to lymphocyte ratio (NLR), which all have shown to be associated with inflammatory conditions [3-7]. A novel hemogram derived indices, MPV to platelet ratio (MPR), have been found to be associated with various inflammatory conditions [8-10].

In present retrospective analysis, we aimed to compare MPR of subjects with hepatosteatosis

to those in healthy controls and find out whether it was associated with serum lipids and glucose levels.

Methods

Patient admissions to our clinic with a diagnosis of hepatosteatosis were retrospectively analyzed between, January 2015 and December 2017. Diagnosis of hepatosteatosis was established by sonography. Control subjects were enrolled to the study from healthy volunteers whom presented to our clinic for a routine check-up. Data of the subjects obtained from computerized database of the institution and patients' files. Subjects with malignant diseases, active infection or inflammatory conditions were excluded.

Age, gender, and laboratory parameters, such as, aspartate transaminase, alanine transaminase, fasting plasma glucose, serum creatinine, serum lipids (LDL cholesterol and triglyceride), and hemogram parameters including white blood cell count (WBC), hemoglobin (Hb), hematocrit (Htc), platelet count (Plt) and MPV were recorded. A MPR value is calculated by simply dividing of MPV by Plt.

Statistical analysis were done with SPSS software (SPSS 15.0 for Windows, IBM Corp., Chicago, IL, USA). Study variables conducted with Kolmogorov- Smirnov test for distribution between study and control groups. While comparison of homogenously distributed parameters were done with independent samples t test and expressed as mean ± standard deviation, comparison of nonhomogeneous parameters were conducted with Mann Whitney U test and expressed as median (minimum-maximum). P values lower than 0.005 were considered statistically significant.

Results

A total of 119 subjects enrolled to the study; 61 in hepatosteatosis group and 58 in control group. Mean age of hepatosteatosis and control groups were 47 ± 12 years and 43,5 ± 10 years, respectively. Age was not statistically different between groups (p=0.08). There were 29 women and 32 men in hepatosteatosis group and 23 women and 35 men in control group. Gender was not statistically different among study groups, either (p=0.39).

Serum creatinine (p=0.23), WBC (p=0.47), Hb (p=0.48), Htc (p=0.85), and Plt (p=0.06) were not significantly different in hepatosteatosis group compared to control group. Fasting plasma glucose, triglyceride, LDL cholesterol, AST, ALT and MPV were significantly different between study groups ($p<0.05$ for all). Mean MPR of hepatosteatosis group (0,04 ± 0,01 fL/mm^3) was significantly higher than the MPR of control subjects (0,03 ± 0,01 fL/mm^3) (p=0.04). Table 1 shows data of the study subjects.

A Pearson's Correlation analyze was revealed significant correlations between MPR and fasting plasma glucose (r=0.26, p=0.004) and between MPR and LDL-cholesterol (r=0.19, p=0.04).

Discussion

Present study showed that hepatosteatosis is significantly associated with elevated levels of MPR, due to increased inflammatory burden. Another important result of the present report is that MPR was significantly and positively correlated with fasting plasma glucose and LDL-cholesterol.

Recent studies focused on the association between MPR and several clinical conditions, ie, cancer, ischemic heart disease and infections. Azab et al reported that MPR was an independent predictor of mortality in

Table 1. Data of the study population.

Parameters		Hepatosteatosis group	Control group	p
Gender	Women (n)	29	23	0.39
	Men (n)	32	35	
		Mean ± SD		
Age (years)		47 ± 12	43,5 ± 10	0.08
WBC (k/mm³)		7,4 ± 1,9	7,7 ± 2,2	0.47
Hb (g/dL)		14,6 ± 1,5	14,4 ± 1,7	0.48
Htc (%)		43 ± 4	43 ± 5	0.85
Plt (k/mm³)		284 ± 131	249 ± 54	0.06
MPR fL/mm³		0,04 ± 0,01	0,03 ± 0,01	0.04
LDL (mg/dL)		133 ± 32	105 ± 20	<0.001
		Median (Min-Max)		
MPV (fL)		9,4 (7,7-19)	8,1 (6,5-10,3)	<0.001
AST (U/L)		24 (11-107)	20 (13-33)	<0.001
ALT (U/L)		38 (10-126)	28 (18-72)	0.001
Glucose (mg/dL)		103 (75-235)	88 (69-114)	<0.001
Creatinine (mg/dL)		0,8 (0,6-1,3)	0,7 (0,6-1,1)	0.23
Triglyceride (mg/dL)		136 (48-561)	98 (52-141)	<0.001

patients with non ST elevation myocardial infarction [9]. Another study found significantly increased MPR levels in subjects with hepatocellular carcinoma compared to controls [10]. Authors also showed increased MPR in patients with infective endocarditis [11].

Hepatic steatosis is the initial clinical picture of NAFLD. It is characterized with increased lipid accumulation in hepatocytes. Steatosis in the liver make susceptible to the attack of inflammatory cytokines, such as, tumor necrosis factor-alpha, transforming growth factor-beta and interleukins [1]. Elevated MPR in subjects with hepatosteatosis detected in present study could be explained by is the associations between hepatosteatosis and inflammation and between MPR and inflammatory conditions.

Diseases characterized with low grade inflammation are associated with elevated MPV values [12]. Since type 2 diabetes mellitus and obesity produce a continuous and low grade inflammatory burden, they are all related increased levels of MPV [13, 14]. Moreover, hepatic steatosis is prevalent in obesity and type 2 diabetes mellitus [1]. Correlation between plasma glucose and MPR in present study suggests the previous findings of the studies in literature. In addition, elevated MPV has been reported in hepatosteatosis, too [15]. Since MPV is numerator and Plt is denominator in calculation of MPR, elevation in MPV increases MPR values.

Despite high MPV values are indicative of platelet activation [12], MPR is better than MPV in predicting platelet activity [16]. This fact has been suggested by Han et al, whom reported greater sensitivity and specifity of PMR compared to MPV in detecting thrombosis [17].

Limitations of present study are retrospective design and relatively small study cohort. However, results of the present report are important, since, to our knowledge, this is the first study pointed out the elevated MPR in hepatosteatosis.

In conclusion, an elevated MPR should alert physicians for hepatosteatosis in otherwise healthy subjects. Therefore, calculation of MPR by automatic hemogram analyzers is advised.

Compliance with ethical standards

The authors declare that they have no conflicts of interest concerning for this article.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Institutional Research Ethics Committee and with the 1964 helsinki declaration and its later amendments or comparable ethical standards.

Informed consent was obtained from all individual participants included in the study.

References

[1]Hijona E, Hijona L, Arenas JI, Bujanda L. Inflammatory mediators of hepatic steatosis. Mediators of inflammation. 2010;2010.

[2]Marra F, Bertolani C. Adipokines in liver diseases. Hepatology. 2009;50(3):957-69.

[3]Aktas G, Sit M, Dikbas O, et al. Could red cell distribution width be a marker in Hashimoto's thyroiditis? Experimental and Clinical Endocrinology & Diabetes. 2014;122(10):572-74.

[4]Clarke K, Sagunarthy R, Kansal S. RDW as an additional marker in inflammatory bowel disease/undifferentiated colitis. Digestive diseases and sciences. 2008;53(9):2521-23.

[5]Aktas G, Alcelik A, Tekce BK, et al. Red cell distribution width and mean platelet volume in patients with irritable bowel syndrome. Przeglad gastroenterologiczny. 2014;9(3):160-63.

[6]Gasparyan AY, Stavropoulos-Kalinoglou A, Toms TE, et al. Association of mean platelet volume with hypertension in rheumatoid arthritis. Inflammation & Allergy-Drug Targets (Formerly Current Drug Targets-Inflammation & Allergy). 2010;9(1):45-50.

[7]Aktas G, Sit M, Dikbas O, et al. Elevated neutrophil-to-lymphocyte ratio in the diagnosis of Hashimoto's thyroiditis. Rev Assoc Med Bras. 2017;63(12):1065-68.

[8]Ates S, Oksuz H, Dogu B, et al. Can mean platelet volume and mean platelet volume/platelet count ratio be used as a diagnostic marker for sepsis and systemic inflammatory response syndrome? Saudi medical journal. 2015;36(10):1186.

[9]Azab B, Torbey E, Singh J, et al. Mean platelet volume/platelet count ratio as a predictor of long-term mortality after non-ST-elevation myocardial infarction. Platelets. 2011;22(8):557-66.

[10]Cho SY, Yang JJ, You E, et al. Mean platelet volume/platelet count ratio in hepatocellular carcinoma. Platelets. 2013;24(5):375-77.

[11]Cho SY, Jeon YL, Kim W, et al. Mean platelet volume and mean platelet volume/platelet count ratio in infective endocarditis. Platelets. 2014;25(8):559-61.

[12] Yuri Gasparyan A, Ayvazyan L, P Mikhailidis D, et al. Mean platelet volume: a link between thrombosis and inflammation? Current pharmaceutical design. 2011;17(1):47-58.
[13] Cakir L, Aktas G, Enginyurt O, et al. Mean Platelet volume increases in type 2 diabetes mellitus independent of HbA1c level. Acta Medica Mediterranea. 2014;30:425-28.
[14] Coban E, Ozdogan M, Yazicioglu G, et al. The mean platelet volume in patients with obesity. International journal of clinical practice. 2005;59(8):981-82.
[15] Aktas G, Alcelik A, Tekce BK, et al. Mean platelet volume and red cell distribution width in hepatosteatosis. National journal of medical research. 2013;3:264-66.
[16] Shin DH, Rhee SY, Jeon HJ, et al. An Increase in Mean Platelet Volume/Platelet Count Ratio Is Associated with Vascular Access Failure in Hemodialysis Patients. PloS one. 2017;12(1):e0170357.
[17] Han JS, Park TS, Cho SY, et al. Increased mean platelet volume and mean platelet volume/platelet count ratio in Korean patients with deep vein thrombosis. Platelets. 2013;24(8):590-93.