



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

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ABSTRACT

Aim: Hepatosteatosi 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 hepatosteatosi to those in healthy controls.

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

Results: Mean MPR of hepatosteatosi group ($0,04 \pm 0,01 \text{ fL/mm}^3$) was significantly higher than the MPR of control subjects ($0,03 \pm 0,01 \text{ fL/mm}^3$) ($p=0.04$). 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 hepatosteatosi in otherwise healthy subjects. Therefore, calculation of MPR by automatic hemogram analyzers is advised.

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

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Introduction

Hepatosteatosi 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 hepatosteatosi. An

inflammatory marker, interleukin-6, have been proposed to be linked to steatosi [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 hepatosteatosi

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 hepatosteatois were retrospectively analyzed between, January 2015 and December 2017. Diagnosis of hepatosteatois 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 \pm 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 hepatosteatois group and 58 in control group. Mean age of hepatosteatois and control groups were 47 ± 12 years and $43,5 \pm 10$ years, respectively. Age was not statistically different between groups ($p=0.08$). There were 29 women and 32 men in hepatosteatois 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 hepatosteatois 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 hepatosteatois group ($0,04 \pm 0,01$ fL/mm³) was significantly higher than the MPR of control subjects ($0,03 \pm 0,01$ fL/mm³) ($p=0.04$). 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 hepatosteatois 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		Hepatosteatosi 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/mm ³)		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/mm ³)		284 ± 131	249 ± 54	0.06
MPR fL/mm ³		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 hepatosteatosi detected in present study could be explained by is the associations between hepatosteatosi 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 hepatosteatosi, 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 specificity 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.

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