

LETTER TO THE EDITOR

Mean Platelet Volume in Patients with Increased Cardiac Troponin I

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Platelets are known to be involved not only in hemostasis but also in inflammation and immunity. Platelet activation via pathologically exaggerated and deregulated versions is a pivotal role in inflammation-induced atherosclerosis, as well as in thrombosis causing cardiovascular events [1]. Platelet size is related with platelet function and activation [2]. In addition, platelet indices were believed to be strong predictors of platelet activation and the parameters related to cardiovascular diseases [3]. Among platelet indices, the mean platelet volume (MPV) is the most commonly used laboratory tool to estimate platelet size and has been suggested as a useful index of platelet activation [4]. MPV has been reported as increased in unstable angina pectoris and myocardial infarction (MI) [5]. However, the studies about platelet indices in cardiovascular disease have been not investigated enough. Especially, there is no study which directly analyzes MPV according to the value of troponin I (cTnI) to the best of our knowledge. In this study, we investigated MPV levels in patients with increased cTnI comparing with both healthy and disease controls with no abnormal cTnI test results as well as their follow-up data.

All the specimens were collected from the individuals aged 20 - 92 years who visited Kyung Hee Medical Center between January 2011 and April 2012. The patient group was composed of 843 specimens from 389 patients with increased cTnI levels above the upper reference limit ($> 0.04 \mu\text{g/L}$). A total of 174 patients showing cTnI results within the reference limit ($\leq 0.04 \mu\text{g/L}$) and receiving confirmation not to have acute coronary syndrome (ACS) were collected as the disease control

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Table 1. Characteristics of patients with increased cTnI in this study.

Patient group	Mean
Age ± SD, years	65.62 ± 14.32
Male to female	338:516
WBC, 10 ⁹ /L (IQR)	10.94 (6.79 - 2.74)
Platelet, 10 ⁹ /L (IQR)	224.86 (155.75 - 277.00)
MPV, fL (IQR)	8.64 (7.80 - 9.20)
cTnI, µg/L (IQR)	3.44 (0.09 - 1.29)
CK-MB, µg/L (IQR)	13.71 (1.90 - 9.50)
NT-proBNP, ng/L (IQR)	8471.73 (353.55 - 7520.25)

Abbreviations: SD - standard deviation, IQR - interquartile range, WBC - white blood cell, MPV - mean platelet volume, cTnI - cardiac troponin I, CK-MB - creatine kinase-MB, NT-proBNP - N-terminal prohormone of brain natriuretic peptide.

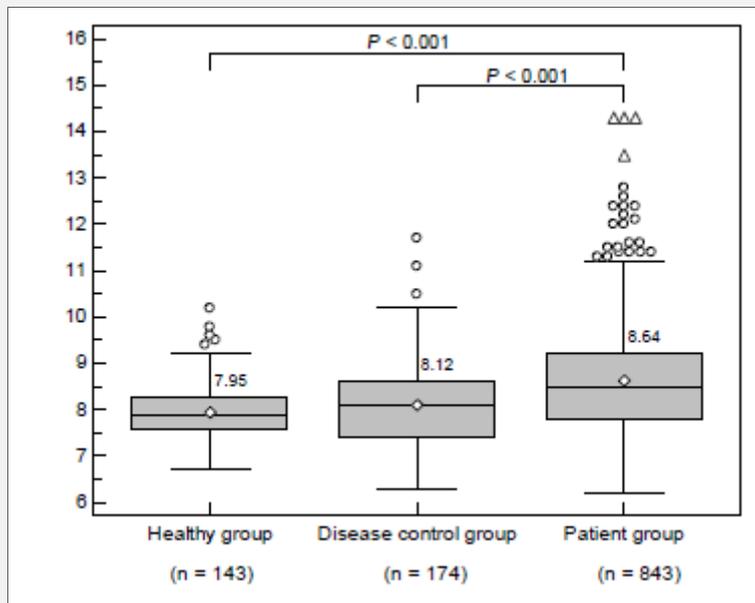


Figure 1. Comparison of MPV values between four groups.

In each box plot, the mean value is reported and is represented by the diamond symbol. The upper, lower ends and inner lines of the boxes correspond to the 3rd and 1st quartile and the median values, respectively. Error bars denote minimum and maximum values, and circles and stars indicate outlier values. In each box plot the mean is reported and is represented by the diamond symbols. The graph shows the statistically significant differences of MPV values between the increased cTnI group (patient group) and the normal cTnI group (disease control group) and between the increased cTnI group (patient group) and the healthy group.

group. For the healthy group, 143 individuals for medical check-ups were enrolled and were used in our previous studies [2,6]. Any individuals with hypertension, diabetes, or smoking were excluded from the healthy group by extensive chart review. Blood was sampled by

venipuncture and collected in tubes containing EDTA. MPV was measured in an Advia 2120 (Siemens Healthcare Diagnostics, Tarrytown, NY, USA) within 2 hours from sampling. cTnI was measured on an Access II (Beckman Coulter Inc., Fullerton, CA, USA) with Accu

TnI reagent (Beckman Coulter Inc., Fullerton, CA, USA). Welch's ANOVA was done to examine the difference of means between the groups. P-values < 0.05 were considered to indicate statistical significance. The statistical analyses were performed using SPSS version 17.0 (SPSS Inc., Chicago, IL, USA) and Excel 2007 (Microsoft, Redmond, WA).

In the increased cTnI group, the mean age was 65.62 (median 68.00) years and the mean of serum cTnI was 3.42 µg/L (Table 1). Significant difference ($p < 0.001$) was observed in means of MPV values between three groups. The mean MPV levels differed significantly between increased cTnI group (8.64 fL) and normal cTnI group (8.12 fL) ($p < 0.001$). There is a significant difference between the increased cTnI group and the healthy group (7.95 fL) when comparing the means of MPV values ($p < 0.001$) (Figure 1). Among the patients with increased cTnI, 292 patients had follow-up data of cTnI levels. cTnI values normalized in 175 patients during follow up. The mean of MPV (8.47 fL) at the time of normalized cTnI was decreased than initial MPV levels in increased cTnI group.

A relationship has recently been reported between MPV and various ranges of coronary artery disease (CAD). Pal et al. reported MPV values in ACS patients were greater than in non-ACS patients [7]. In other studies, a greater MPV value in MI patients was observed compared with unstable CAD or stable CAD [8,9]. Khandehar et al. and Khode et al. also demonstrated a higher MPV value in stable CAD in comparison to a healthy control group [9,10]. The result of this study also indicated that in the increased MPV group, MPV levels were significantly higher than those of disease control group. Significant differences were seen in the MPV results between the increased cTnI group and the healthy group. We have found a direct relationship between cTnI and MPV. cTnI is a widely accepted gold standard marker for definitive diagnosis of AMI due to its high specificity and selectivity. Therefore, increased MPV along with cTnI and an understanding of the pathophysiological role of platelets in ACS is conclusive evidence that MPV can play a role in the diagnosis of ACS.

In addition, there were some studies that investigated the impact of MPV on short-term and long-term prognosis in patients with stable CAD or MI, whose results were controversial. We found a significant decrease in value of MPV (8.34 fL) after the normalization of cTnI ($p = 0.009$), which means that MPV can be used during follow-up. On the other hand, another important finding is MPV levels after normalization of cTnI were still higher than those in the healthy group and the disease control group.

However, the amount of MPV data in the follow-up was relatively small compared to original MPV data in patients with increased cTnI. Therefore, a large study with longitudinal follow-up should be investigated to analyze the change of MPV in an individual with ACS in the future. This can be the foundation to reveal the role of MPV in diagnosis, follow-up, and prediction of the

prognosis in cardiovascular diseases.

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Declaration of Interest:

There is no potential conflict of interest relevant to this article.

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