

# Is mean platelet volume itself a sufficient indicator of coronary syndrome X?

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Dear Editor,

We read with deep interest the paper "Evaluation of the mean platelet volume in patients with cardiac syndrome X" by Demirkol et al. (1). The authors (1) aimed to evaluate the mean platelet volume (MPV) in patients with cardiac syndrome X (CSX) and compare patients with coronary artery disease (CAD) with a control group. We have a minor criticism concerning their methodology.

Although statistical significance was defined as  $p < 0.05$  and the  $p$ -values of MPV were given, the  $p$ -values of all variables should have been provided in the paper (1). It seems that some of the variables are not compatible with a normal distribution. However, the continuous variables were only reported as mean  $\pm$  standard deviation (SD). In addition, the authors (1) used a one-way analysis of variance (ANOVA) or Kruskal-Wallis test to compare the variables. ANOVA is used to analyze the means of two or more samples. The Kruskal-Wallis test is the nonparametric equivalent of the ANOVA test. The statistical test used for each variable in the study is thus unclear (1).

CSX is an angina with signs associated with decreased blood flow to the heart tissue but with normal coronary arteries. A number of systemic microvascular abnormalities, including endothelial cell dysfunction, diffuse atherosclerosis, and systemic inflammation, result in reduced blood flow (2,3). Platelets play a key role in atherothrombosis and inflammation. MPV, an index of platelet function, is a potential marker of platelet reactivity. Therefore, recent studies have focused on platelet function and inflammatory markers to determine their importance in cardiovascular diseases, including CSX (4,5). Most conditions, including traditional risk factors (e.g., hypertension, atherogenic lipoproteins, obesity, and hyperglycemia) and many inflammatory conditions (e.g., infection and inflammatory diseases), can influence MPV (4,5). However, the authors (1) did not exclude local or systemic infections or any previous history of infection (particularly  $< 3$  months).

In addition, the authors (1) did not analyze inflammation markers, such as C-reactive protein (CRP), even though

their role in inflammation has been previously reported in CAD. It is still unclear whether MPV measurement is the best method to determine the inflammation in patients with CSX. If other proven inflammatory markers (in addition to MPV) had been screened and were found to correlate with the MPV of these patients, the conclusions would have been more definitive. Therefore, the MPV should be evaluated together with the other serum inflammatory markers. Although the authors (1) did not include other markers relevant to CSX in their paper, we agree with their follow-up letter that suggests that MPV itself (without other inflammatory markers) may not provide comprehensive information about ongoing inflammation (6). Furthermore, in addition to the  $p$  values, the exact MPV values for each group should have been provided in order to analyze the entire study group to determine the clinical impact of the MPV.

We would like to congratulate the authors (1) for highlighting the efficacy of MPV in patients with CSX. MPV appears to be a novel marker and an important indicator and predictor of atherothrombosis together with traditional risk factors and other proven inflammatory markers. Large-scale prospective clinical studies with these recommendations are now needed.

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No potential conflict of interest was reported.

**DOI:** 10.6061/clinics/2013(07)30