

The platelet volume in patients with cardiac syndrome X

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Demirkol et al. (1) compared the mean platelet volume (MPV) in the plasma of patients with cardiac syndrome X, patients with coronary artery disease and healthy controls. They observed that the MPV in patients with cardiac syndrome X and coronary artery disease was significantly higher than that observed in the control group. There were no significant differences in the MPV between the cardiac syndrome X and the coronary artery disease groups. Although this study is interesting, we would like to make a minor criticism based on its methodological aspects.

Blood was collected in a Vacutainer tube containing ethylenediaminetetraacetic acid (EDTA) to measure the MPV. The authors analyzed the blood samples after two hours of venipuncture. However, a two-hour delay after blood sampling can cause abnormal MPV measurement results. This abnormality is important in tubes containing EDTA. As we know, platelets exhibit a time-dependent swelling when blood samples are anticoagulated with EDTA; however, this swelling does not occur in the presence of citrate (2). With impedance counting, the MPV increases over time as the platelets swell in EDTA. An increase of 7.9% within 30 min and an overall increase of 13.4% over 24 hours have been reported, although the majority of this increase occurs within the first 6 hours (3). The recommended optimal measuring time for MPV is 120 min after venipuncture (4). For reliable MPV measurements, the potential influence of the anticoagulant must be carefully controlled either by using an alternative anticoagulant, such as citrate, or by standardizing the time delay between sampling and analysis (less than 2 hours).

The pathophysiology of cardiac syndrome X is still being debated; however, endothelial dysfunction leading to a reduced coronary microvascular dilatory response and increased coronary resistance are thought to play important roles. We wish to add our experience with respect to this subject. In our previous study, we investigated whether MPV increased in patients with idiopathic dilated cardiomyopathy and whether increased MPV correlated with the degree of coronary microvascular dysfunction (5). We have found that MPV is an independent predictor of lower coronary flow reserve. Therefore, we believe that impaired coronary microvascular function as a result of endothelial dysfunction plays an important role in increasing the MPV.

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