Comment on “Relationship Between Inflammatory Markers and Contrast-Induced Nephropathy in Patients With Acute Coronary Syndrome After Coronary Angiography”

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We read with interest the article by Zorlu and Koseoglu entitled “Comparison of the relationship between inflammatory markers and contrast-induced nephropathy in patients with acute coronary syndrome after coronary angiography.”¹ This study reported that mean platelet volume (MPV) to lymphocyte ratio (MPVLR) is a strong predictor maker for contrast-induced nephropathy (CIN). We would like to mention some concerns about the study.

Both MPV and MPVLR are inexpensive and easy laboratory tests. The MPV has been reported to increase in many diseases.² It can be useful in monitoring the severity of many diseases when measured under appropriate laboratory conditions. Increased MPV indicates an increase in the number of young, large, and active platelets in peripheral blood.³,⁴ Large and active platelets release active substances and they may be involved in occlusive arterial diseases.⁴ Increased inflammation and oxidative stress lead to the generation of young platelets from the bone marrow.³,⁴ Increased oxidative stress and inflammation may lead to CIN.⁵ Since both oxidative stress and inflammation may be involved in CIN development and increased MPV, it is reasonable to find a high MPVLR in patients developing CIN.

Many comorbid conditions are associated with increased MPV. The number of diabetic and hypertensive patients was higher in the CIN group than in the non-CIN group in the study by Zorlu and Koseoglu.¹ Mean platelet volume values of both diabetic and hypertensive patients are higher than for healthy individuals.² Therefore, the MPVLR in these patients may be high before the development of CIN. Patients with diabetes and/or hypertension have increased oxidative stress and chronic inflammation.⁶ These patients are already at high risk for the development of CIN.⁷ We do not know whether their patients’ blood glucose levels and blood pressure are well or poorly controlled.¹ HbA₁c for diabetic patients and blood pressure values for hypertensive patients should be specified in the study.¹ Thyroid diseases cause an increased MPV⁸; however, there is no information about thyroid diseases in the study by Zorlu and Koseoglu. If the MPVLR before CIN development is provided, the power of the study will increase.

Not evaluating diabetic and hypertensive nephropathy is another deficiency in the study.¹ Hyperglycemia, hyperlipidemia, and high blood pressure promote the development of diabetic nephropathy (DN).⁹ The triglyceride and glucose levels and the number of hypertensive patients in the CIN group were significantly higher than in the non-CIN group.¹ Zorlu and Koseoglu excluded patients with estimated glomerular filtration rate (eGFR) <30 mL/min in their study.¹ However, it is unclear whether their patients who developed CIN had previous DN. Although serum creatinine and eGFR values were normal, DN may have started. Spot urine or 24-hour urine albumin and creatinine levels of diabetic patients should be included in the study by Zorlu and Koseoglu. Patients with DN have a significantly higher risk of developing CIN than those without DN.¹⁰ A previous study reported that the MPVLR was significantly higher in patients with DN than in patients with type 2 diabetes mellitus (T2DM) without DN.¹¹ The neutrophil to lymphocyte ratio, MPV, and platelet distribution width values were also higher in patients with DN than those with T2DM but without DN.¹² We speculate that some of their patients may have DN, and these patients may already have a higher MPVLR before CIN develops. Further studies on the relationship between MPVLR and CIN are needed.

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