Dear Editor

We would like to thank Dr. Varol for his recent interest in our article that evaluated mean platelet volume (MPV) before and after renal transplant as a potential predictor of renovascular thrombosis. He asked for the methodology of blood sampling to measure MPV in our study. Previously, it was suggested to use tubes with a high concentration of sodium citrate to obtain more reliable measures of MPV. However, this recommendation did not meet routine practice demands. Instead, most laboratories, as in our institution, continue to rely on ethylenediaminetetraacetic acid as the standard hematologic anticoagulant by further shortening processing times. Mean platelet values have been shown to be independent of the anticoagulation with ethylenediaminetetraacetic acid or citrate if measured within 1 hour of sampling. In accord with this recommendation, full blood counts of all the patients at our institution are routinely measured in K3 ethylenediaminetetraacetic acid within 15 minutes of venipuncture.

Many factors are known to influence the MPV including smoking, obesity, diabetes mellitus, prediabetes, hypertension, hypercholesterolemia, the metabolic syndrome, some antihypertensive agents, and immunosuppressive drugs. In our study, all patients were children, and none of them smoked. As specified in our original article, obesity was not seen in any of our patients before transplant, and the MPV levels were measured, and found to be decreased by the end of the first month after transplant. In such a short time, no serious weight loss was seen in any patients. None of the patients had a diagnosis of diabetes mellitus or prediabetic status.

Hypertension is a prevalent complication occurring in 80% to 85% of all kidney transplant recipients. The pathogenesis of posttransplant hypertension is multifactorial including pretransplant hypertension, donor hypertension, renin secretion from the native kidney, graft dysfunction, recurrent disease, and immunosuppressive treatment. Conversely, an abnormally high prevalence of the metabolic syndrome has been seen in patients receiving solid-organ transplants, and some of the pathogenetic factors might include cyclosporine and corticosteroids. In our study, no significant differences in blood pressures and serum lipid profiles were seen in any of the patients, either before or after renal transplant. We know that hyperglycemia, hypercholesterolemia, hypertension, and thrombocytopenia are associated with high MPV levels, and such adverse effects of immunosuppressive drugs (cyclosporine or tacrolimus in combination with mycophenolate mofetil and prednisolone) were noted.

There is a paucity of research that investigates the relation between the MPV and use of immunosuppressive agents. In general, increases in the MPV have been reported. Reis and associates compared the effects of sirolimus and cyclosporine, and found that hemoglobin, hematocrit, MPV, and platelet distribution width were significantly higher in the sirolimus group than in the cyclosporine group. In another study, Gasparyan and associates reported a significant increase in MPV after anti-TNF-alpha therapy in patients with rheumatoid arthritis, but more studies are required to determine the relation between MPV and various immunosuppressive agents.
agents in different patient populations. Finally, if the mentioned changes were seen in patients’ glucose and lipid levels and blood pressure values, the MPV would be expected to increase. Therefore, in our opinion, any reduction in the MPV after renal transplantation cannot be connected to the mentioned risk factors or immunosuppressive drugs.

References