# Re: Erectile Dysfunction Is Positively Correlated with Mean Platelet Volume and Platelet Count, But Not with Eosinophil Count in Peripheral Blood

Penile erection is a vascular phenomenon, and blood flow via the small vessels of the penis is very dependent to their structural and functional changes. Instead of being thought of as a late result of a localized vascular disease, vasculogenic erectile dysfunction (ED) is nowbeginning to be considered an initial sign of generalized vascular disease. The diagnosis of ED and the succeeding assessment of underlying cardiovascular risk could improve general preventive procedures of vascular health in men.

Erectile dysfunction (ED) is now documented as a marker of greater cardiovascular risk both acutely and chronically and regarded as an early indicator of widespread vascular disease predicting all-cause mortality, cardiovascular mortality, coronary artery diseases (CAD), stroke, and peripheral artery disease in men with and without known CAD.[1] Notably, ED shares with CAD similar risk factors and is principally vasculogenic, demonstrating the common source of endothelial dysfunction.[2]

Vlachopoulos and colleagues[3] have over several years examined the independent link between ED and cardiovascular disease (CVD) using some biomarkers as a means of detecting the men most at risk of a CVD. They evaluated 92 757 subjects and found that ED was correlated with increased CVD and all-cause mortality. The most recent review by Gandaglia and colleagues[4] who carried out a systematic review of the relationship between ED and CVD reported that ED and CVD should be considered as 2 different signs of the same systemic disease with ED usually foregoing CVD, and ED should, then, be regarded as an early marker for CVD of precise significance in the asymptomatic younger men and in those with diabetes mellitus.

There is no uncertainty that the men with ED aged 30 to 60 years are at higher risk of having undiagnosed silent CAD, and it might well be that a combination of biomarkers, vessels wall stiffness, and multidetector cardiac computed tomography is the best modality to evaluate these individuals. It remains vital because the risk is investigated to prompt the cardiologist in particular that it is within their responsibility, and the general physician who detects a patient with ED (asking routinely) should immediately order an assessment of cardiovascular risk system even once symptoms of a CVD are not existing.

With the acknowledgement that endothelial dysfunction is the common contributing factor linking vascular disease to ED came the understanding that ED may not just be a result of vascular disease, particularly CAD, but a harbinger of silent coronary disease-'a sentinel'.[6] Moderate-to-severe but not mild ED in a health screening research was considered to increase the 10 year relative risk of developing CAD by 65% and stroke by 43%.[7] All men with ED and no cardiac symptoms require a detailed cardiac investigation. We should consider of ED as standing for Erectile Dysfunction, Endothelial Dysfunction, and Early Detection.

The common denominator for these speciously different problems is endothelial dysfunction, a principal etiology of ED.[8] In this issue of the Urology Journal, Otunctemur and colleagues have reported for the first time a study of 130 patients with ED. They matched these patients with a control group of normal subjects (n = 100) without clinical evidence of arterial disease and without ED and searched for mean platelet volume (MPV), and platelet count (PC). They found that MPV and PC levels were significantly higher in ED group. An interesting finding in this study is that patients with higher PC (OR = 1.005; 95% CI: 1.003-1.010) and MPV (OR = 1.256; 95% CI: 1.088-1.4) had increased risk for development of ED.

MPV has developed in recent years asa potential independent risk factor for poor clinical outcomes in patients with CAD.[9,10] Because MPV is an indicator of platelet activation and associates with agreeability, larger and hyperactive platelets can speed up the development of intra coronary thrombus and thus play avital role in the pathophysiology of vascular artery disease. In recent years, the idea of MPV as a predictor of an hostile prognosis in acute coronary syndromes was extensively studied, with encouraging results.[11] If such an impressionis valid, then MPV might be a smart prognostic factor, as it is routinely measured as a part of the complete blood cell count (CBC).Measurement of MPV is fast, cheap, and widely available for all physicians.

Despite the broad evidence mentioned above, MPV measurement in clinical practice is hampered by several pitfalls. First, it must be emphasized that of all blood cells, platelets are the most fragile components. It is known that platelet volume increases after blood drawing, particularly in ethylenediaminetetraacetic acid (EDTA)-coatedtubes.[12] Previous investigations also have not yielded us with a consistent cut-off value. The threshold value in researches was usually derived ad hoc using receiver operating characteristic (ROC) curves; less often, it was derived from values in healthy volunteers. According to my knowledge, it varies from 8.9 to 11.5 fL.[13,14] Furthermore, there is a lack of data in specific populations, such as patients later after acute coronary syndrome, where the thrombotic risk is lesser than in the acute phase. Only few researches have focused on such peoples and many of them initiated in the thrombolytic era.[15]

The study of Otunctemur and colleagues published in this issue of the Urology Journal provides us imperative data about the correlation of MPV to increased incidenceof ED. In logistic regression analysis, the MPV was

Mohammad Reza Safarinejad, MD
Clinical Center for Urological Disease Diagnosis and Private Clinic Specializing in Urological and Andrological Genetics, Tehran, Iran.
E-mail: info@safarinejad.com.

confirmed to be the only independent predictor of ED incidence. I would like to highlight the amazing fact that traditional risk factors of ED, such as age, serum lipid profile, hypertension, body mass index (BMI) or diabetes mellitus, did not have any significant impact on ED incidence. Unfortunately, these results were not completely analyzed or discussed by the authors; therefore, the implication of their findings in clinical practice is difficult. If these data will be confirmed in further studies, a rigorous study for underlying mechanisms is needed. Thus, the key point is whether routine measurement of MPV on admission could alter our clinical management, as “statistically significant” does not necessarily imply “clinically significant.” Hence, could the blood level of MPV guide our clinical practice? Or, is it merely a "population" prognosis indicator lacking of "individual" clinical impact? Unfortunately, there is no further study, and these questions cannot be responded to yet; therefore, further studies are required to find the importance of MPV measurement in the clinical evaluation of patients with ED.

Overall, the study of Otunctemur and colleagues offersfurther evidence about the value of MPV measurement in risk stratification of patients with ED. Regardless of all controversies, MPV measurement should not be ignored as a marker of impairedprognosis of patients with ED. Congratulations to authors for their excellent work!

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