

Complete blood cell count components and coronary slow-flow phenomenon

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Dear editor

Despite the implementation of preventive strategies, ischemic heart disease and stroke remain the main causes of mortality and morbidity worldwide.^{1,2} Of the cardiovascular diseases, coronary slow-flow phenomenon (CSFP), with a prevalence rate of 1%–7% among patients undergoing diagnostic coronary angiography, has been found to be associated with cardiovascular events, including cardiac arrhythmia and acute coronary syndrome.^{3–5} However, the potential mechanisms involved in the pathogenesis of CSFP remain unknown. Microvascular and endothelial dysfunctions, inflammation, diffuse atherosclerosis, and increased platelet aggregability have been reported to be the main possible etiologies for CSFP.^{6,7}

We read the article by Altas et al⁸ regarding the relationship between the eosinophil count and the CSFP with great interest. They showed that of the complete blood count components (white blood cells, neutrophils, eosinophils, hemoglobin, and platelets), eosinophils were associated with the CSFP. In this study, eosinophil count was found to be elevated in patients with the CSFP compared with those with normal coronary arteries. In addition, higher eosinophil count was directly correlated with thrombolysis in myocardial infarction frame count in the CSFP group.

When interpreting this study, we should consider some points which are of great importance.

Biomarkers are used as diagnostic tools for risk stratification of cardiovascular diseases, of which complete blood cell count components have been shown to be a good and easily available predictor of cardiovascular events, particularly among patients with coronary artery disease.⁹ In addition to ischemic heart diseases, complete blood cell count components have also been found to be associated with the presence of CSFP; however, study results have been inconsistent in the literature. Several reports have demonstrated that the mean platelet volume is higher in patients with CSFP compared to individuals with normal coronary arteries;^{10–12} however, in one study the elevated mean platelet volume has not been associated with CSFP.¹³ Other platelet volume indices, including platelet distribution width and platelet–large cell ratio, have been associated with the presence of CSFP.⁶ On the other hand, the relationship between leukocyte subtypes and the CSFP has been inconsistent in previous studies. In most of the studies, total white blood cell count has not been the predictor of CSFP.^{6,13–15} In contrast, Akboga et al¹⁶ found that a higher white blood cell count is associated with CSFP. Regarding the red blood cell subtypes, results have also been diverse among studies. Two studies demonstrated that red cell distribution width correlated with the presence of CSFP,^{15,17} while in one study it did not.⁶ Furthermore, the elevated levels of

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hemoglobin¹¹ and hematocrit¹⁵ have been associated with the CSFP. In addition to these regular complete blood cell count components, some novel biomarkers have also been found to be associated with the CSFP, including elevated neutrophil-to-lymphocyte ratio,¹⁴ increased platelet-to-lymphocyte ratio,¹⁶ and decreased lymphocyte-to-monocyte ratio.¹⁸ All these novel biomarkers are inflammation-based and have been useful in diagnosing other inflammatory diseases. White blood cell to mean platelet volume ratio is another novel biomarker examined in coronary artery disease and metabolic syndrome,^{19,20} which may be useful to detect the presence of CSFP as well.

Given the findings of previous studies, it is likely that the two main pathophysiologic mechanisms involved in the development of CSFP may include the enhancement of inflammation status and thrombogenesis. However, due to inconsistent results with regard to the biomarkers, further studies are required to elucidate the pathogenesis of this phenomenon. In addition, the inconsistent findings regarding the complete blood cell count may be explained by the notion that the prevalence of CSFP is low and the majority of studies included small sample sizes. Further large-scale studies, including all these parameters in the final analysis, can clarify this inconsistency.

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