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Factors Influencing the Risk of No-Reflow Development

LETTER TO THE EDITOR

To the Editor,

I read with interest the article titled "The Association Between Atherogenic Index of Plasma and No-Reflow Phenomenon in Acute Coronary Syndrome"[1] published in the latest issue of your journal. I would like to offer my thoughts and evaluations regarding this article. No-reflow phenomenon during percutaneous coronary intervention (PCI) is characterized by a reduction or sudden loss of antegrade coronary blood flow, despite the absence of a clear cause leading to flow loss (such as spasm, dissection, distal macrothrombus, in situ thrombosis, or residual coronary stenosis).[2] Various mechanisms have been considered in the pathophysiology of no-reflow, including distal embolization of plaque and/or thrombus, microvascular damage, reperfusion myocardial injury from oxygen radical production, myocardial necrosis, release of active tissue factor from dissected plaque, vasoconstriction secondary to increased alpha-adrenergic tone, and the release of thromboxane A2 and serotonin from platelets.[2] Even if PCI is technically flawless, flow loss may still occur after stent placement. In acute coronary syndrome (ACS), the likelihood of no-reflow development is higher in cases with a high thrombus burden, prolonged reperfusion time, long and diffuse lesions, and in patients with degenerated saphenous vein grafts (SVG).[3] The incidence of no-reflow during PCI has been reported to be between 12% and 25% in some studies, while specifically evaluating SVG in PCI reveals a no-reflow rate of 15%-42%.[3]

The study design is commendable, but I have some critiques. As mentioned earlier, SVG PCIs are highly associated with no-reflow.[3,4] Differences in the proportions of SVG PCI patients between groups in the study will likely affect the statistical results. However, there is no data in the study about how many of the included patients underwent SVG PCI. If patients who underwent SVG PCI were excluded from the study, adding this information to the exclusion criteria would be beneficial. Additionally, there is no information in the study about the timing of PCI in relation to the minutes of ACSs. As known, the risk of no-reflow increases as the reperfusion time in ACS patients extends.[5] Therefore, it would be helpful to specify whether there is a significant difference in reperfusion time between the groups. Furthermore, lipid profiles of patients may vary depending on whether blood samples were taken in a fasting or postprandial state and at what time. Clarifying the conditions under which these blood samples were collected would be useful.

In conclusion, I believe that paying attention to the points mentioned above will strengthen the study. I extend my thanks to the authors of the study and the esteemed editor who presented us with this valuable work.

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