

Reply to Letter to the Editor: "Unmet Need for Deep Diving into Intracoronary Imaging"

To the Editor,

First, we would like to thank the authors¹ for emphasizing the key points and for their suggestions. In a study involving 7526 patients, the authors highlighted that 45% of acute coronary syndrome (ACS) cases resulted from plaque rupture. They further noted that in the ACS population aged 18-50, the primary histopathological findings in culprit lesions were plaque rupture and plaque erosion on a fibrous plaque background, whereas calcified nodules were observed less frequently.

Intravascular ultrasound (IVUS) did not reveal coronary plaque rupture; however, coronary thrombosis was observed in our case. The patient presented during nighttime hours, and IVUS was unavailable then. Additionally, the IVUS device at our center operates at 20 MHz, and we lack access to a high-definition IVUS device (transducers with higher frequencies, ≥ 45 MHz). It is well known that high-definition IVUS offers better near-field spatial resolution and sufficient tissue penetration for a more precise evaluation of the entire vessel wall.² Additionally, plaque erosion is a significant mechanism in the pathophysiology of ACS. Although optical coherence tomography (OCT) can provide strong evidence of plaque erosion, detecting it through IVUS is challenging. Moreover, even with high-definition IVUS, demonstrating plaque erosion becomes significantly difficult in the presence of thrombus.³

Optical coherence tomography is an imaging technique that is more effective than IVUS in detecting plaque erosion and rupture. However, studies have shown that evaluating plaque characteristics using either OCT or IVUS remains challenging in cases where intracoronary thrombus is present.⁴ In our case, the angiographic examination revealed a large thrombus affecting the distal left main coronary artery, the ostium of the circumflex artery, and the proximal segment of the left anterior descending artery. Platelet aggregation typically occurs at the narrowest point of the lumen during plaque erosion or rupture, indicating that the thrombus in our patient was likely related to underlying coagulation factors.⁵

In conclusion, using OCT or high-definition IVUS could have provided more detailed information to further elucidate the underlying etiology. However, we could not perform an OCT evaluation due to these modalities' unavailability. Considering the clinical, laboratory, and imaging findings available, we believe that the ACS event in our patient was primarily driven by thrombogenic factors rather than plaque erosion.

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LETTER TO THE EDITOR REPLY

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