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Unmet Need for Deep Diving into Intracoronary Imaging

LETTER TO THE EDITOR

To the Editor,

We have read this interesting case report by the authors with great excitement.[1] A 28-year-old man presented with anterolateral ST-segment elevation myocardial infarction after heavy alcohol and energy drink consumption. Coronary angiography revealed a massive thrombus at the left anterior descending (LAD) artery ostium, which was successfully treated with a combination of balloon angioplasty, thrombus aspiration, and potent antiaggregant drugs without stent implantation. In addition, intravascular ultrasound (IVUS) modality was utilized to better understand the characteristics of the culprit lesion (CL).[1] Plaque rupture, thin-cap fibroatheroma, and red thrombus formation are frequently reported in the CLs causing acute coronary syndrome (ACS).[2] In a review of 7526 ACS patients with a mean age of 62.4 years, CL morphologies were investigated using optic coherence tomography (OCT). Accordingly, plaque rupture was the predominant CL morphology and thin-cap fibroatheroma was observed in 45% of all ACS patients.[3] In the ACS population between 18-50 years of age, plaque rupture and plaque erosion on a fibrous plaque background is the main histopathologic finding in the CLs, and calcific nodules are more rarely identified.[4] In an OCT-based prospective-observational study, in histopathologic groups with both plaque rupture and plaque erosion, the frequency of thin-cap fibroatheroma, microchannel, and macrophage density were found to be lower, but fibrous cap thickness and intimal thickness were found to be higher in the ACS population younger than 35 years compared to those older than 35 years.[5]

The author stated that "Control coronary angiography on the 4th day of hospitalization using IVUS guidance demonstrated a thrombus at the proximal segment of the LAD artery without plaque erosion or rupture and Thrombolysis in myocardial infarction (TIMI) grade 3 flow at the LAD artery," but it is essentially known that it is very difficult to even identify plaque rupture with an IVUS device at a frequency of 20 MHz. Therefore, it can be said that the authors' concluding statement "LAD artery without plaque erosion or rupture" cannot be valid for this patient based on these findings. Between 24-32 seconds of the IVUS series in Video 5, a fibrous plaque extending from the proximal LAD to the LAD ostium (90°-270° eccentric plaque) and minimally to the distal left main coronary artery (30°-60°), an estimated 10%-20% plaque volume of the lumen is observed.

In addition, the fibrotic structure of the proximal LAD lesion detected by IVUS, the absence of apparent plaque rupture, and the absence of calcific nodules suggest plaque erosion as a possible pathologic finding. Finally, the low-resolution power of the IVUS device used and the fact that the IVUS recording was taken 4 days after the acute event were estimated as the reasons for the absence of thrombus on intracoronary imaging.

Therefore, the association between intracoronary thrombus and the methylenetetrahydrofolate reductase (MTHFR) mutation described in this case was considered to be a coincidental association, and the MTHFR mutation in this patient was considered to be an innocent bystander in the face of the current acute coronary event.

According to high-resolution IVUS- or OCT-based studies, fibrous plaque is detected in 70% of patients with young-onset ACSs, similar to this case.[3] The

Aykun Hakgör[1]
Berhan Keskin[2]
Seda Tanyeri Uzel[3]
Barkın Kültürsay[4]

[1]*Department of Cardiology, İstanbul Medipol University, Medipol Mega University Hospital, İstanbul, Türkiye*
[2]*Department of Cardiology, University of Health Sciences, Kocaeli City Hospital, Kocaeli, Türkiye*
[3]*Department of Cardiology, University of Health Sciences Kartal Koşuyolu Training and Research Hospital, İstanbul, Türkiye*
[4]*Department of Cardiology, Tunceli State Hospital, Tunceli, Türkiye*

Corresponding author:
Aykun Hakgör
✉ aykunhakgor@gmail.com

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presence of plaque erosion could have been clearly demonstrated in this case, especially if OCT had been used. However, according to basic IVUS principles, the absence of attenuation, ruptured plaque morphology, and calcific nodules in the lesion makes plaque erosion a potential diagnosis in this patient. In conclusion, we think that for the diagnosis of plaque erosion to be more accurate in this case report, 60 MHz IVUS or OCT is required to confirm this diagnosis.

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