

IMAGES IN MEDICINE

Left Main Coronary Artery Bifurcation Thrombus Interventionally Removed

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ABBREVIATIONS

LAD = left anterior descending (coronary artery)

LCx = left circumflex (coronary artery)

LMCA = left main coronary artery

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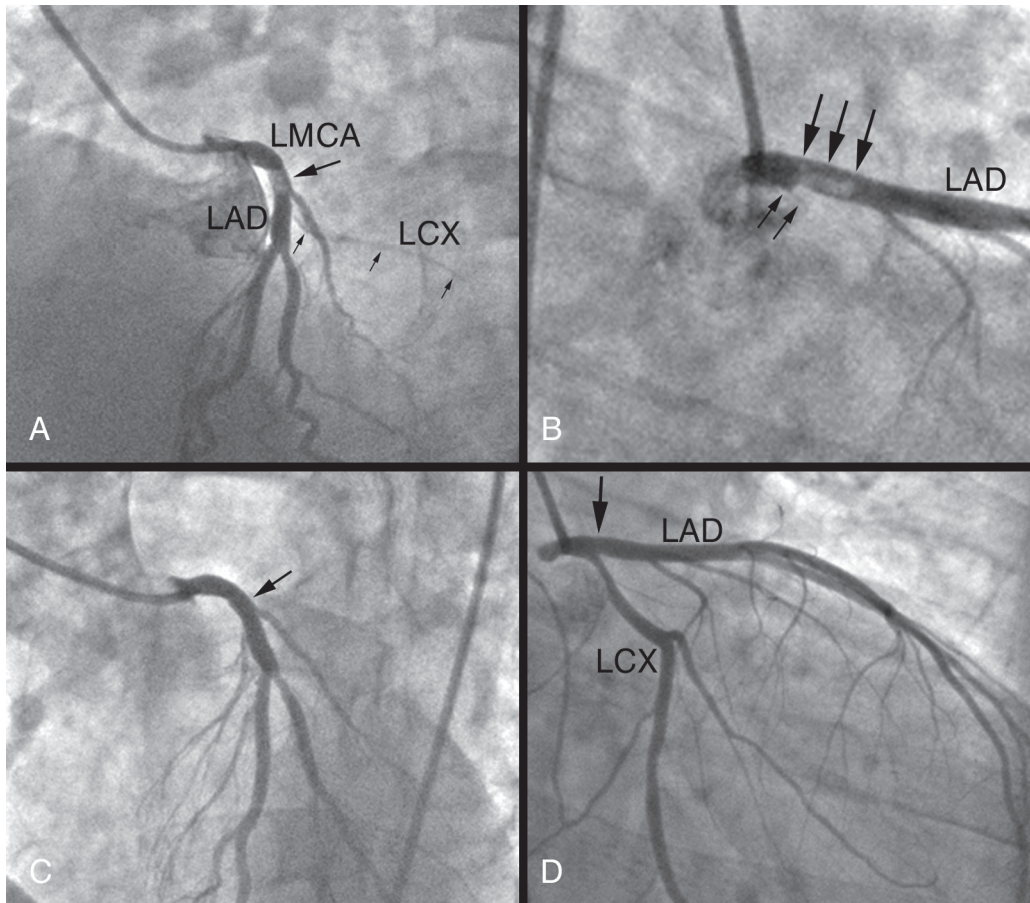
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A 43-year-old male heavy smoker presented with severe chest pain associated with ST elevation in the inferolateral and ST depression in the anterior electrocardiographic leads. Coronary angiography showed the left coronary artery with a thrombus mounted upon the carina of the left main coronary artery (LMCA) bifurcation (Panels A and B, large arrows). The thrombus was protruding into the origin of the left anterior descending (LAD) coronary artery partially occluding it and was also extending into the origin of the left circumflex (LCX) coronary artery occluding it virtually completely (Panels A and B, small arrows). The left coronary artery was otherwise normal as was the right coronary artery.

Due to hemodynamic instability and perceived severe imminent risk for the patient if the LAD were to occlude and since traces of the occluded LCX were hardly visible, the LAD alone was immediately wired and by using the Export aspiration catheter (Medtronic, Minneapolis, MN, USA), the LAD thrombus was removed (Panel C, arrow). The LCX remained completely obliterated (Panel C). During the procedure, a bolus of 10,000 IU of heparin and 600 mg of clopidogrel plus 325 mg of aspirin were administered. Also a glycoprotein IIb/IIIa inhibitor was given for 48 hours. The patient's condition improved and stabilized and his hospital stay was uneventful except for a small q-wave inferolateral infarction as estimated by peak cardiac enzyme values and by echocardiography. He remained on dual antiplatelet treatment. On the eighth day, a repeat coronary angiogram was obtained. The LMCA bifurcation and the LAD were free of thrombus (Panel D, arrow). Most importantly, the LCX was now patent with a TIMI III flow and with no visible thrombus (Panel D). However, a reverse tapering was apparent in the proximal part of the LCX and a mild stenosis downstream.



Heavy smoking, a well-known thrombogenic factor, could be incriminated for the presence of thrombus in this particular patient,^{1,2} albeit other causes of thrombophilia could certainly be culpable. Supportive imaging with either intravascular ultrasound or optical coherence tomography could give further clues to the pathophysiology of the event,^{3,4} however, this would add considerable delay in the performance of the procedure with potential harm. With regards to the initial lack of opacification of the LCX and its subsequent re-appearance, its narrow proximal lumen, in contrast to that of the LAD, was probably easier to be totally occluded by the thrombus and remained occluded by residual thrombus at the end of thromboaspiration. Apparently, this residual thrombus was gradually dissolved endogenously with the assistance of the anti-thrombotic therapy employed and thus patency of the LCx artery was finally restored



as demonstrated in the repeat coronary angiogram performed 8 days later (Panel D). The decision to limit the intervention to a quick thromboaspiration procedure³ of the LMCA/LAD thrombus and avert a catastrophe finally appeared prudent, although a second procedure was contemplated if clinically indicated to recanalize the LCx, which was not finally needed as the LCx was found patent at the repeat angiogram (D). Prompt restoration of flow to the LAD initially saved the patient and either quick reperfusion of the LCx aided by antithrombotic therapy (particularly use of a IIb/IIIa inhibitor) and/or opening of collaterals probably led to a limited infarct size.

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