Vasospastic angina, plaque erosion, ischemia, and cardiac arrest: Four of a kind or a straight?

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DOI: 10.33963/v.kp.96979

Received:
May 4, 2023

Accepted:
August 11, 2023

Early publication date:
August 24, 2023

A 56-year-old woman, a current smoker with a history of arterial hypertension and dyslipidemia was previously examined due to recurrent syncope with associated chest pain in recent months. Normal cardiac morphology and function were confirmed by echocardiography, and coronary computed tomography angiography showed a noncalcified plaque with 40% stenosis in the left anterior descending coronary artery (LAD) (Figure 1A). Single antiplatelet therapy and a statin were started.

One month later, she was admitted to the intensive care unit after resuscitation following out-of-hospital cardiac arrest. Post-return of spontaneous circulation electrocardiogram showed ST-segment elevation in the anterior leads, which led to emergent coronary angiography, but no obstructive coronary artery disease was found.

On admission, the patient presented satisfactory neurological and cardiac development with significantly increased troponin levels. Anterior myocardial edema (Figure 1B) was found on T2 mapping images without late gadolinium enhancement (Figure 1C) on cardiac magnetic resonance. Due to suspicion of vasospastic angina, an acetylcholine (Ach) invasive provocation test was performed. The first Ach bolus of 20 mcg in the LAD induced chest pain along transient ST-segment elevation in the anterior leads and severe spasm in the mid anterior descending coronary artery (Figure 1D–E) relieved by intracoronary nitroglycerin. In addition, optical coherence tomography assessment of the LAD revealed a small erosion in the mid segment (Figure 1F). Dual antiplatelet therapy, dihydropyridine calcium channel blocker, and long-acting nitrates were started, and a subcutaneous implantable cardioverter defibrillator was implanted in secondary prevention.

We have reported a case of an out-of-hospital cardiac arrest secondary to acute ischemia due to vasospastic angina and with the additional finding of plaque erosion. In this scenario, a causative role of spasm leading to plaque erosion has been proposed (a straight), and we believe that explanation is more feasible than the simultaneous coincidence of several factors (four of a kind).

Article information

Conflict of interest: None declared.

Funding: None.

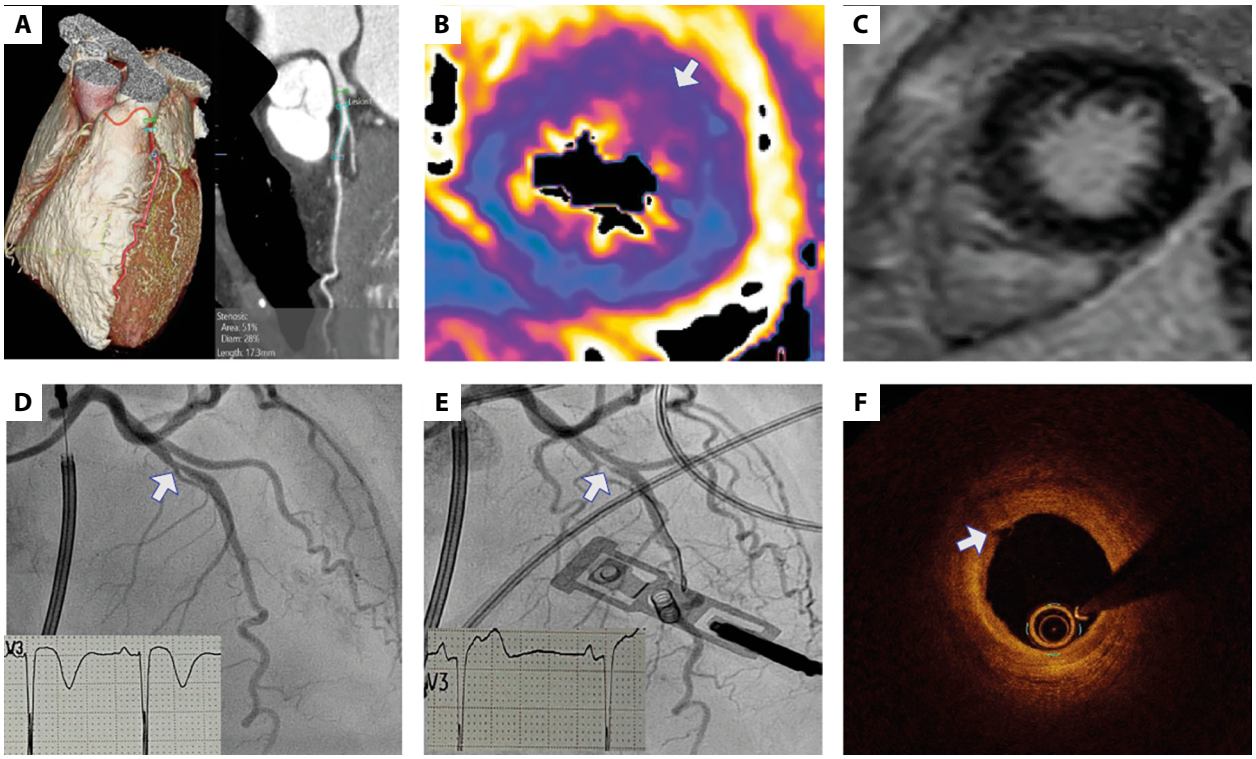

Figure 1. A. Computed tomography angiography; noncalcified plaque with 40% stenosis in the left anterior descending coronary artery. **B, C.** Cardiac magnetic resonance; anterior myocardial edema on T2 mapping (**B**) without late gadolinium enhancement (**C**). **D, E.** Severe spasm in the mid-anterior descending coronary artery. **F.** Optical coherence tomography, small erosion in the mid segment

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