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Case Report: Hidden Dissection of the Left Descending Coronary Artery Mimicking Tako-Tsubo Cardiomyopathy

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Summary

A 44-year old woman was admitted to hospital with an acute coronary syndrome ten days after an acute thoracic trauma. The promptly performed coronary angiography showed plane coronary arteries. Due to the hypokinetic anterior wall a Tako-Tsubo cardiomyopathy was supposed.

A few days later the patient again emerged massive chest pain and ST-segment elevations. In the coronary angiography now a spontaneous dissection of the left descending coronary artery could be shown. The current case demonstrates the importance of clarifying ambiguous myocardial infarctions by using diagnostic tools such as intravascular ultrasound or optical coherence tomography.

Case Report

A 44-year-old woman without classical cardiovascular risk factors was referred to the coronary care unit (CCU) with severe angina pectoris lasting for 90 min. The initial ECG demonstrated significant elevations of ST segments in leads V2–V5 and distinct Q waves were already apparent in leads V1–V3 (Fig. 1a). Also cardiac enzymes were slightly elevated with troponin T of 16.3 ng/l. Coronary angiography (CAG) revealed mostly plane coronary arteries without any significant stenosis, whereas the mid part of the left descending coronary artery (LAD) appeared somewhat slightly hazy (Fig. 1b). The global systolic left ventricular function was moderately reduced (ejection fraction of 45%). Because of a severe hypokinesis of the anterolateral, apical as well as parts of the inferior segments, a Tako-Tsubo cardiomyopathy was supposed. This suspicion was strengthened by the presence of enhanced contraction of the anterobasal and posterobasal segments as well as by the fact that the patient was female. In addition, the patient had suffered stress, having tumbled off a bicycle [1, 2] (Fig. 1c). A conservative treatment approach was decided upon; therapy was initiated with a beta blocker, ACE inhibitor and statin, and continued with acetyl salicylamide 100 mg and clopidogrel 75 mg. The patient reported being free from chest pain, and the ECG showed the stage of a subacute myocardial infarction with deep negative T-waves in the chest leads V2–V6 (Fig. 2a).

On day 3 of hospital stay, she again emerged massive chest pain and presented new significant elevations of ST segments in the chest leads V2–V5 (Fig. 2b). Troponin T levels rose to 828 ng/l. Coronary angiography was performed again and revealed now a spontaneous dissection of the mid-LAD with subsequent complete occlusion of the LAD (Fig. 2c). An attempt at revascularisation by percutaneous transluminal coronary angioplasty failed, and the decision was made in consultation with cardiac surgeons to continue with conservative therapy with dual antiplatelet therapy, beta blocker, ACE-in-

hibitor, and a statin. The decision was based on the triple event with already distinct Q-waves in leads V2–V4.

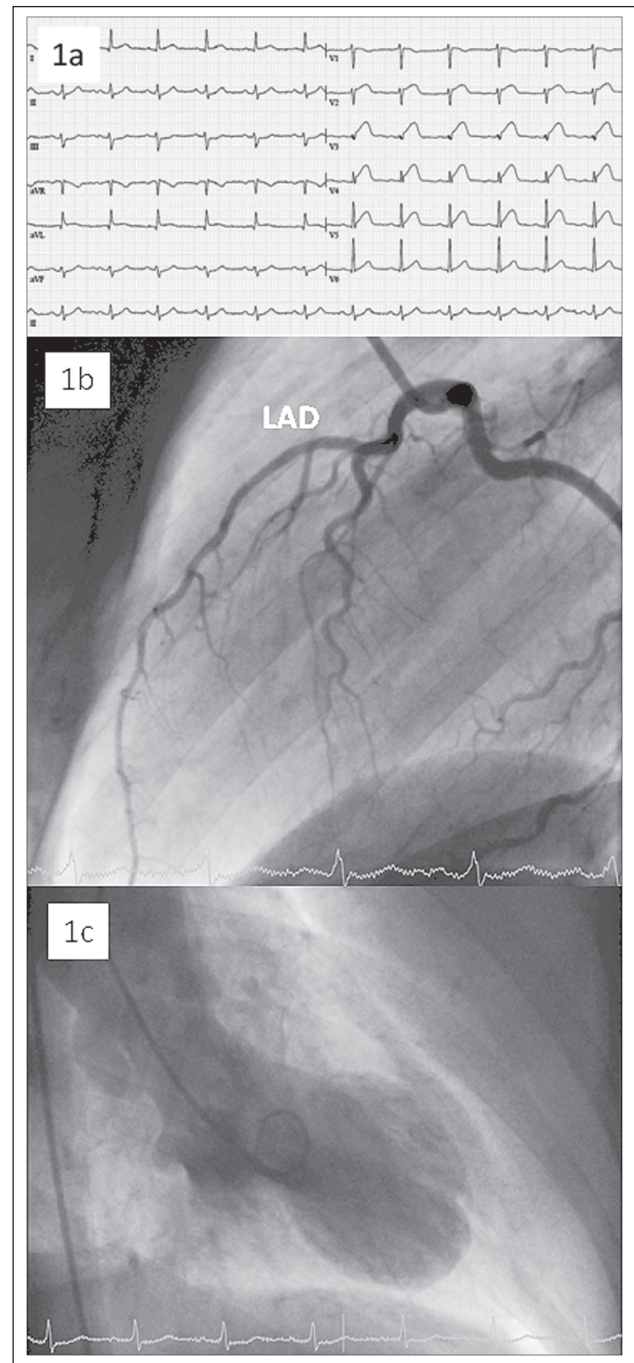


Figure 1. (a): Initial ECG showing anterior STEMI; (b): First coronary angiography showing no obstruction/stenosis of the left descending coronary artery (LAD). Right coronary artery and circumflex artery also showed no abnormalities; (c): Global left ventricular function was reduced moderately with anterolateral, apical as well as inferior hypokinesis.

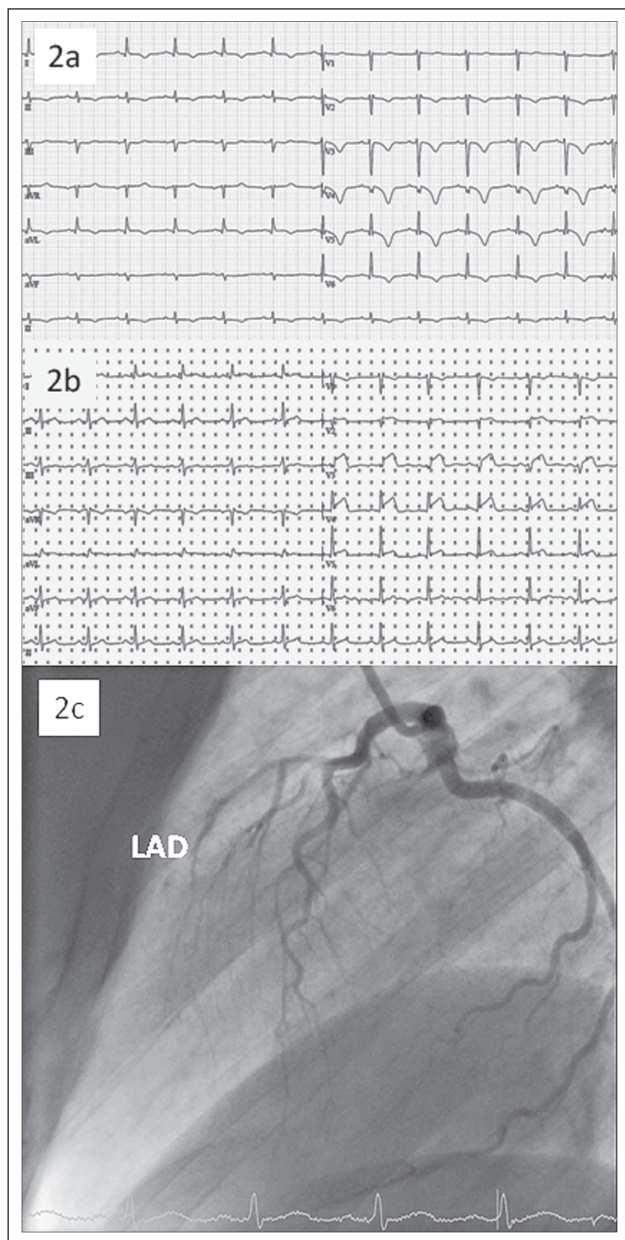


Figure 2. (a): ECG one day after the first coronary angiography showing subacute anterior STEMI; (b): ECG three days after the first coronary angiography showing again significant anterior ST segment elevations; the patient once again developed massive retrosternal chest pain; (c): Second coronary angiography revealed complete occlusion of the proximal LAD due to spontaneous dissection.

The case history of the patient showed that she had suffered blunt thoracic trauma about ten days before admission to the CCU; she had hit her chest against the handlebars of the bicycle with which she had jumped into a lake in playful spirit, and had developed severe chest pain.

It is likely that the prolonged and massive chest pain she suffered was caused not only by musculoskeletal trauma *per se*, but also by an acute myocardial infarction due to a coronary artery dissection. This supposition is supported by the fact that, when she again experienced the same kind of oppressive chest pain about ten days later for which she was admitted to hospital, in the ECG slight Q waves in V1–V3 were already present, suggesting that a myocardial infarction had taken place in a part of the anterior wall.

On admission to hospital for the intermittent angina pectoris occurring again ten days after the trauma, recurring myocardial infarction was diagnosed. Strikingly, no stenotic coronary artery disease could be seen at that time. In retrospect the dynamic dissection of the LAD was not visible at that time.

Despite ongoing dual antiplatelet therapy, extensive dissection of the LAD progressed to complete occlusion, which was revealed by the second coronary angiography.

After the initial, inconspicuous coronary angiography, and despite markedly elevated cardiac enzymes, Tako-Tsubo cardiomyopathy was suspected. However, when the patient three days later again showed symptoms of acute myocardial infarction, a second coronary angiography was performed, revealing dissection of the LAD.

The patient herself considered her chest pains after the blunt thoracic trauma as being musculoskeletal in character; however, they were no doubt compounded by myocardial infarction she had suffered. She was advised to continue therapy with aspirin, clopidogrel, beta blocker, ACE inhibitor, and statin.

Cardiac magnetic resonance imaging performed four days after the second coronary angiography revealed a transmural late enhancement pattern of the pronounced akinetic anterior myocardial wall showing early and late microvascular obstruction with a left ventricular ejection fraction of 40% and elevated end-systolic volume.

Coronary artery dissection is a rare clinical condition with variable causes including trauma, iatrogenic lesions from angiography and spontaneous dissections [3, 4]. The possibility of severe injury to the heart even after primarily blunt chest trauma requires careful follow-up monitoring and, if indicated, extensive non-invasive as well as invasive diagnosis and treatment. The current case demonstrates the clinical importance of clarifying ambiguous myocardial infarctions by using, if necessary, diagnostic tools such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT).

Disclosures

None.

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