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Letter to the Editor

Multiple and recurrent spontaneous coronary artery dissection: Angiographically visible and invisible dissections in the same patient

Shams Y-Hassan [a,*], Kerstin Cederlund [b], Brynjölfur Mogensen [a], Loghman Henareh [a]

[a] Karolinska Institute at Karolinska University Hospital-Huddinge, Department of Cardiology, Sweden
[b] Department of Radiology, Karolinska University Hospital Huddinge and Department of Clinical Science, Intervention and Technology, Division of Medical Imaging and Technology at Karolinska Institutet, Stockholm, Sweden

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Spontaneous coronary artery dissection (SCAD) is an under-recognized and often missed cause of acute coronary artery disease, which may be fatal during the acute stage. Once the acute phase has passed, the prognosis of the disease is favorable. The first angiographic case of SCAD was reported in the right coronary artery in 1978 in a 55-year-old man, who survived an acute myocardial infarction [1]. The disease is predominantly afflicting women and commonly associated with a predisposing arteriopathy and a precipitating stress factor [2,3]. SCAD accounts for almost a quarter (24%) of myocardial infarction in young women [4]. Herein, we report on a case of multiple SCAD triggered by an emotional stressor and presented with acute chest pain. She developed recurrent silent SCAD detected during follow-up coronary angiography (CAG).

49-year-old woman presented with a sudden onset of chest pain after learning a very disappointing social notification. Her past history was unremarkable. Her parents had ischemic heart disease at the age of 50 years. She was a chronic smoker. The patient developed an attack of syncope prior to hospital arrival. The chest pain lasted more than one hour. The first electrocardiogram (ECG) showed no remarkable changes. On clinical examination, the patient was sad; she had regular pulse 70/min, the blood pressure was moderately elevated 170/93 mm Hg. The auscultation of the heart and lungs were normal. There was mild to moderate elevation of the cardiac biomarker in the form high sensitivity troponin T max 689 ng/L 5 h after admission. Conventional treatment for non-ST-elevation myocardial infarction (NSTEMI) was initiated. Invasive CAG one day after admission revealed stenosis in a long segment of left anterior descending artery (LAD) from the middle part to the apical segment with radiolucent line causing multiple lumen and stagnation of contrast dye at some parts of the vessel wall (Fig. 1A, B, and C white arrows); these changes were consistent with spontaneous coronary artery dissection (SCAD). A segment of the left main stem, the proximal part of LAD and the first 2–3 cm of the first large diagonal branch were relatively narrower than the normal parts (Fig. 1A, B, and C black arrows). The left circumflex artery (LCx) and the right coronary artery (RCA) were normal (Fig. 1A, B, C and D). The LAD was open and the patient was in a stable clinical condition. A conservative treatment strategy was adopted. Because of an episode of chest pain, the CAG was repeated 3 days after the first CAG. The previous findings in the coronary arteries were unchanged. The patient continued on dual antiplatelet therapy, β-blockers and angiotensin converting enzyme inhibitor and discharged one week after the admission day. Follow-up CAG 11 weeks after the first CAG showed complete normalization of the left main stem, the proximal part of LAD and the diagonal branch (Fig. 2A, B, and C black arrows). There was also healing of the major parts of the previously visible SCAD in the middle

* Corresponding author at: Karolinska University Hospital, Huddinge, Department of Cardiology, S-141 86 Stockholm, Sweden.
E-mail address: shams.younis-hassan@karolinska.se (S. Y-Hassan).

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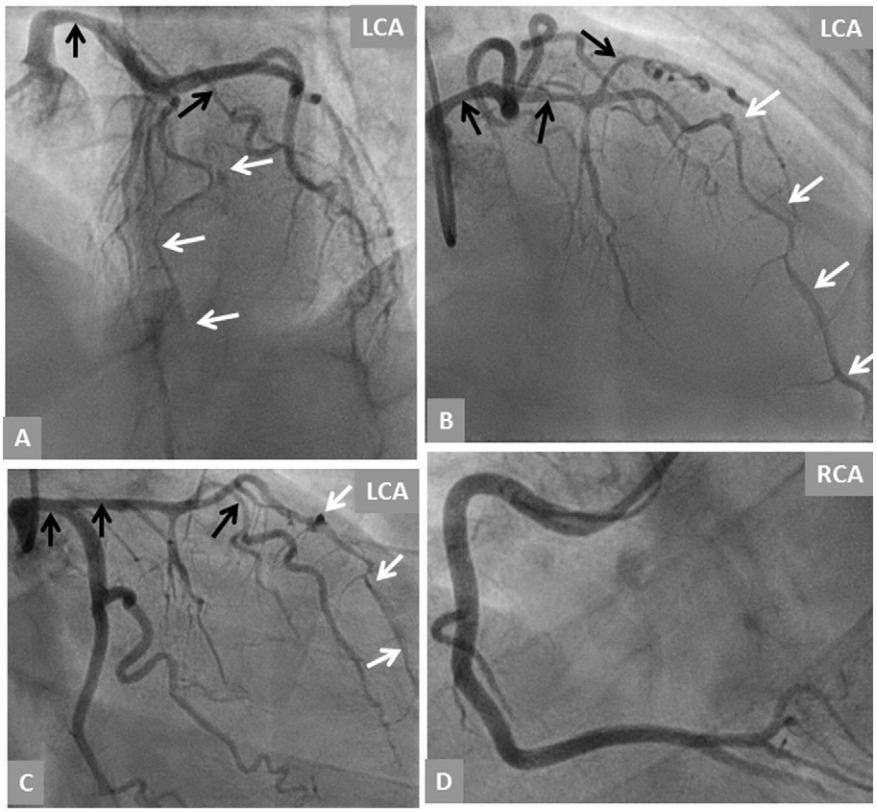

Fig. 1. Coronary angiography, left coronary artery (LCA) in the cranial left anterior oblique (LAO) (A), in the cranial right anterior oblique (RAO) (B) and in the caudal RAO (C) reveals both visible spontaneous coronary artery dissection (SCAD) in the left anterior descending artery (LAD) (white arrows) and non-visible SCAD in the left main stem, the proximal part of LAD and the first large diagonal artery (black arrows). The right coronary artery (RCA) is normal (D).

and peripheral segments of the LAD; only a short segment of LAD showed a white line (intimal flap) but no staining of the vessel wall with the contrast dye (Fig. 2A and B white arrow). Unexpectedly the right CAG showed appearance of multiple radiolucent lines causing multiple lumens in the proximal and the middle segments of right coronary artery but there were no signs of contrast stagnation in the vessel wall indicating that changes were most probably partially healed SCAD in the right coronary artery, which had normal blood flow. These angiographic findings were confirmed by multi-slice computed tomography (CT) angiography (Fig. 3A and B thin white arrows). In the left coronary artery, an intimal flap was seen in the left main stem confirming that the previous invisible SCAD involved even the left main stem and a similar intimal flap was seen in the middle part of LAD (Fig. 3C white arrows). There were no signs of atherosclerotic plaque in the coronary arteries. The patient denied any history of severe chest pain during the 11 weeks between the coronary angiographies. Treatment with a conservative strategy was continued and she was asymptomatic during follow-up 8 months after the initial presentation. Informed consent has been obtained from the patient to publish the case.

Many interesting points in the SCAD of the present patient deserve discussion; the SCAD was multiple, recurrent, symptomatic during the index presentation, silent during recurrence; It was visible in some segments and invisible in other segments. A predisposing factor such as fibromuscular dysplasia (FMD), connective tissue disorders, systemic inflammatory conditions, hormonal therapy was not found in the patient apart from being a woman. There was no evidence of FMD in the renal arteries examined by multislice CT angiography. The initial SCAD in our patient was precipitated by an emotional factor where the patient suffered chest pain suddenly after a psychological trauma. Among the reported precipitating factors for the SCAD in the literature are intense emotional stressors (as death in the family, breakdown of marriage), physical stressors (intense aerobic workouts, heavy weight lifting), bearing down activities (intense coughing, retching/vomiting), and drugs (methamphetamines, cocaine) [5].

The left CAG showed signs of both visible and invisible SCAD. The CAG confirmed the diagnosis of type 1 SCAD according to Shaw et al.'s [6] classification with angiographically visible signs of SCAD with longitudinal radiolucent lines produced by the intimal flaps with two lumens of different radio-opacity and hang-up of contrast dye in some parts of the vessel wall. These changes were clearly seen in the middle part of LAD. However there was some smooth narrowing of a segment of the left main stem, proximal part of LAD and the first few centimeters of a diagonal branch but had no visible angiographic signs of SCAD. These segments of the left coronary artery revealed also to be SCAD. The evidences for that were the following: the changes did not respond to intracoronary nitroglycerin injection during the acute stage; they were completely normalized during follow-up CAG. In addition, multi-slice CT angiography during follow-up showed signs consistent with healed SCAD involving the left main stem. It should be acknowledged that invisible SCAD in the current patient was not suspected during the initial CAG. The invisible SCAD, which involved the left main stem, proximal segments of LAD and the diagonal branch is consistent with type 2 SCAD according to Saw et al.'s [6] classification. Type 2 SCAD is actually the most common type of SCAD comprising 67% of SCAD lesions [4] and needs to be recognized by the angiographers. The diagnosis of invisible SCAD can be confirmed by endovascular image studies such as intravascular ultrasound or optic coherence tomography [2] but the interventionists should be aware of the potential risks in association with endovascular imaging [7].

Unexpectedly the right coronary artery, which was completely normal during the index presentation showed multiple radiolucent lines in the proximal and middle parts of right coronary artery dividing the vessel into multiple lumens. The right coronary SCAD extended even into