

Case report: spontaneous coronary artery dissection and suspicion of takotsubo cardiomyopathy in a patient presenting with T-wave inversions, severe QTc prolongation, elevated cardiac biomarkers, and apical akinesia

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Received 14 August 2019; first decision 4 September 2019; accepted 27 May 2020; online publish-ahead-of-print 28 August 2020

Background

In patients suspected of acute coronary syndrome, but where the coronary angiography (CAG) has shown unobstructed coronary arteries differential diagnoses include spontaneous coronary artery dissection and takotsubo cardiomyopathy. This case report presents a patient with spontaneous coronary artery dissection but diagnostic signs suspicious of takotsubo cardiomyopathy. Which leads to a consideration of the co-existence of the diseases.

Case summary

A 57-year-old woman was acutely admitted to the emergency ward with sudden development of chest discomfort, palpitations, and dyspnoea. At hospitalization, the electrocardiography showed T-wave inversions in I, aVL, and V2, and Troponin I was elevated. Initial echocardiography revealed apical akinesia consistent with takotsubo cardiomyopathy. Initially, a diagnosis of acute coronary syndrome or takotsubo cardiomyopathy was suspected. The patient was further diagnostically assessed with CAG including optical coherence tomography which showed spontaneous coronary artery dissection in the left anterior descending artery. At follow-up 3 months later, CAG showed a fully healed coronary artery, and repeated echocardiography showed normalization of the left ventricular function.

Discussion

In this case report, initially, acute coronary syndrome was suspected due to electrocardiography with T-wave inversions and elevated cardiac biomarkers. Takotsubo cardiomyopathy was suspected when echocardiography showed apical ballooning, but CAG with optical coherence tomography revealed a spontaneous coronary artery dissection. Interestingly no severe obstructions of coronary arteries were seen, and follow-up echocardiography showed fully regained myocardial function. This leads to the debate as to whether this might be a case of co-existing spontaneous coronary artery dissection and takotsubo cardiomyopathy.

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Handling Editor: Domenico D'Amario

Peer-reviewers: Ruben Casado Arroyo, John Kanakakis and Luca Arcari

Compliance Editor: Rahoul Mukherjee

Supplementary Material Editor: Deepti Ranganathan

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Keywords

Case report • Spontaneous coronary artery dissection • Takotsubo • Acute coronary syndrome • QTc prolongation • Apical akinesia

Learning points

- Spontaneous coronary artery dissection may co-exist with or lead to takotsubo cardiomyopathy.
- A conservative approach is recommended¹ in spontaneous coronary artery dissection where acute revascularization is not mandated. This case report supports this treatment strategy.

Introduction

This case report presents a patient with spontaneous coronary artery dissection (SCAD) who presented with signs suspicious of takotsubo cardiomyopathy.

Patients with SCAD often present with chest pain (60–90%), electrocardiographic (ECG) abnormalities with ST-elevation myocardial infarction or non-ST-elevation myocardial infarction like changes and positive cardiac biomarkers.¹ Spontaneous coronary artery dissection is defined as acute development of a false lumen in the outer third of the tunica media layer of the coronary artery. Spontaneous coronary artery dissection is considered to develop due to two different pathophysiologic mechanisms. *The inside-out model*: where the lumen is formed by a tear in the coronary artery endothelium and tunica intima layer allowing blood to enter from the true lumen. *The outside-in*: where the lumen is formed by a tear in the vasa-vasorum bleeding directly in the tunica media layer. In either situations, the developing false lumen dissects the coronary artery and may compress the true coronary artery lumen leading to acute ischaemia.¹ Spontaneous coronary artery dissection has been found in 2% of coronary angiographies (CAG) performed in patients suspected of having acute coronary syndrome (ACS). Patients with SCAD are typically younger than ACS-patients (~50 years old), and the majority are females (up to 80%).² The prevalence of SCAD in women under 50 years of age with signs of ACS has been found to be ~30%.^{3–5} Spontaneous coronary artery dissection patients, typically, have fewer traditional cardiovascular risk factors than patients with atherosclerotic coronary disease.¹

In takotsubo cardiomyopathy, the patients typically present with chest pain often in relation to severe emotional or physical stress. The ECG can present with ST-elevations, ST-depressions, T-wave inversions, and prolonged QTc. Almost every patient has elevated cardiac biomarkers. In classical cases of takotsubo cardiomyopathy, the echocardiographic changes are apical hypo-/akinesia and apical ballooning.⁶ The pathophysiological mechanisms behind takotsubo cardiomyopathy are believed to be myocardial injury caused by increased sympathetic stimulation and catecholamines.⁷

Timeline

Day 1	Patient experienced dyspnoea, chest pain, and palpitations, and she was acutely admitted to the emergency ward. The electrocardiography (ECG) showed lateral T-wave inversions in I, aVL, and V2, coronary biomarkers were elevated, and echocardiography showed apical akinesia and left ventricular ejection fraction (LVEF) of 40%. Due to suspicion of acute coronary syndrome (ACS), antiplatelet and anticoagulant therapy was initiated.
Day 2	Transferred to an invasive centre for further work-up. Progression of ECG abnormalities with T-wave inversions in V2–V6, I, II, and aVL and QTc prolongation to 657 ms.
Day 3	Computed tomography showed mid-left anterior descending artery (LAD) with long stenosis 90% and distal LAD suspicious of stenosis. Coronary arteriography (CAG) with optical coherence tomography (OCT) revealed a mid-/distal LAD with a long dissection and near-complete intimal tear without any luminal entrance. (Type 2b spontaneous coronary artery dissection). Dual antiplatelet therapy was continued, and due to reduced LVEF ACE-inhibitor and beta-blocker treatment initiated.
After 2 months	New echocardiography was performed and showed remission of apical akinesia and normalized LVEF 60%
After 3 months	New CAG with OCT: showed normal coronary arteries with a completely healed LAD without any stenoses or false lumens.

Case presentation

A 57-year-old woman was acutely admitted to the emergency ward after acute onset of pain in her left arm accompanied by

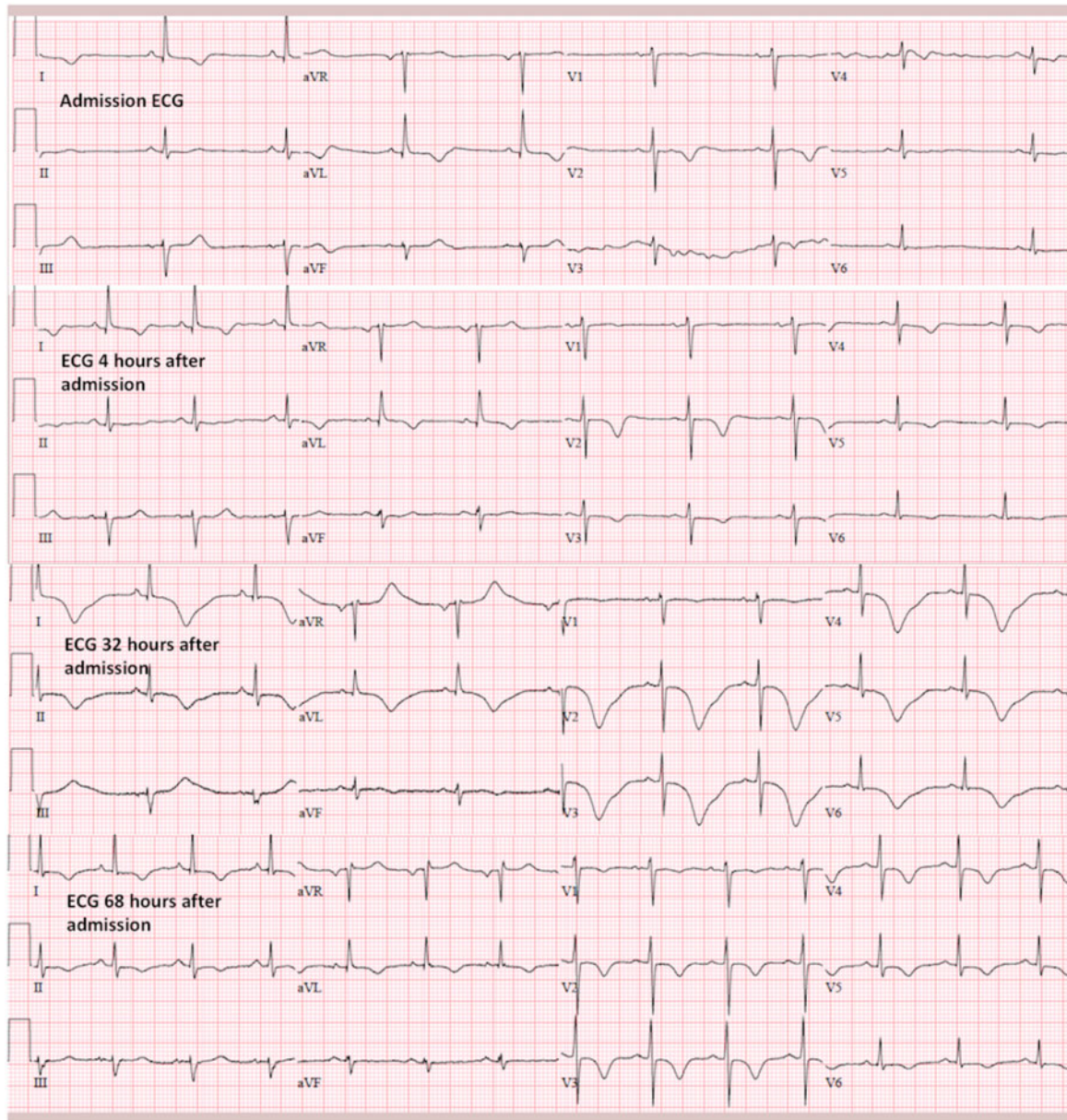


Figure 1 Showing from the top: electrocardiography at hospitalization, 4 h after hospitalization, 32 h after hospitalization, and 68 h after hospitalization.

chest discomfort, dyspnoea, and palpitations during sedentary work. At her general practitioner, her blood pressure had been elevated to 190/100 mmHg. Nitroglycerine had been administered, which had decreased the pain and chest discomfort. She had no previous medical history other than the use of propranolol for tremor. With regards to cardiac risk factors, only prior smoking was noted.

At the emergency ward, the ECG showed sinus rhythm with T-wave inversions in lead I, aVL, and V2 (Figure 1). The first troponin I (TnI) was 2910 ng/L (ref. level: normal < 15). Anticoagulant and antiplatelet therapy was prescribed as acetylsalicylic acid 300 mg,

ticagrelor 180 mg, and subcutaneous low-molecular-weight heparin (Fondaparinux) 2.5 mg. She was started in atorvastatin 80 mg and administered morphine and nitroglycerine as needed. Her pain remitted completely shortly after hospitalization. Second TnI was 3940 ng/L, and an additional ECG showed progression of the T-wave inversions in lead V1–V6 (Figure 1). Echocardiography was performed and showed mid-apical akinesia and apical ballooning. The estimated left ventricular ejection fraction (LVEF) was 40%. Strain analysis was performed and global longitudinal strain score (GLS) was found decreased at -13.2% (Figure 2). Due to the abnormalities found on the echocardiography takotsubo cardiomyopathy was suspected

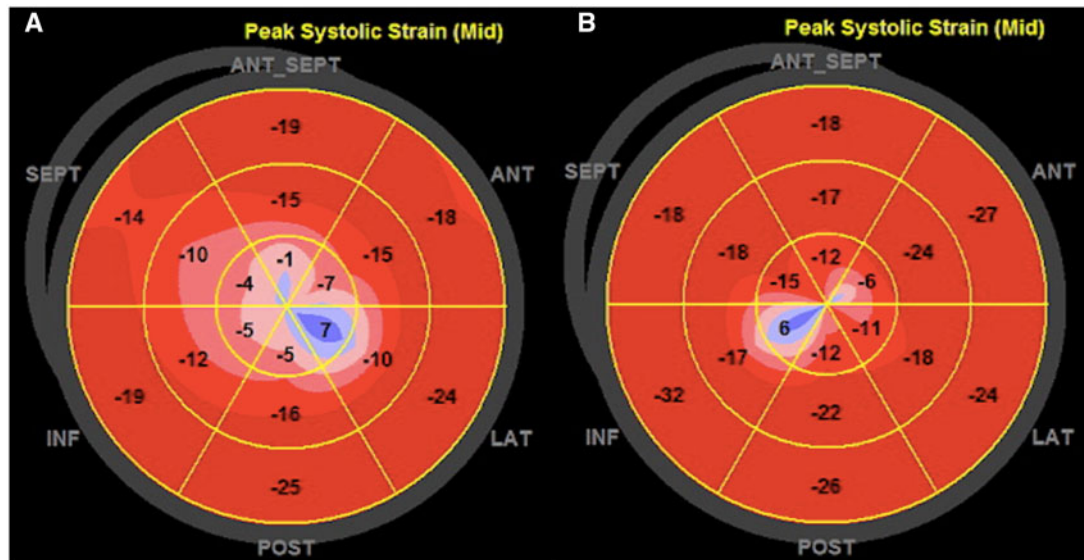


Figure 2 (A) Bull's eye from the initial echocardiography where the global longitudinal strain score is -13.2%. (B) Bull's eye from echocardiography at follow-up where the global longitudinal strain score is -17.9%.



Figure 3 Computerized tomography coronary angiography showing longitudinal stenosis of the left anterior descending.

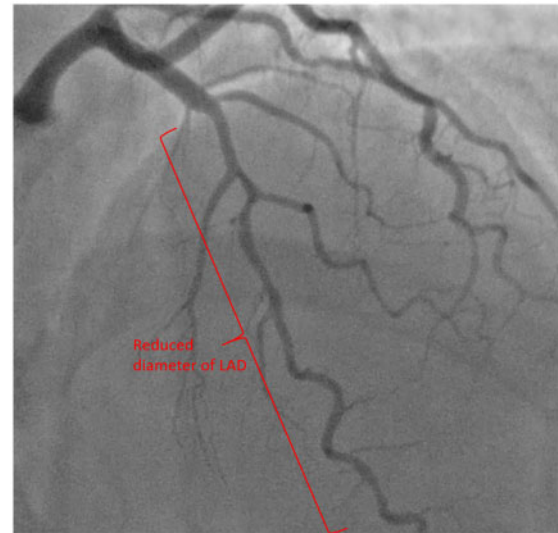


Figure 4 Showing the coronary angiography where shortly after the ostium of the first diagonal in the left anterior descending artery a sudden reduced diameter of the artery is noted.

and, for further assessment, she was transferred to a cardiac invasive centre for subacute CAG.

At the invasive centre, ECG showed extensive T-wave inversions in V2–V6, I, II, and aVL with QTc prolongation to 657 ms. (Figure 1). Third round of Tnl had decreased to 920 ng/L. Due to enrolment in a

current clinical trial, very early computed tomography (CT), a CT-CAG was performed which showed an Agatston-score of 0, suspicion of a long 90% stenosis of the mid-left anterior descending artery (LAD), and suspicion of stenosis in the distal LAD (Figure 3). Coronary angiography was performed which showed a 'step-down' of the diameter of the mid-LAD lumen. (Figure 4). An optical coherence tomography (OCT) was therefore performed to visualize and

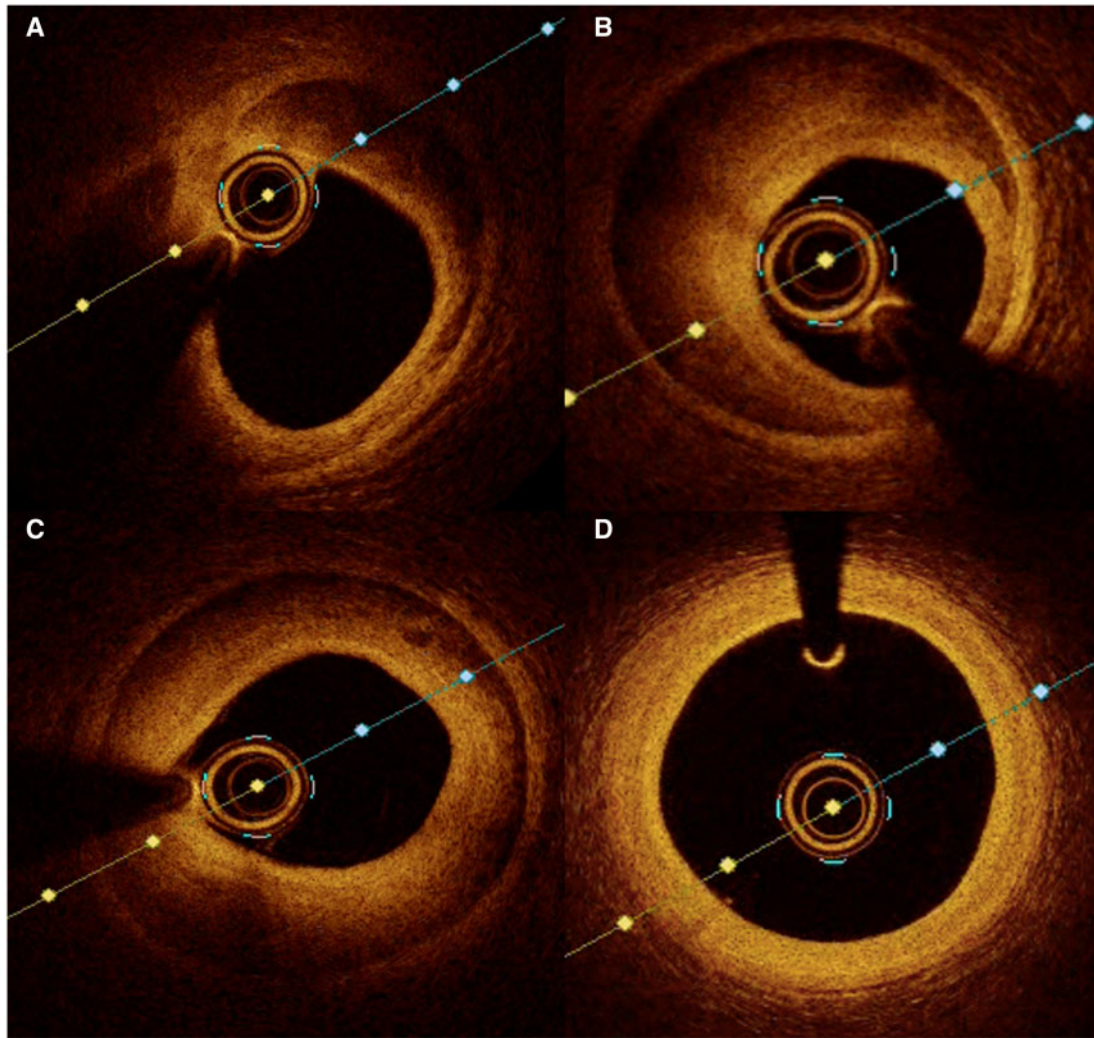


Figure 5 (A, B, C) Optical coherence tomography scan, where a false lumen is seen in the tunica media of the left anterior descending artery thereby showing spontaneous coronary artery dissection. (D) Repeated optical coherence tomography scan 3 months later showing a fully healed coronary artery with no false lumen.

diagnose the flow-limitation further. The OCT showed an extensive coronary artery dissection beginning in the mid-LAD, with a near-complete intimal tear, without any suspicion of bleeding entering from the true coronary lumen. No obstructions of side branches from the LAD were seen (Figure 5). The CAG was, otherwise, without atherosclerosis. The patient was therefore diagnosed with SCAD type 2b according to the Saw *et al.* classification.¹ According to current guidelines,¹ a conservative strategy was chosen and dual antiplatelet therapy (DAPT) treatment was continued. Because of LVEF of 40%, heart-failure medication with ACE-inhibitor (Ramipril 1.25 mg \times 2) and beta-blocker (metoprolol succinate 25 mg \times 1) was initiated and planned up titrated in the heart-failure outpatient clinic. Approximately 3 days after hospitalization, the T-wave inversions decreased and QTc prolongation was reduced to 476 ms; however, the ECG was still with remaining T-wave inversions in I, II, aVL, and V1–V6 (Figure 1).

Two months after, repeated echocardiography showed a normalized LV function with LVEF of 60% and a normalized GLS-score at -17.9% (Figure 2). Three months after a repeated CAG was performed which showed normal coronary arteries with no stenosis in the LAD. Optical coherence tomography was performed and showed the dissection in the LAD to be completely healed without any false lumen (Figure 5). The ECG was also normalized without any T-wave inversions. After the repeated CAG, atorvastatin, metoprolol succinate, and DAPT treatment was discontinued.

Discussion

In this case, several diagnostic signs suspicious of takotsubo cardiomyopathy were seen. These included ECG with T-wave inversions and prolonged QTc and only transient echocardiographic abnormalities.

As the initial echocardiography showed mid-apical ballooning and reduced ejection fraction to 40%, but the follow-up echocardiography, 2 months later, showed normalization of ejection fraction and myocardial function.

Interestingly, the LAD was only moderately narrowed and no obstructions of the side branches from the LAD were found. This leads to the consideration as to whether the akinesia is fully explained by the dissection. One might consider if this patient was presenting with the co-existence of takotsubo cardiomyopathy and SCAD. The suspicion of this co-existence has been reported earlier.

Y-Hassan and Bohm⁸ reported a case with SCAD in the largest diagonal artery. In that case, ventriculography showed apical ballooning and hypokinesia. They performed cardiac magnetic resonance (CMR) imaging which revealed late gadolinium enhancement (LGE) in a limited area of the lateral apical region, with no LGE in the remaining hypokinetic area, which led to the suspicion of co-existing takotsubo cardiomyopathy. A LGE-CMR scan might have helped to further assess as to whether the elevated biomarkers and reduced LVEF, in this case, was due to takotsubo cardiomyopathy or SCAD induced myocardial ischaemic injury.

Conclusion

In this case report, we present a case story of a middle-aged woman with only prior smoking as cardiovascular risk factor. She presented with chest discomfort and pain in her left arm and after CAG with OCT she was diagnosed with SCAD. Due to apical ballooning with reduced ejection fraction, a suspicion of takotsubo cardiomyopathy was also raised. One might therefore consider if this is a case of co-existing SCAD and takotsubo cardiomyopathy. At follow-up, both the ventricular function and the coronary artery were normalized. This case thereby represents a good outcome of a conservative treatment strategy.

Lead author biography



Kåre Peter Frederiksen graduated in medicine from the University of Copenhagen. Through basic clinical training (intern year), he employed at an emergency ward and in General Practice. Then, he employed in a 1-year introduction position in internal medicine (residency) at the Department of Cardiology at Zealand University Hospital. During medical school, he gained experience

with research through assisting at a neurologic research project which investigated miRNAs potential as diagnostic biomarkers.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidelines.

Conflict of interest: none declared.

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