

# Spontaneous coronary artery wall haematoma: success of conservative management despite alarming extension: a case report

Serigne Cheikh Tidiane Ndao <sup>1\*</sup>, Amer Zabalawi <sup>1</sup>, and Martine Gilard<sup>2</sup>

<sup>1</sup>Yves Le Foll General Hospital, 10 rue Marcel Proust, 22000 Saint-Brieuc, France; and <sup>2</sup>Blanche University Hospital, 2, Avenue Foch, 29200 Brest Cavale, France

Received 1 April 2019; first decision 30 April 2019; accepted 4 September 2019; online publish-ahead-of-print 23 September 2019

## Background

Spontaneous coronary artery dissection (SCAD) is a particular mode of presentation of acute coronary syndrome. It preferentially affects the young woman with little or no classical risk factor for atheromatous disease.

## Case summary

In this report, we present a classical non-ST-segment myocardial infarction (NSTEMI) condition in link with a spontaneous coronary artery wall haematoma. A 43-year-old female patient who did not have any risk factors for atheromatous disease presented with NSTEMI. The coronary angiogram (CA) revealed a moderate smooth stenosis of the proximal left anterior descending artery (LAD) that ended just before the take-off of a septal branch. Intracoronary imaging by optical coherence tomography (OCT) visualized a large intramural haematoma reducing the coronary artery lumen. The patient was managed conservatively with antithrombotic regimen, nitrates, and close monitoring with repeated CA. Evolution was favourable despite striking extension of coronary haematoma towards distal LAD. She was then discharged and has been asymptomatic on follow-up visits. Planned repeat CA and OCT at 3 months showed a quite normal coronary artery appearance of the LAD with significant regression of haematoma.

## Discussion

Precise data regarding SCAD epidemiology remains to be determined. The angiographic pattern of our case recalls the Type 2 described by Saw team. But OCT was necessary to confirm the diagnosis. We manage our patient conservatively with close monitoring, as largely suggested by current state of the art, regarding the good haemodynamic status, and absence of ongoing ischaemia despite an evolution severe stenosis.

## Keywords

Coronary wall haematoma young woman • OCT • Conservative treatment • Case report

## Learning points

- Spontaneous coronary artery dissection (SCAD) is a rare form of acute coronary syndrome. It preferentially affects young women with little or no risk factors for atheromatous disease.
- The intracoronary imaging (optical coherence tomography and intravascular ultrasound) remains the reference examination to carry out the diagnosis of SCAD with certainty.
- The reference treatment should be as less interventional and invasive as possible with close monitoring of the patient.

\* Corresponding author. Tel: +33 769 759 307/+33 296 017 639, Email: [serignecheikhtidiane@yahoo.fr](mailto:serignecheikhtidiane@yahoo.fr); [sctndao@gmail.com](mailto:sctndao@gmail.com)

Handling Editor: Gabor G. Toth

Peer-reviewers: Sameh Shaheen, Rami Riziq Yousef Abumualeq, Panagiotis Xaplanteris, and Rafael Vidal-Perez

Compliance Editor: Rahul Muherjee

Supplementary Material Editor: Ross Thompson

© The Author(s) 2019. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact [journals.permissions@oup.com](mailto:journals.permissions@oup.com)

## Introduction

Spontaneous coronary artery dissection (SCAD) is a rare form of acute coronary syndrome (ACS). It includes coronary wall haematomas and SCADs. It preferentially affects young women with little or no risk factors for atheromatous disease.<sup>1,2</sup> It is sometimes associated with fibromuscular dysplasia, Marfan or Ehlers–Danlos syndrome, peripartum, oral contraceptives, or cocaine use but most cases are idiopathic. The diagnosis confirmation often requires the use of intracoronary imaging. Treatment should be guided by the extent of the lesions, the myocardial ischaemia, and the haemodynamic status. It is based in most cases on a conservative strategy except for the ischaemic or hemodynamically unstable patients who need revascularization.

## Timeline

Timeline	Events
Day 1	<p>Constrictive chest pain radiating to both arms for 20 min with an elevated US troponin of 236 (normal &lt; 14 pg/mL).</p> <p>Echo: left ventricular ejection fraction (LVEF) 45%, severe apical and anterior wall hypokinesia</p> <p>Coronary angiogram (CA): moderated smooth stenosis of the proximal left anterior descending artery (LAD).</p> <p>Optical coherence tomography (OCT): large intramural haematoma reducing the coronary artery lumen.</p> <p>Medically managed (aspirin, beta-blocker, and statin) with close monitoring on Intensive Cardiac Care Unit.</p>
5 days later	<p>Chest pain recurrences with good response to nitroglycerine and rise in troponin.</p> <p>CA: significant worsening of the stenosis with an extension of the wall haematoma towards the mid-LAD with normal run flow.</p> <p>Heart-team discussion: maintain conservative management with in-hospital monitoring.</p> <p>She remained asymptomatic with optimized medical treatment.</p>
After 2 weeks	<p>No chest pain recurrences.</p> <p>CA control: extension of the haematoma to the distal LAD.</p> <p>Patient discharged with same treatment and regular follow-up.</p>
After 3 months	<p>Totally asymptomatic.</p> <p>CA: quite normal appearance of the LAD.</p> <p>OCT: significant regression of haematoma with dispersed, vanishing haematoma remnants.</p> <p>Cardiac magnetic resonance imaging: limited apical myocardial late gadolinium enhancement with normal LVEF.</p>
1 year later	<p>No symptoms, normal LVEF with persistent limited apical sequelae</p>

## Case presentation

A 43-year-old female patient with no cardiovascular risk factors, weight 57 kg, height 170 cm, presented to our hospital with constrictive chest pain at rest radiating to both arms for 20 min disappearing spontaneously. Admitted to the coronary care unit for monitoring, clinical examination was normal, blood samples found elevated ultrasensitive troponin at 236 for a normal <14 pg/mL. She was not pregnant. The electrocardiogram recorded sinus rhythm at 63/min with negative T waves at V1 and V2 leads. The echocardiography showed a moderate left ventricle dysfunction (left ventricular ejection fraction 45%) with severe apical and anterior wall hypokinesia.

The patient was referred to our Cath lab after receiving Aspirin 250 mg IV, oral Ticagrelor 180 mg, and Fondaparinux 2.5 mg subcutaneous. Coronary angiography showed a long (22 mm) moderate smooth stenosis (50%) of the proximal left anterior descending artery (LAD) (*Figure 1A and B*). Optical coherence tomography (OCT) analysis highlighted an intramural coronary haematoma starting at the ostium of the artery to its middle part without a dissection plane visualized (*Figure 1C*).

The patient was admitted to our intensive cardiac care unit for monitoring. She has been treated with aspirin 75 mg, bisoprolol 2.5 mg, and atorvastatin 80 mg orally. She was asymptomatic with no signs of haemodynamic instability and normal clinical examination. Five days later, she presented several recurrences of chest pain with good response to nitroglycerine administration associated to an increased level of troponin; average blood pressure was 125/85 mmHg. The angiogram control revealed a significant worsening of the stenosis with an extension of the wall haematoma towards the middle part of the LAD (*Figure 2*) with normal run flow (TIMI 3).

After heart-team discussion, a conservative strategy was decided upon with hospital monitoring and medical treatment with aspirin, beta-blockers, and nitrates. Chest pain totally disappeared without recurrences. Angiogram control at 2 weeks (*Figure 3*) showed an extension of the haematoma to the distal LAD.

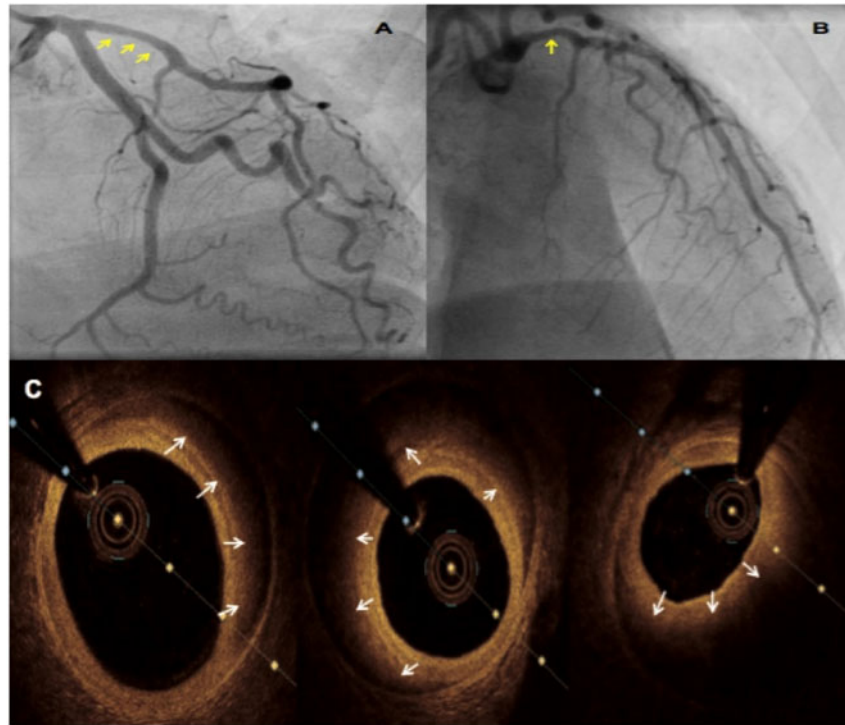
In the following days, the patient remained totally asymptomatic and was then discharged with the same treatment. Strict rest was also suggested.

The patient has been consulting in our centre once a week for 3 months.

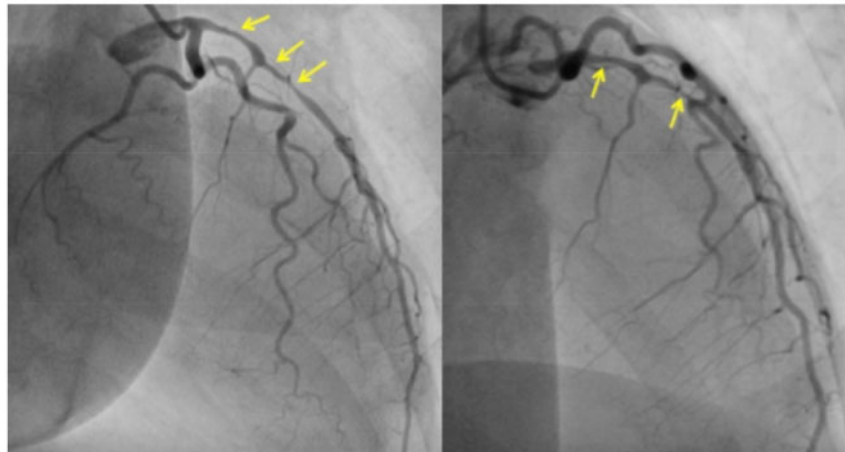
After 3 months, she was totally asymptomatic. Cardiac magnetic resonance imaging showed limited apical myocardial late gadolinium enhancement with normal left ventricle ejection fraction. Coronary angiogram showed a quite normal appearance of the LAD (*Figure 4A–C*). Optical coherence tomography control showed a significant regression of the LAD wall haematoma with dispersed, vanishing haematoma remnants (*Figure 4D*).

At 1 year, she remained asymptomatic and her echocardiography showed persistent limited apical akinesia with normal left ventricular function.

- An angio computed tomography (CT) scan revealed diffuse atheromatous infiltration of both carotid and vertebral arteries.
- An aortic and renal artery CT scan did not find any sign for fibromuscular dysplasia.



**Figure 1** Coronary angiography showing moderate smooth stenosis of the proximal left anterior descending artery that ended just before the take-off of a septal branch, see yellow arrows (A, B); optical coherence tomography (C) visualizing a large intramural haematoma reducing the coronary artery lumen (see white arrows).



**Figure 2** Angiogram control (5 days after onset of symptoms) showing an extension of intracoronary haematoma to the mid-left anterior descending artery. Significant worsening of the stenosis.

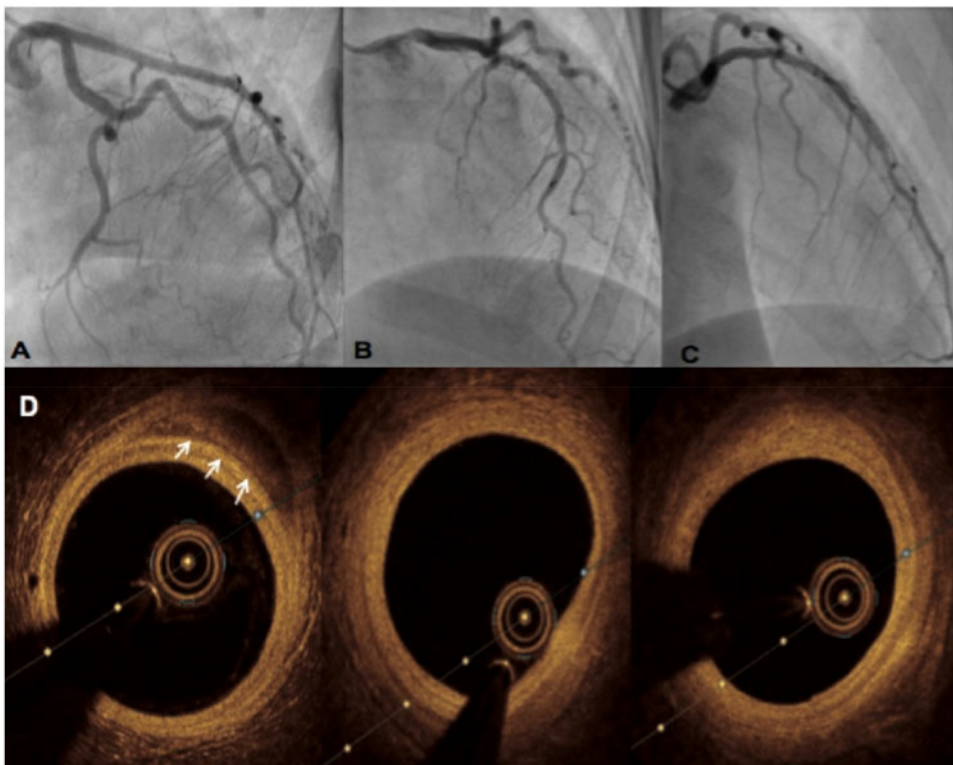
## Discussion

Haematomas and SCADs are an under-diagnosed pathology because they have been unknown for a long time. The exact prevalence remains to be determined, around 1.7–4% of ACS in some

angiographic series.<sup>3,4</sup> Japanese and Canadian series report a prevalence of about 30% of all ACS with an elevation of troponin in women under 60 years.<sup>5,6</sup> Several contributing factors have been reported in the literature with no clear causal links including systemic inflammatory diseases, toxic causes (cocaine and cannabis), and



**Figure 3** Coronary angiography (15 days after onset of symptoms) showing an extension of the haematoma to the distal left anterior descending artery (see yellow arrows).



**Figure 4** Coronary angiogram at 3 months (A–C) no obvious stenosis seen; optical coherence tomography control (D) 3 months later showing regression of the left anterior descending artery wall haematoma with dispersed, vanishing haematoma remnants (lunar crescent—D arrows).

connective tissue diseases. However, several series show a well-established association between SCAD and fibromuscular dysplasia and pregnancy.<sup>7</sup> Our patient did not present any of these precipitating factors.

The different clinical presentations of patients with SCAD reported in the literature are predominantly ST-segment myocardial infarction in 26–55% of cases<sup>2,8,9</sup> and non-ST-segment myocardial infarction in 47.3% of cases.<sup>2</sup> Sudden death and cardiogenic shock are

not exceptional with respective proportions of 2.8–10%<sup>1,9</sup> and 10.9%.<sup>2</sup> Chest pain remains the most frequent symptom described, 95.9% of cases.<sup>10</sup>

Regarding the angiographic aspect, our case recalls the Type 2 described by Saw<sup>11</sup> with the presence of diffuse stenosis of variable severity. A single coronary artery is concerned in our case roughly reflecting the data of some series, with according to Saw *et al.*,<sup>7</sup> 45–61% of the LAD and its branches, 15–45% involvement of the circumflex artery, and the right coronary artery and its branches in 10–39% of cases. The left main artery is involved in 0–4% of cases. Mono-vessel disease remains the most common.

Apart from the classical angiographic aspects described by Saw and recently refined by Motreff *et al.*,<sup>2</sup> the diagnosis of SCAD can be challenging even for an experienced interventional cardiologist. The intracoronary imaging (OCT and intravascular ultrasound) remains the reference examination to carry out the diagnosis of SCAD with certainty and finds especially its interest in the ambiguous forms without intimal rupture as in our case. Potential mechanism causing haematoma without dissection could be the 'outside-in' mechanism where the causal event is the primary disruption of a vasa vasorum micro-vessel leading to haemorrhage directly into the tunica media.<sup>7</sup>

Optical coherence tomography can also guide a possible angioplasty procedure in some cases.

Management is entirely empirical, inspired mainly by sporadic cases reported in the literature, due to the lack of randomized trials, large registries, and guidelines. The practices are generally based on heart-team discussion, taking into account the clinical and haemodynamic tolerance, TIMI flow, and the extension of the coronary involvement.

A recent position paper on the subject suggests conservative management as far as flow is maintained and in the absence of ongoing ischaemia or infarction followed by a period of inpatient monitoring.<sup>7</sup> Our patient was treated with long-term aspirin alone as suggested by most experts, beta-blocker, statin, and nitrates.<sup>12</sup> In order to avoid extension of intramural haematoma we did not administer heparin.<sup>12</sup>

Coronary revascularization is associated with increased risk of complications but should be performed in patients with ongoing symptoms of ischaemia or haemodynamic compromise.<sup>7</sup>

Multicentre registry and prospective study are needed to address definitely the best management strategy of this particular pathology.

## Conclusion

Haematoma and SCAD is a rare and peculiar form of ACS affecting mostly young women.

A diagnostic approach based on multimodal imaging is needed.

Coronary angiography remains the reference examination and should be supplemented by intracoronary imaging, particularly OCT.

In this singular condition, the reference treatment should be as less interventional and invasive as possible with close monitoring of the patient.

## Lead author biography



Serigne Cheikh Tidiane Ndao, MD was born on 1 December 1984 in Kaffrine, Senegal. He received his Interventional Cardiologist Fellowship from Yves Le Foll General Hospital, Saint-Brieuc, France (2016–19). He completed his graduation in Interventional Cardiology 2018 (Paris Descartes University France) and completed

his echocardiography graduation in May 2019 (Bordeaux University France).

## Supplementary material

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

## Acknowledgements

The authors wish to thank Payot Laurent, Delaunay Regis, Moukda Khounlaboud (Yves Le Foll General Hospital).

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

**Consent:** The author/s 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 guidance.

**Conflict of interest:** none declared.

## References

1. Tweet MS, Eleid MF, Best PJM, Lennon RJ, Lerman A, Rihal CS, Holmes DR, Hayes SN, Gulati R. Spontaneous coronary artery dissection: revascularization versus conservative therapy. *Circ Cardiovasc Interv* 2014;**7**:777–786.
2. Motreff P, Malcles G, Combaret N, Barber-Chamoux N, Bouajila S, Pereira B, Amonchot A, Citron B, Lussion J-R, Eschaliere R, Souteyrand G. How and when to suspect spontaneous coronary artery dissection: novel insights from a single-centre series on prevalence and angiographic appearance. *EuroIntervention* 2017; **12**:e2236–e2243.
3. Nishiguchi T, Tanaka A, Ozaki Y, Taruya A, Fukuda S, Taguchi H, Iwaguro T, Ueno S, Okumoto Y, Akasaka T. Prevalence of spontaneous coronary artery dissection in patients with acute coronary syndrome. *Eur Heart J Acute Cardiovasc Care* 2016;**5**:263–270.
4. Rashid HNZ, Wong DTL, Wijesekera H, Gutman SJ, Shanmugam VB, Gulati R, Malaipan Y, Meredith IT, Psaltis PJ. Incidence and characterisation of spontaneous coronary artery dissection as a cause of acute coronary syndrome—a single-centre Australian experience. *Int J Cardiol* 2016;**202**:336–338.
5. Nakashima T, Noguchi T, Haruta S, Yamamoto Y, Oshima S, Nakao K, Taniguchi Y, Yamaguchi J, Tsuchihashi K, Seki A, Kawasaki T, Uchida T, Omura N, Kikuchi M, Kimura K, Ogawa H, Miyazaki S, Yasuda S. Prognostic impact of spontaneous coronary artery dissection in young female patients with acute myocardial infarction: a report from the Angina Pectoris-Myocardial Infarction Multicenter Investigators in Japan. *Int J Cardiol* 2016;**207**:341–348.
6. Saw J, Aymong E, Mancini GB, Sedlak T, Starovoytov A, Ricci D. Nonatherosclerotic coronary artery disease in young women. *Can J Cardiol* 2014; **30**:814–819.
7. Adlam D, Alfonso F, Maas A, Vrints C; Writing Committee. European Society Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection. *Eur Heart J* 2018;**39**: 3353–3368.

8. Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, Gersh BJ, Khambatta S, Best PJM, Rihal CS, Gulati R. Clinical features, management, and prognosis of spontaneous coronary artery dissection. *Circulation* 2012;**126**:579–588.
9. Lettieri C, Zavalloni D, Rossini R, Morici N, Etori F, Leonzi O, Latib A, Ferlini M, Trabattoni D, Colombo P, Galli M, Tarantini G, Napodano M, Piccaluga E, Passamonti E, Sganzerla P, Ielasi A, Coccato M, Martinoni A, Musumeci G, Zanini R, Castiglioni B. Management and long-term prognosis of spontaneous coronary artery dissection. *Am J Cardiol* 2015;**116**:66–73.
10. Saw J, Mancini GBJ, Humphries KH. Contemporary review on spontaneous coronary artery dissection. *J Am Coll Cardiol* 2016;**68**:297–312.
11. Saw J. Coronary angiogram classification of spontaneous coronary artery dissection. *Catheter Cardiovasc Interv* 2014;**84**:1115–1122.
12. Hayes S, Kim E, Saw J, Adlam D, Arslanian-Engoren C, Economy K, Ganesh S, Gulati R, Lindsay M, Mieres J, Naderi S, Shah S, Thaler D, Tweet M, Wood M. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. *Circulation* 2018;**137**:e523–e557.