VIA MEDICA

CASE REPORT

Rahim Kanji[1,2], Danial Dinarvand[2,3], Ying X. Gue[2], Diana A. Gorog[1,2,3]

[1]National Heart and Lung Institute, Imperial College, London, United Kingdom
[2]East and North Hertfordshire NHS Trust
[3]University of Hertfordshire, Hertfordshire, United Kingdom

# Myocardial infarction with non-obstructive coronary arteries in young women presenting with ST-segment elevation myocardial infarction: a case series

**Corresponding author:**

Prof. Diana A Gorog, National Heart & Lung Institute Imperial College, Dovehouse Street, London SW3 6LY, United Kingdom, e-mail: d.gorog@imperial.ac.uk

Medical Research Journal 2020; Volume 5
10.5603/MRJ.a2020.0006
 ISSN 2451–2591

## ABSTRACT

**Introduction.** Myocardial infarction with non-obstructive coronary arteries (MINOCA) is an increasingly recognised entity, with comparable mortality to myocardial infarction with obstructive coronary artery disease (CAD).

**Case presentation.** We present the cases of two young females presenting to hospital with ST-segment elevation myocardial infarction without obstructive CAD. Common to both cases was the acute onset of chest pain with no prior cardiac history, minimal cardiac risk factors, and the use of hormone-based contraception. The first patient had an ostially occluded left anterior descending artery (LAD). Flow was restored with balloon inflation and the administration of tirofiban. However, no underlying obstructive CAD was identified, which was confirmed with repeat angiography and optical coherence tomography. The cause was later attributable to plaque erosion, after learning the results of a normal thrombophilia screening. The second patient had ST-segment resolution on arrival to the catheter lab, and on angiography, she had TIMI II flow down the LAD due to significant thrombus burden. Similarly, balloon inflation and tirofiban were administered to improve flow, and non-obstructive CAD was confirmed with repeat angiography and OCT 48 hours later. As with patient 1, this patient too had normal thrombophilia screening results. Both patients were discharged with dual-antiplatelet therapy and secondary prevention, and were advised against hormone-based contraception.

**Discussion.** Patients with MINOCA tend to be younger, with a higher female-to-male preponderance. Multiple causes have been identified, highlighting the importance of following a diagnostic algorithm. This will enable correct treatment, which may differ from that for patients with obstructive coronary disease, thus improving prognosis.

**Key words:** MINOCA, STEMI, OCT, thrombus

## Learning points

- Myocardial infarction with non-obstructive coronary arteries (MINOCA) is not a benign condition and carries a similar mortality to that seen in patients with obstructive coronary artery disease.
- Patients with MINOCA tend to be younger, with fewer cardiac risk factors, and have a greater female-to-male preponderance. They usually present with non-ST-segment elevation myocardial infarction.
- Causes of MINOCA can be multiple, emphasising the importance of following a diagnostic algorithm to ensure correct diagnosis and treatment, which may differ to that for patients with obstructive coronary artery disease.

**Timeline**

| | Time | Event |
|---|---|---|
| Case 1 | Past medical history | Smoker, obesity, rheumatoid arthritis, sleep disorder, hormonal contraception. |
| | Day of presentation | ST-segment elevation myocardial infarction, with an ostially occluded left anterior descending artery LAD. |
| | Day of presentation | Treatment: Tirofiban and balloon inflation to restore flow<br>Diagnostic: Optical coherence tomography (OCT) and thrombophilia screening |
| | 24 hours | Peak troponin: 1791 ng/l<br>Thrombophilia screening: Negative<br>Echo: Moderately impaired left ventricle, with hypokinesis in LAD territory |
| | 36 hours | Repeat angiography and OCT: No obstructive coronary artery disease. |
| | 72 hours | Discharged |
| | 3 weeks | Represented with chest pain. Troponin negative.<br>Repeat angiography and OCT: Mild plaque disease. |
| Case 2 | Past medical history | Smoker, obesity, family history of ischaemic heart disease, anxiety/depression, gastro-oesophageal reflux disease, hormonal contraception |
| | Day of presentation | ST-segment elevation myocardial infarction, with resolution on arrival to catheter lab.<br>Angiography: TIMI II flow in LAD due to thrombus burden |
| | Day of presentation | Treatment: Tirofiban and balloon inflation<br>Diagnostic: Thrombophilia screening |
| | 24 hours | Peak troponin: 576ng/l<br>Thrombophilia screening: Negative<br>Echo: Mild to moderately impaired left ventricle, with hypokinesis in LAD territory |
| | 48 hours | Repeat angiography and OCT: Mild plaque disease, with reduction in thrombus burden. |
| | 72 hours | Discharge |

## Introduction

Myocardial infarction with non-obstructive coronary arteries (MINOCA) was previously thought to be a benign condition, and such patients were often previously reassured and under-investigated and under-treated. In comparison to patients with myocardial infarction attributable to obstructive coronary artery disease (CAD), causes of MINOCA may be numerous, necessitating targeted investigations in a timely manner. We present the cases of two young women admitted with MINOCA, presenting with ST-segment elevation myocardial infarction (STEMI). We detail the presentation and management, and highlight the need for targeted investigations and treatment tailored to the underlying cause.

### Patient 1:

A 25-year-old woman presented via ambulance with chest pain beginning at rest, which was central, dull, and associated with dyspnoea and diaphoresis. Cardiovascular risk factors included a two pack-year smoking history and obesity. Past medical history included rheumatoid arthritis and sleep disorder, for which she took methotrexate and amitriptyline, respectively. She additionally had a contraceptive implant.

On arrival, examination and baseline observations were normal. The ECG showed ST-segment elevation in leads I and aVL, ST-segment depression in leads V1-V3, and ST-segment depression and T wave inversion in leads III and aVF (Fig. 1). She was given loading-dose dual antiplatelet therapy (DAPT).

Emergency coronary angiography showed that the left mainstem was unobstructed, and the left anterior descending artery (LAD) was occluded at the ostium with thrombus (Fig. 2A) with thrombolysis in myocardial infarction (TIMI) zero flow. A Terumo Runthrough guidewire was advanced into the distal LAD. Sequential 2.0 mm, 2.5 mm, and 3.0 mm semi-compliant balloons were inflated at the LAD ostium to fragment the thrombus and aid flow. Tirofiban was administered at the start of the procedure and continued for 12 hours.

Balloon pre-dilatation restored TIMI III flow, but there remained significant thrombus burden. However, no obstructive lesions (> 50%) were identified in the LAD. Optical coherence tomography (OCT) was performed (Fig. 2E), but the images were significantly degraded

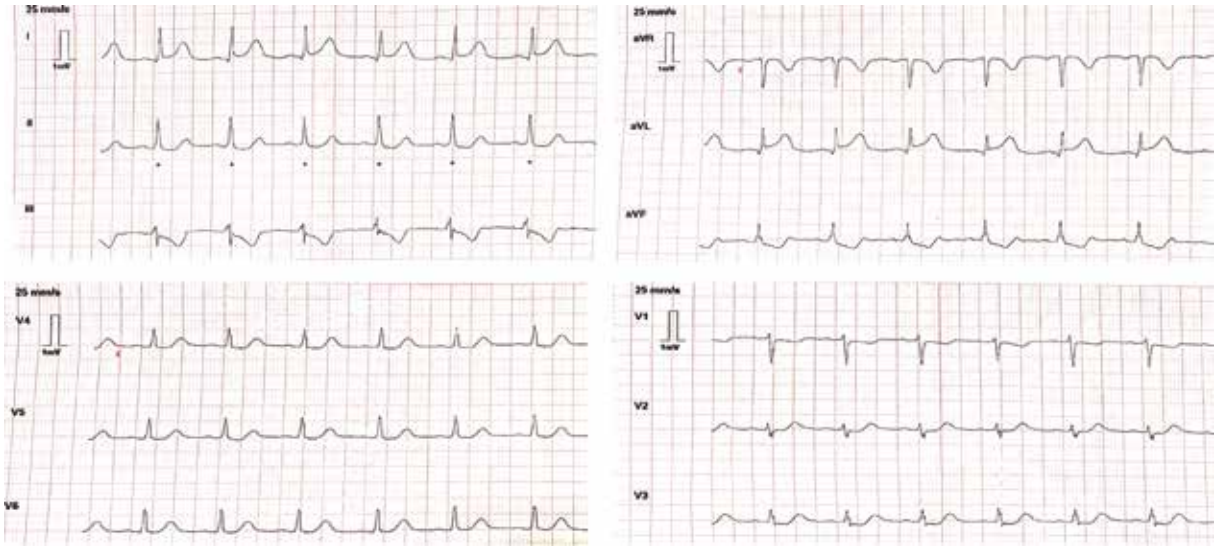

**Figure 1.** 12-lead ECG highlighting ST-segment elevation in leads I and aVL, ST-segment depression in leads V1 – V3, and ST-segment depression and T-wave inversion in leads III and aVF

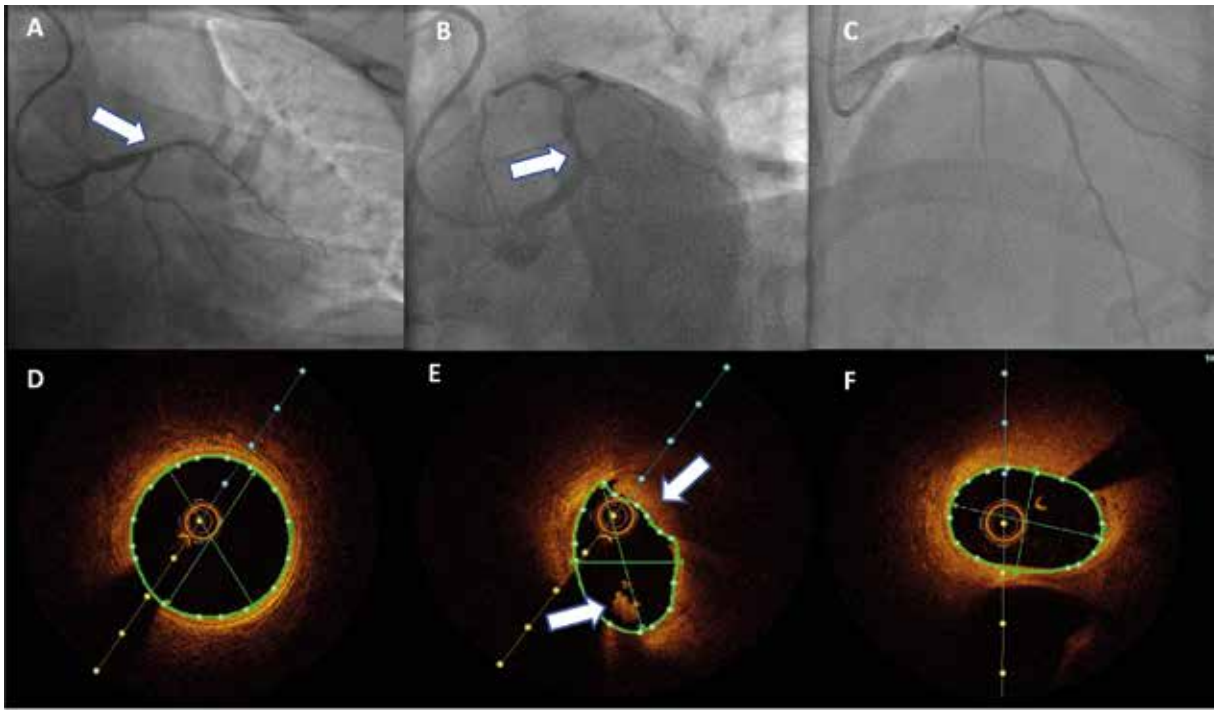


**Figure 2.** Coronary angiography (Panels A–C): A — proximally occluded LAD; B — mild disease in the proximal LAD after tirofiban and low-pressure, semi-compliant balloon dilatation; C — mild atherosclerosis in the proximal and mid LAD. Optical coherence tomography (panels D–E): D — healthy distal vessel; E — thrombus in the proximal LAD 12 hours after tirofiban infusion; F — proximal LAD 3 weeks after the primary event, highlighting mild atheroma, with no evidence of dissection or plaque rupture

by thrombus, a well-known limitation of this imaging modality. Troponin on admission was 19.4 ng/l and peaked at 1791 ng/l. The remaining blood test results were otherwise normal. Transthoracic echocardiography on day 2 after admission showed a moderately impaired left ventricle, with an akinetic anteroseptum, apex, and mid anterior walls. The basal to mid infero-septum was hypokinetic. Thrombophilia screening including lupus anticoagulant, proteins C and S, and antithrombin were normal.

Repeat angiography 36 hours later showed absence of obstructive CAD, with a minimal lumen area in the proximal LAD of 4.9 $mm^2$. On the basis that the presentation was probably attributable to plaque disruption, the patient was discharged home on DAPT with aspirin 75 mg o.d. and ticagrelor 90 mg b.i.d., atorvastatin 80 mg o.d., and advised to stop smoking and arrange removal of her contraceptive implant.

The patient re-presented three weeks later with mild chest pain. Serial troponin I measurements were normal and repeat angiography and OCT were performed. These confirmed mild LAD atheroma without evidence of coronary dissection or plaque rupture (Fig. 2F).

## Patient 2:

A 31-year-old woman presented to the emergency department with chest pain, radiating to her left shoulder, associated with nausea and diaphoresis. The ECG in the ambulance confirmed anterolateral ST-segment elevation and reciprocal inferior ST-segment depression (Fig. 3), and she was given loading-dose DAPT. The pain and ECG changes had normalised by the time she arrived at hospital. Cardiac risk factors included an 8 pack-year smoking history, and a family history of ischaemic heart disease and obesity. Past medical history included anxiety, depression, and gastro-oesophageal reflux disease, for which she took omeprazole, amitriptyline, and the combined oral contraceptive pill. Baseline observations and cardiovascular examination were unremarkable.

Urgent angiography was performed through the right radial artery. The left mainstem, left circumflex, and right coronary arteries were unobstructed. The LAD had TIMI II flow with large thrombus burden in the proximal to mid vessel (Fig. 4A, 4B). Tirofiban was given as an intravenous bolus followed by a 12-hour infusion. Two Terumo Runthrough wires were advanced into the distal LAD and diagonal. A 2.0 mm semi-compliant balloon was inflated to low pressure in both the LAD and diagonal to improve flow. Although this improved flow, significant thrombus burden remained. The patient was pain-free and returned to the ward, and was treated with DAPT, atorvastatin, bisoprolol, and ramipril. Baseline troponin I on admission was not obtained because the blood sample had haemolysed, but the peak troponin I was 576 ng/l. Echocardiography showed mild to moderately impaired left ventricular function, with akinesia of the mid to apical anterior, apical lateral and septal walls, and hypokinesis of the mid anteroseptal and apical inferior segments. A thrombophilia screening including lupus anticoagulant, proteins C and S, and antithrombin were normal.

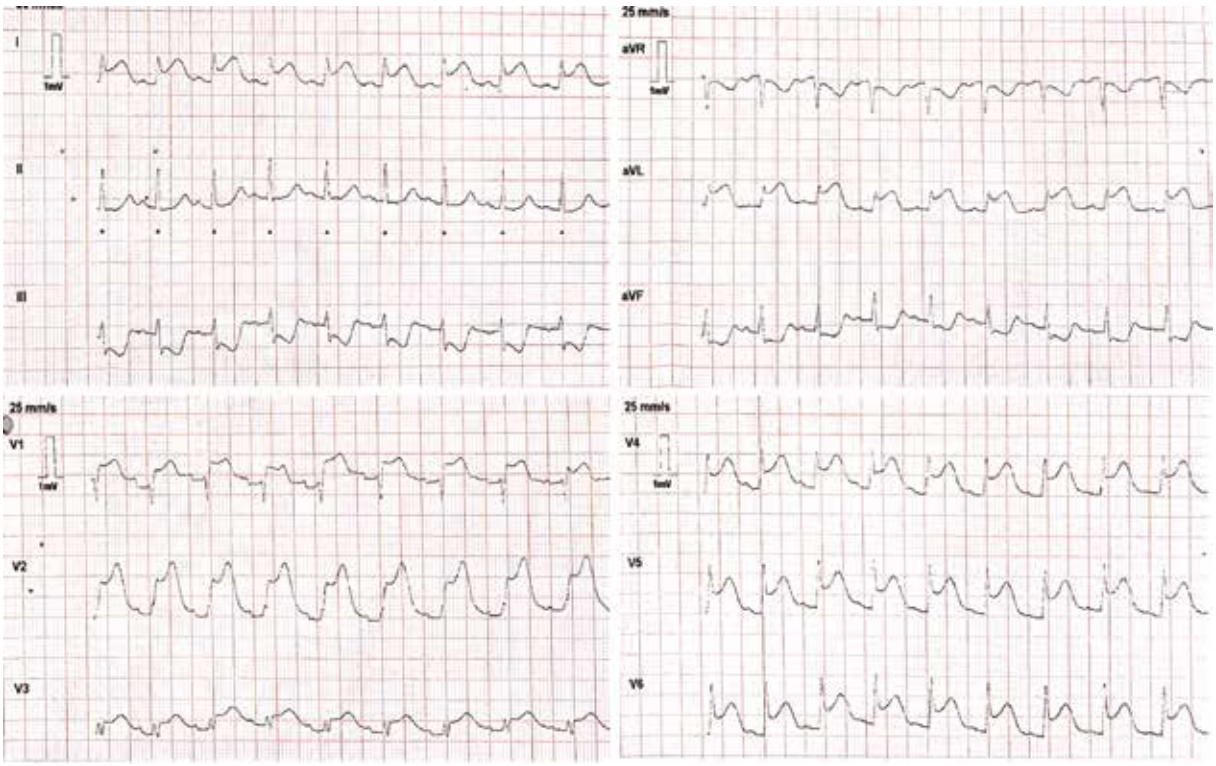


**Figure 3.** 12-lead ECG highlighting anterolateral ST-segment elevation, with reciprocal inferior ST-segment depression

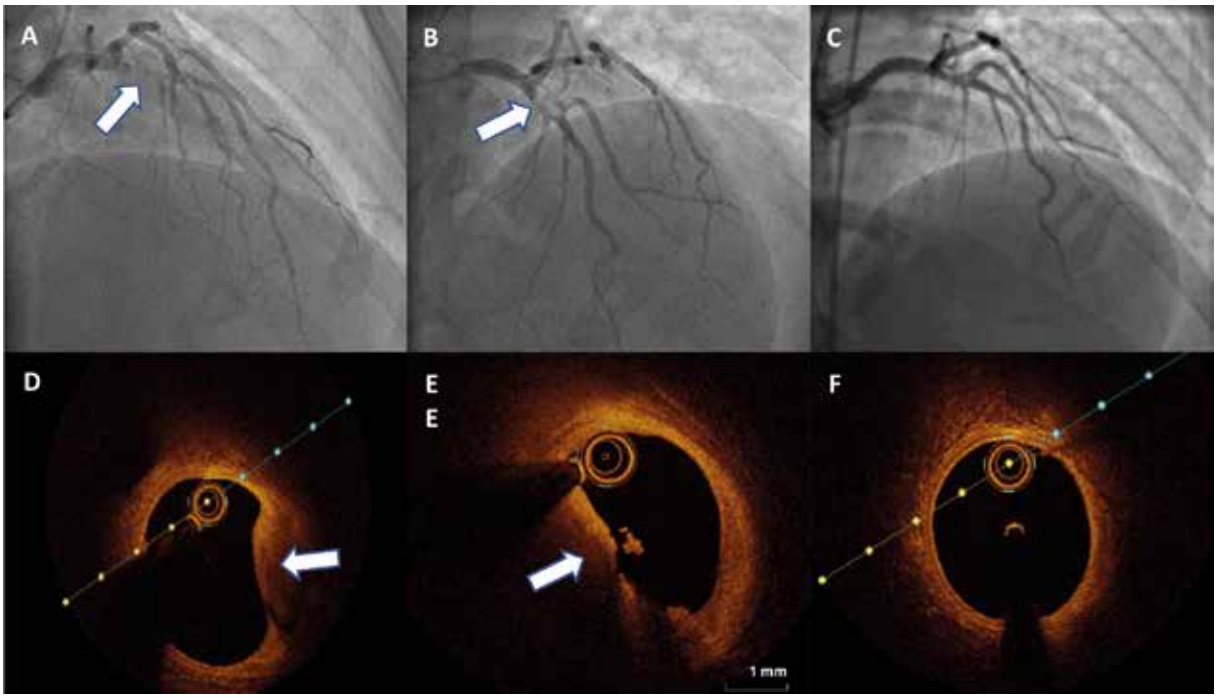


**Figure 4.** Coronary angiography (panels A–C): A, B — significant thrombus burden in the proximal LAD and diagonal; C — significantly reduced thrombus burden in the proximal LAD after tirofiban bolus and low pressure semi-compliant balloon dilatation. Optical coherence tomography (panels D–E): D — mild atheroma in the ostioproximal LAD; E — persistent thrombus in the LAD after tirofiban infusion; F — healthy distal vessel

Repeat angiography and OCT 48 hours after presentation revealed significant reduction in thrombus burden (Figure 4E). There was mild plaque in the proximal LAD with no obstructive CAD. The patient was discharged home on DAPT (aspirin 75 mg o.d. and ticagrelor 90 mg b.i.d), atorvastatin 80 mg o.d., bisoprolol 2.5 mg o.d., and ramipril 2.5 mg o.d. (in addition to drugs on presentation), with advice to abstain from smoking and hormone-based contraception.

## Discussion

We present the case of two young women, admitted with myocardial infarction, manifesting in chest pain, ECG changes of anterior ST-segment elevation, and raised cardiac troponin I. At angiography, coupled with OCT, both patients exhibited significant thrombus burden within the LAD, resulting in complete or partial occlusion of flow, without underlying obstructive CAD. Compared to patients with MI attributable to obstructive CAD, patients with MINOCA tend to be younger, with a higher female-to-male preponderance, and more often present with non-ST-segment elevation (than with ST-segment elevation) myocardial infarction[1]. Coronary causes of MINOCA presentation include plaque disruption (rupture or fissure), profound coronary spasm, and coronary embolisation. The aetiology is important to ascertain because plaque disruption in non-obstructive CAD may be safely managed medically[2], with avoidance of stenting[3], which carries procedure-related risks and subsequent risks of in-stent restenosis.

Spasm is usually apparent at angiography if the patient has ST-elevation during angiography, although provocation testing may be required[4]. Patients with thromboembolic disease are usually identified on thrombophilia screening and require anticoagulation and identification of possible embolic origin.

## Conclusion

We hope these two cases highlight the importance of identifying the aetiology of MINOCA, which is increasingly recognised and now understood to carry significant mortality and morbidity risk. Early recognition and targeted investigations, including the use of the recently proposed MINOCA algorithm[5], will hopefully improve the prognosis in this hitherto under-recognised and under-appreciated condition[1].

## Statement of Consent

The authors confirm that written consent for submission and publication of this case series, including images and associated text, has been obtained from the patients, in line with COPE guidance

## Funding

This article was not funded by any external sources

## Disclosures

The authors declare that there are no conflicts of interest to disclose

## Abbreviations

CAD — Coronary artery disease
DAPT — Dual antiplatelet therapy
LAD — Left anterior descending artery
MINOCA — Myocardial infarction with non-obstructive coronary arteries
OCT — Optical coherence tomography
STEMI — ST-segment elevation myocardial infarction
TIMI — Thrombolysis in myocardial infarction

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