

A Lead to the Culprit

ECG CHALLENGE

A 45-year-old woman presented with persistent chest pain. She had no past medical history and was otherwise fit and well. A 12-lead ECG (Figure 1) recorded by the ambulance team triggered the primary percutaneous intervention pathway. On transit to the primary percutaneous intervention center, the patient suffered a ventricular fibrillation arrest, and sinus rhythm was successfully restored after 3 minutes of resuscitation protocol. What is the likely angiographic finding that would explain the 12-lead ECG pattern?

Please turn the page to read the diagnosis.

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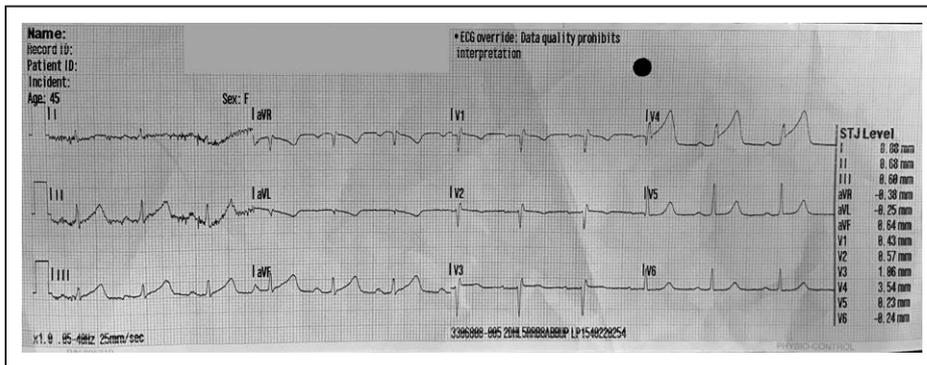


Figure 1. Initial ECG.

12-lead ECG first recorded by the first response ambulance team. J-point ST segment deviation depicted on the right hand side (STJ Level).

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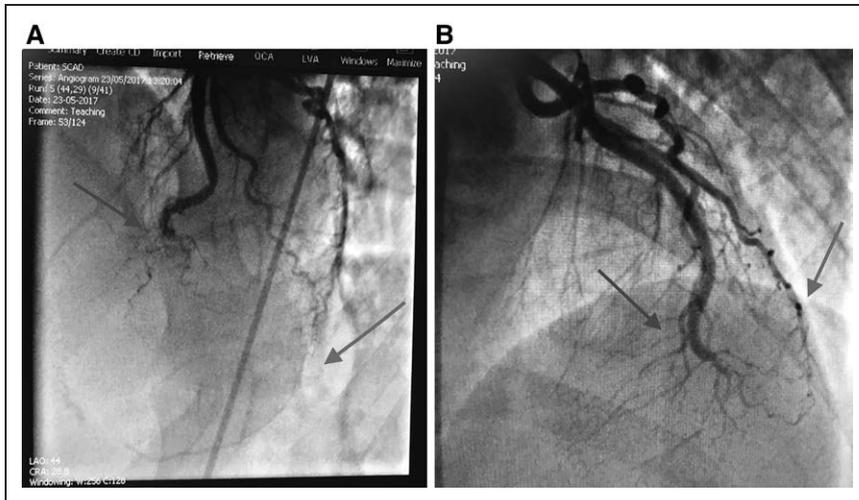


Figure 2. Initial angiogram. Left and right anterior oblique cranial projections. Coronary angiography demonstrates a distal occlusion of the left anterior descending artery (blue arrow) with collateral retrograde filling from the left circumflex artery (red arrow).

RESPONSE TO ECG CHALLENGE

The ECG revealed isolated 3.5-mm ST-segment elevation of lead V4. Among the contiguous leads, V3 had 1.1-mm J-point elevation whereas V5 had 0.2-mm J-point elevation. In addition, ≈0.6-mm ST-segment elevation occurred in the inferior leads II and II and aVF. Many hospitals follow GUSTO (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) thrombolysis guidelines to determine when to trigger the primary percutaneous intervention pathway, namely, the presence of >1-mm ST-segment elevation over 2 contiguous limb leads or >2-mm ST-segment elevation over 2 contiguous precordial leads. The history and localized ECG changes could be consistent with acute ischemia, albeit the culprit vessel could not be reliably discerned on this ECG alone. Although this patient did not meet guideline criteria for primary percutaneous intervention, the cardiac arrest and ongoing symptoms led the patient to have immediate angiography, demonstrated in Figure 2.

Coronary angiography revealed an occlusion in the distal left anterior descending (LAD) artery with retrograde filling by the left circumflex artery. The patient demographics prompted suspicion of spontaneous coronary artery dissection, which was confirmed using high-resolution intravascular ultrasound. The distal LAD true lumen was successfully wired, and intravascular ultrasound confirmed the location within the true vessel lumen (Figure 3). Serial balloon dilatations resulted in restoration of brisk anterograde flow in the LAD, cessation of collateral filling by the left circumflex (Figure 4), and concomitant resolution of symptoms with normalization of the ECG by the end of the case (Figure 5). In retrospective studies, revascularization has been shown

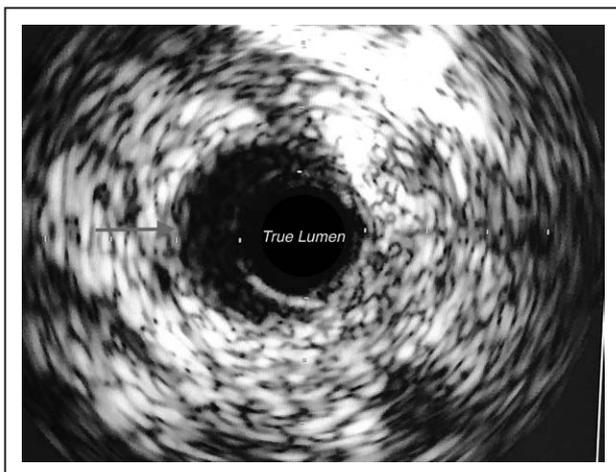


Figure 3. Intravascular ultrasound. Intravascular ultrasound depicting crescent-shaped false lumen (red arrow) surrounding the true lumen in the center of the image (labeled). Septal branches were also noted to be arising from the true lumen.



Figure 4. Angiogram after the procedure Coronary angiography demonstrates restoration of brisk anterograde flow in large wraparound left anterior descending artery with cessation of retrograde filling from the left circumflex.

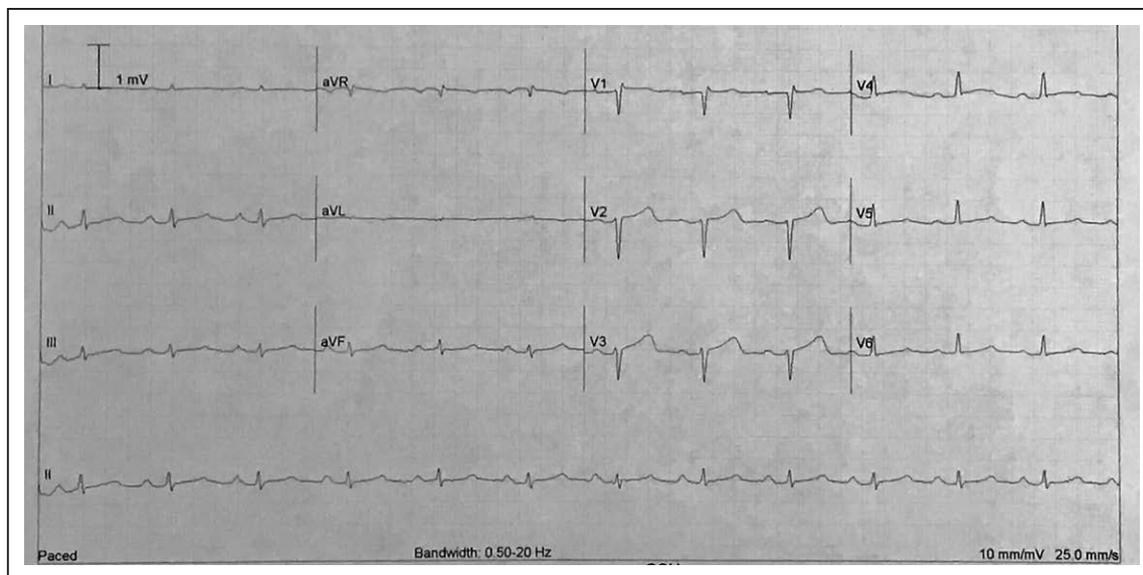


Figure 5. ECG after the procedure.

Normalization of ST-segment changes in ECG after the procedure.

to be associated with a high rate of technical failure in spontaneous coronary artery dissection, whereas a high likelihood exists for complete healing of the dissection with conservative management. This finding has led to a growing consensus in favor of conservative therapy where clinically possible (noted by the presence of brisk anterograde flow and hemodynamic stability) with close observation during the acute period to ensure the dissection does not progress.¹

The angiogram explained the unique initial ECG findings, whereby good collateral blood supply from the left circumflex artery protected the apical left ventricular segments and corresponding ECG leads (V5 and V6) from transmural ischemia. Coronary collaterals are an alternative blood source to myocardium jeopardized by ischemia, and in individuals with coronary artery disease, the presence of collaterals limits infarct size after myocardial infarction. Among individuals without coronary artery disease, preformed collateral arteries prevent myocardial ischemia during a brief vascular occlusion in 20% to 25%.² The right coronary artery was disease free, yet interestingly the borderline ST segment elevation in the inferior leads resolved after balloon angioplasty of the LAD vessel. After balloon angioplasty, it became apparent that the LAD was a large wraparound vessel extending beyond the apex, capable of producing remote zone ischemia manifesting as inferior ST elevation.³

This case highlights how preformed collateral arteries can produce uncharacteristic ECG patterns in the presence of an acutely occluded major artery, likely protecting this patient from sustaining a larger infarct. Nev-

ertheless, by reacting to the malignant clinical course, timely diagnosis and appropriate intervention were achieved, and the patient made an excellent recovery.

DISCLOSURES

None.

AFFILIATIONS

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FOOTNOTES

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REFERENCES

1. Tweet MS, Eleid MF, Best PJ, Lennon RJ, Lerman A, Rihal CS, Holmes DR Jr, Hayes SN, Gulati R. Spontaneous coronary artery dissection: revascularization versus conservative therapy. *Circ Cardiovasc Interv*. 2014;7:777–786. doi: 10.1161/CIRCINTERVENTIONS.114.001659.
2. Wustmann K, Zbinden S, Windecker S, Meier B, Seiler C. Is there functional collateral flow during vascular occlusion in angiographically normal coronary arteries? *Circulation*. 2003;107:2213–2220. doi: 10.1161/01.CIR.0000066321.03474.DA.
3. Brymer JF, Khaja F, Marzilli M, Goldstein S. "Ischemia at a distance" during intermittent coronary artery occlusion: a coronary anatomic explanation. *J Am Coll Cardiol*. 1985;6:41–48.

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