Where Is the Culprit Lesion?

Antoni Bayés de Luna, MD
Miguel Fiol-Sala, MD

ECG CHALLENGE

A 67-year-old man who had experienced a previous heart attack 6 years ago now presents with severe constrictive chest pain for >2 hours with profuse sweating and low blood pressure. An ECG is recorded (Figure 1), and immediately after, the cardiac catheterization laboratory is activated. No prior ECGs are available. What is the diagnosis and what is the precise anatomic lesion?

Please turn the page to read the diagnosis.

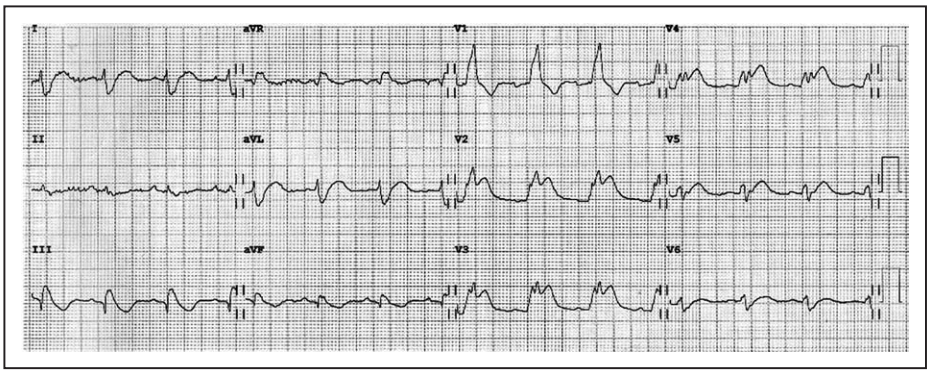

Figure 1. ECG recorded by the prehospital emergency service, which activated the catheterization laboratory.

Correspondence to: Antoni Bayés de Luna, MD, Fundación Investigación Cardiovascular, Institut Català Ciències Cardiovasculars–ICCC, C/S. Antoni Mª Claret, 167, 08025 Barcelona, Spain. E-mail abayes@csic-iccc.org

ECG RESPONSE

The interpretation of the ECG is based on QRS-ST changes combined with the clinical state of the patient. The ECG changes are as follows (Figure 1).

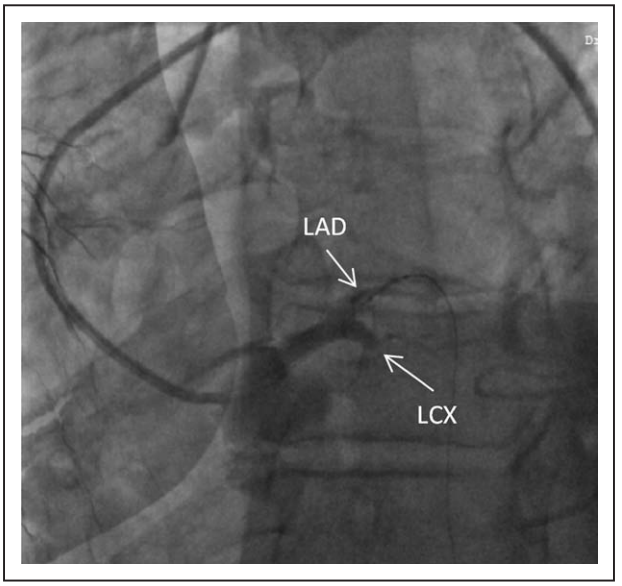

Figure 2. The coronary angiography shows total occlusion of both left anterior descending plus left circumflex (LCX) and left anterior descending coronary arteries (LADs; left main trunk equivalent).

The ECG demonstrates sinus rhythm at 82 bpm with a very long QRS duration of at least 160 ms as a result of right bundle-branch block. We suspect the right bundle-branch block is probably new in the setting of acute ischemic injury, because the perfusion of the right bundle branch depends on the first septal artery, a branch of the left anterior descending coronary artery (LAD). In the horizontal plane, lead V1 demonstrates a wide R wave pattern taller than 10 mm with a slow terminal ascending part without ST-segment elevation and a wide R with important notches in the plateau and significant ST elevation in V2 to V4. Leads V5 to V6 have low voltage, an RS pattern, and a positive T wave. In the frontal plane, the QRS shows low voltage in all leads with a prominent terminal R in lead aVR as a result of the right bundle-branch block and an S1 Q3 pattern.

The most important observation in this case is that interpretation of this ECG clearly suggests[1] ST-elevation acute myocardial infarction as a result of occlusion of the left main trunk (LMT) or its equivalent (proximal LAD plus left circumflex).[1,2] The coronary angiography of this patient shows recent complete occlusion of the LAD and chronic occlusion of LAD plus left circumflex without collateral circulation (Figure 2).

As opposed to a proximal LAD occlusion, in cases of total occlusion of LMT or equivalent,[1,2] there is ST elevation in multiple right/middle precordial leads starting in V2, but not in V1. In V1, the ST segment is isoelectric. In contrast, in cases of ostial occlusion of proximal LAD, V1 also demonstrates ST elevation (the same usually occurs in lead aVR). The reason for this discrepancy that in cases of total occlusion of the LMT, the occlusion encompasses both the LAD and LAD plus left circumflex. Occlusion of the LAD plus left circumflex produces ST-segment depression in right precordial leads that counteracts the

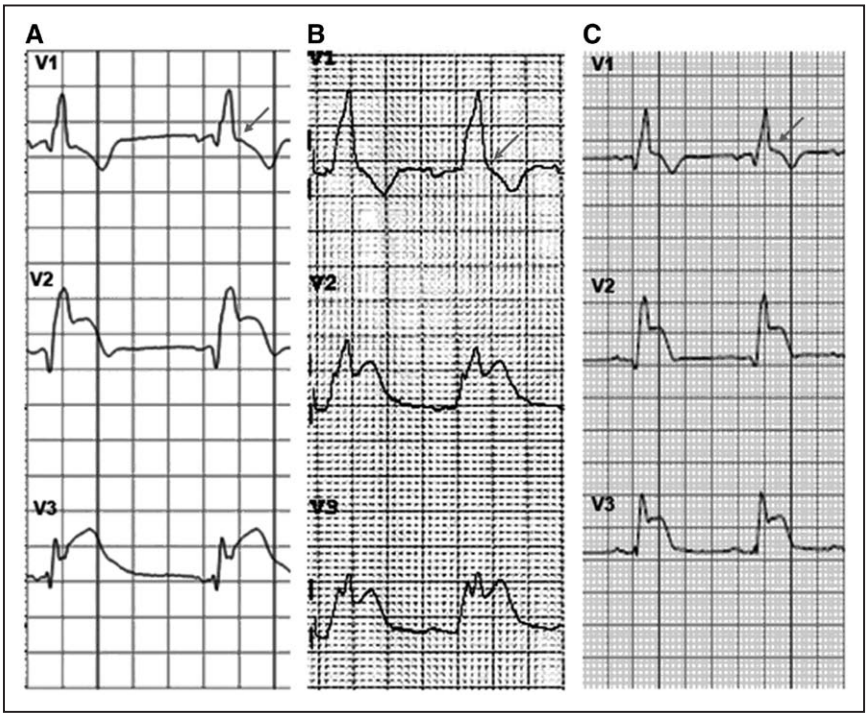

Figure 3. This figure shows the typical pattern of right precordial leads in total occlusion of the left main trunk (LMT) (A), equivalent to LMT (B), and proximal left anterior descending coronary artery (LAD) occlusion (C). See in all cases right bundle-branch block (RBBB) and ST elevation in V2 and V3 with no elevation in V1 in **A** and **B**, whereas ST elevation from V1 to V3 is observed in **C** (proximal LAD).

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ST elevation in V1. Therefore, the presence of ST elevation in V1 supports the diagnosis of isolated LAD proximal occlusion, whereas its absence supports the diagnosis of LMT total occlusion or equivalent (Figure 3).

It is also important to distinguish between the LMT subocclusion pattern and the LMT total occlusion or equivalent pattern. The former[3] corresponds to cases of non—ST-elevation acute coronary syndrome with ≥7 leads with ST depression and ST elevation in lead aVR >V1. The pattern we are describing here is equivalent to a LMT total occlusion and is clearly different from the electrocardiographic point of view (ST-elevation acute myocardial infarction versus non—ST-elevation acute myocardial infarction) and, more importantly from the clinical point of view, because the prognosis of the total LMT occlusion is worse.

Unfortunately, the patient progressed to irreversible cardiogenic shock during the procedure, which was not unexpected, because the prognosis in these cases is grim. In our series[2] of 7 cases of total occlusion of LMT (1 equivalent) without collateral circulation, the cardiac catheterization laboratory was activated in all cases, but only 3 patients survived to emergent percutaneous coronary intervention. The other 4 patients died of cardiogenic shock.

In summary, we present an equivalent of total occlusion of the LMT, which can be recognized from the ECG at presentation because the pattern of ST elevation is different than the pattern observed in the precordial leads of isolated proximal LAD occlusion. Our patient presented with total occlusion of the proximal LAD, having had previous LAD plus left circumflex total occlusion. In this situation, the clinical presentation and ECG are the same as in cases of total occlusion of LMT with ST elevation observed in precordial leads V2 to V4, but not in V1, and frequently accompanied by cardiogenic shock.

DISCLOSURES

None.

AFFILIATIONS

From the Cardiology Autonomous University of Barcelona and ICCC–St. Pau Hospital and Quiron Universitary Hospital, Barcelona, Spain (A.B.d.L.); and Palma Institute of Health Research, Hospital Son Espases, Palma de Mallorca, Spain (M.F.-S.).

FOOTNOTES

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