STEMI RELAPSE IN A PATIENT WITH FACTOR V LEIDEN MUTATION... TIME TO HANDLE THIS!

CASE PRESENTATION

A 51-year-old man presented at the Emergency Department complaining of **CHEST PAIN** started 30 minutes before (NRS 6/10).

His past medical history was consistent with **HETEROZYGOUS FACTOR V LEIDEN** (FVL) symptomatic for pulmonary embolism (2008) on OAC therapy and **ANTERIOR STEMI** (1998) treated with balloon angioplasty without stenting.

Cardiovascular risk factor: **HYPERTENSION**; **SMOKING** habit and **DYSLIPIDAEMIA**.

CASE PRESENTATION

VITAL SIGN: BP 140/100 mmHg; SpO2 100% room air; T 36.3 °C; Wt. 93 kg; Ht 175 cm.

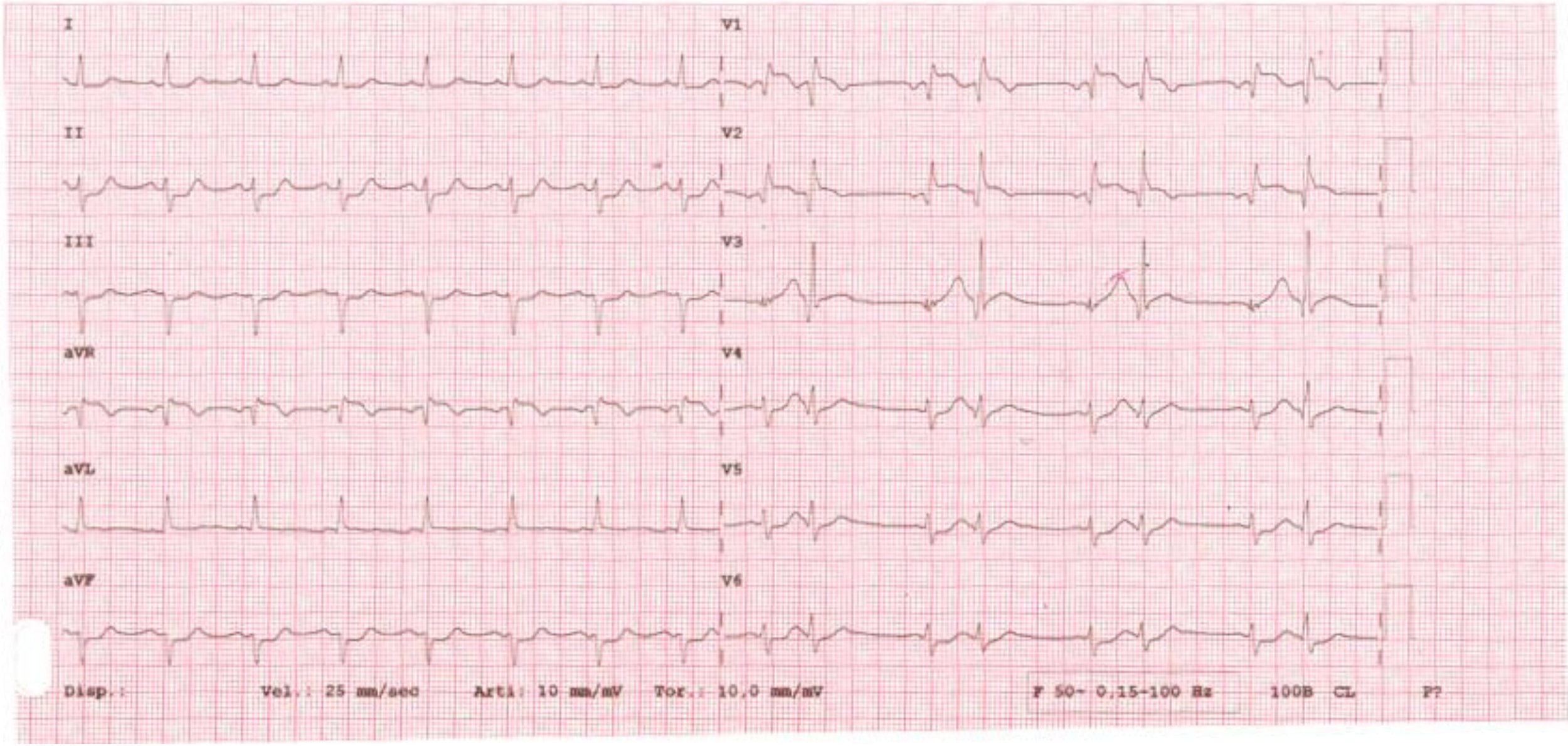
PHYSICAL EXAMINATION: No rales, rhonchi, wheezes, or rubs. Regular cardiac rate and rhythm.

CHEST X-RAY: No active focal parenchymal thickening or pleural effusions. Cardiac image with signs of left ventricular hypertrophy.

ECHOCARDIOGRAM: LVEF 45%; apical and antero-septal akinesia; MR ++; TR +; TAPSE 23 mm

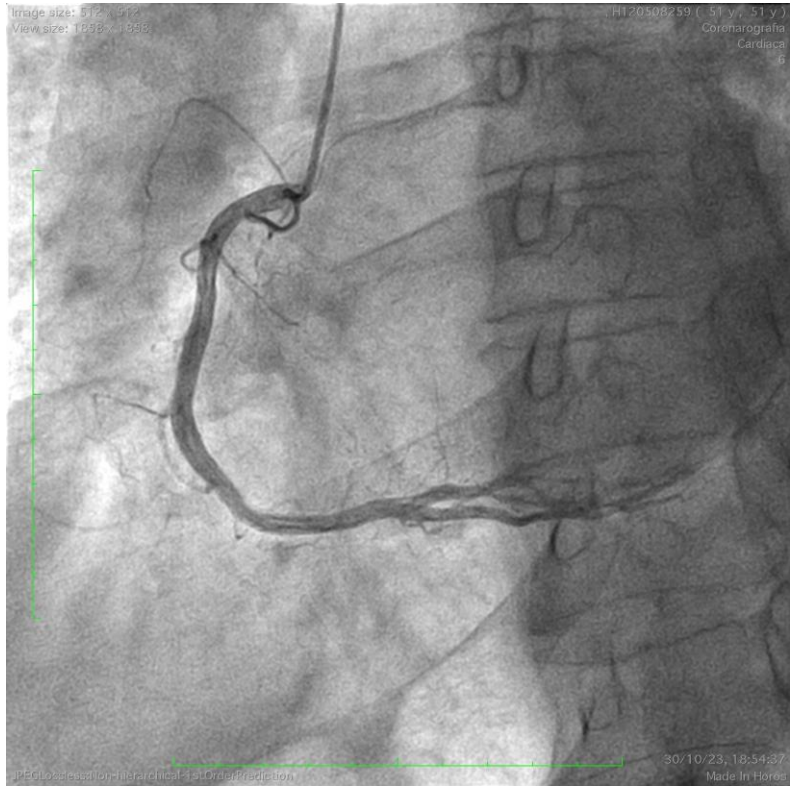
BLOOD TEST: Hb 17.1 g/dL; creatinine 0.83 mg/dL; PLT 305 X 10^9; cTnI 5 ng/L; K^+ 4.00 mmol/L

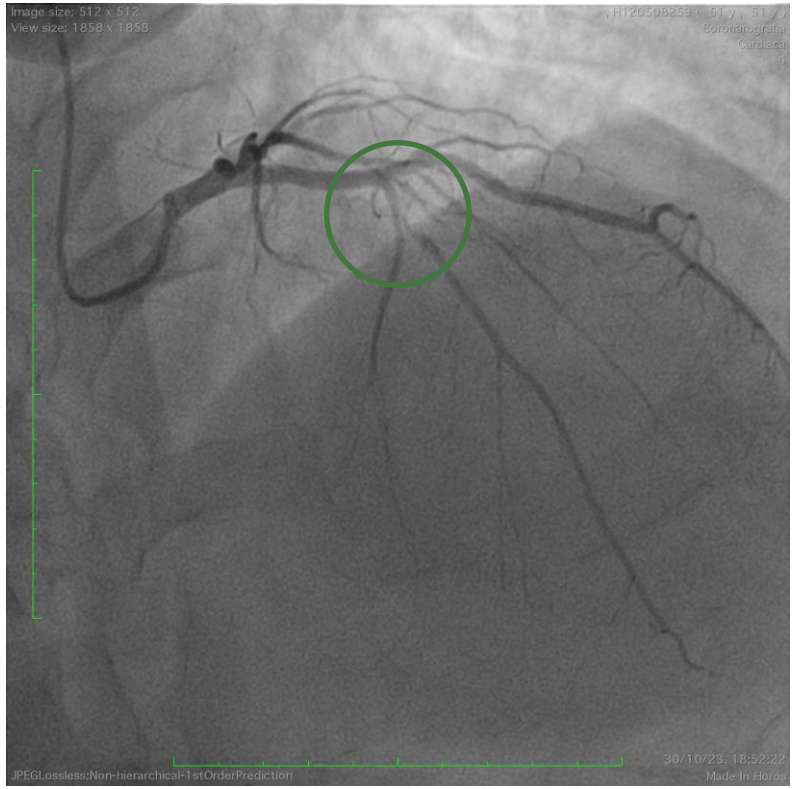
ECG: sinus rhythm 93 bpm; incomplete RBBB; LAFB; ST elevation V1-V4 with reciprocal ST-depression.

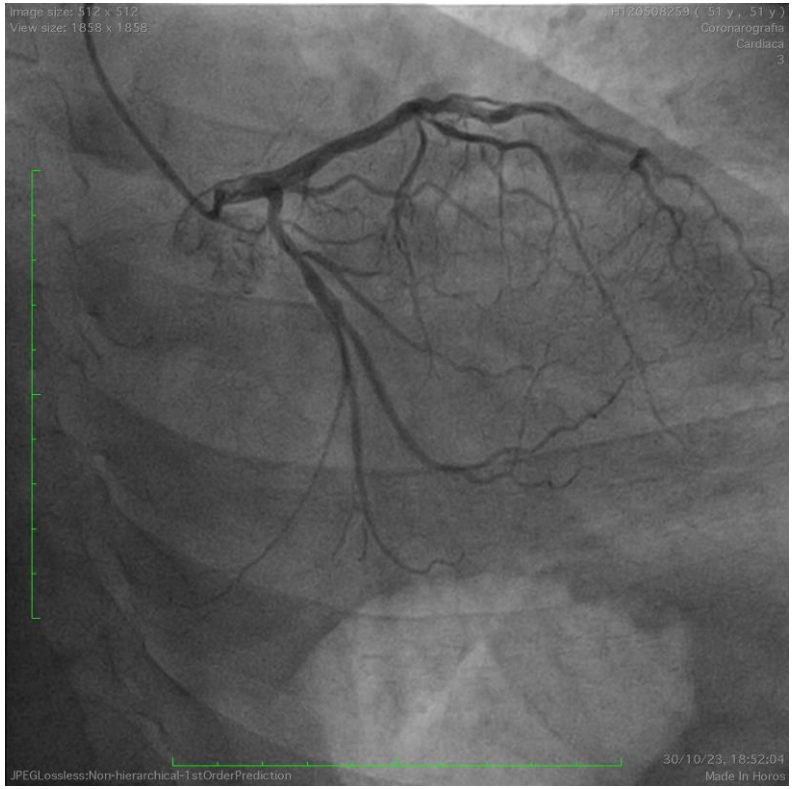


MANAGEMENT

Emergency coronary angiography was performed showing an **60-70%** stenosis of the left anterior descending artery [same location of the previous myocardial infarction] with no flow limitation.



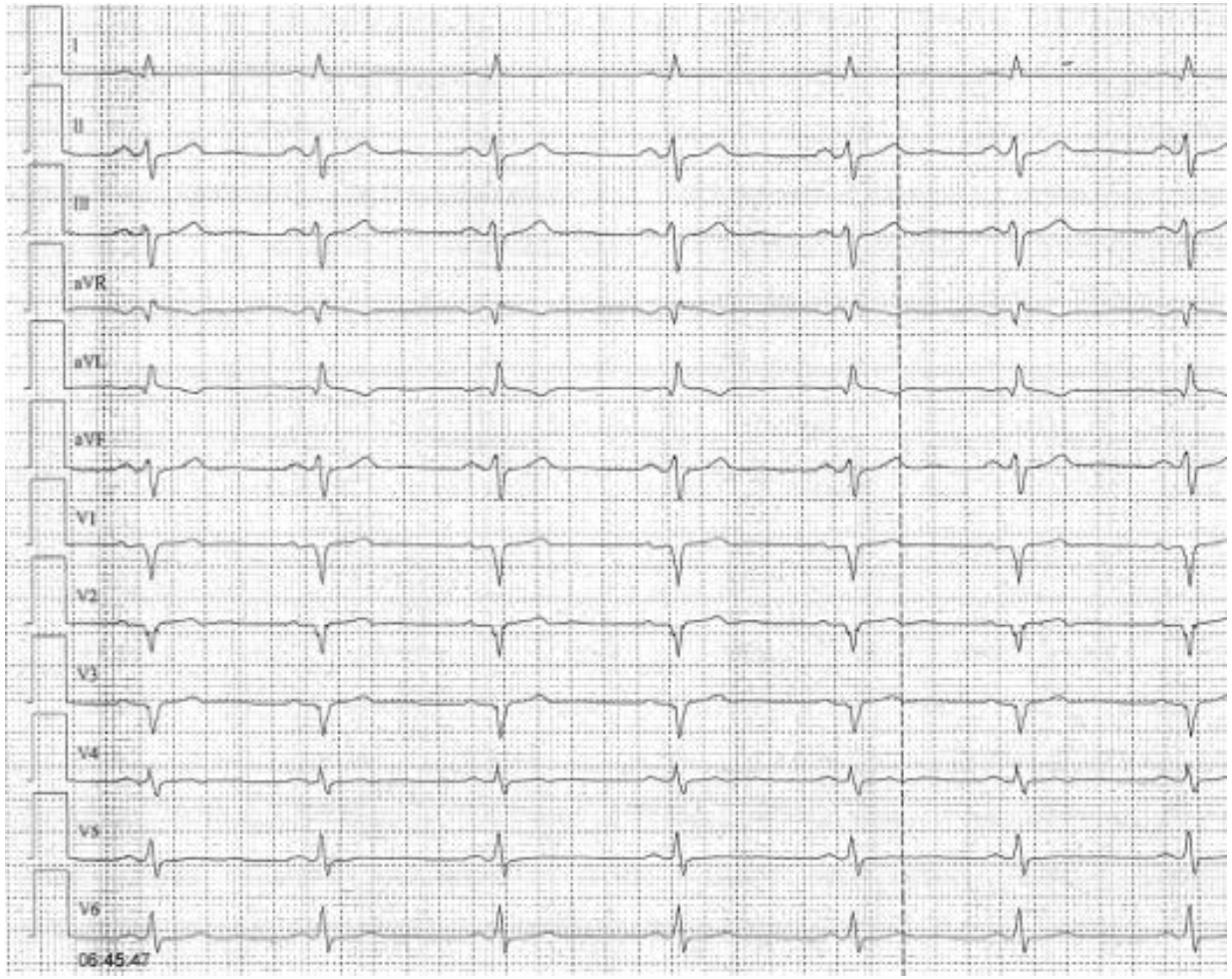




MANAGEMENT

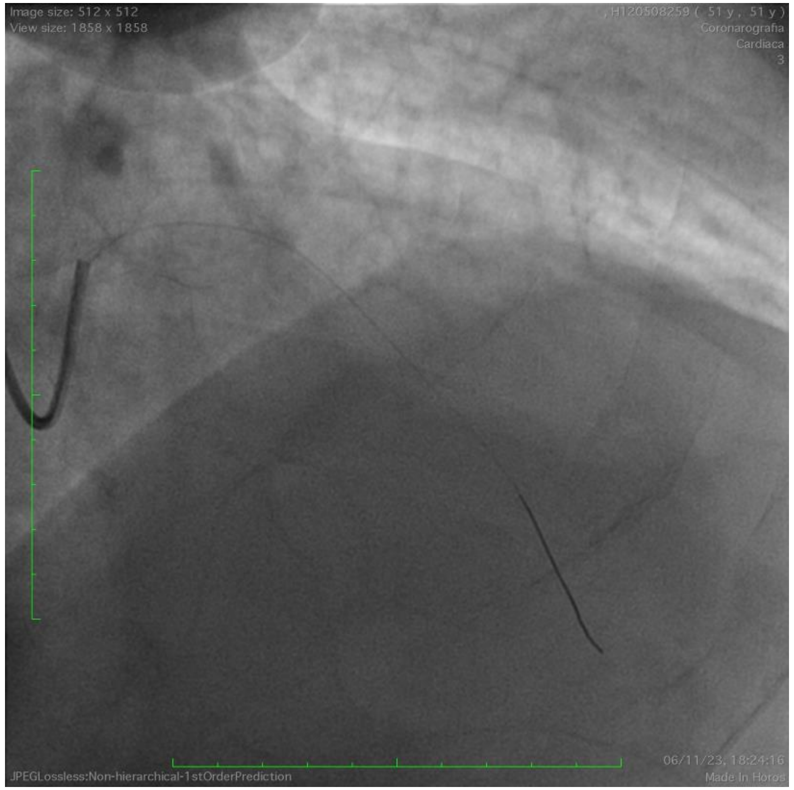
Due to concomitant **SYMPTOM REGRESSION** and **ECG IMPROVEMENT**, revascularization was thus deferred.

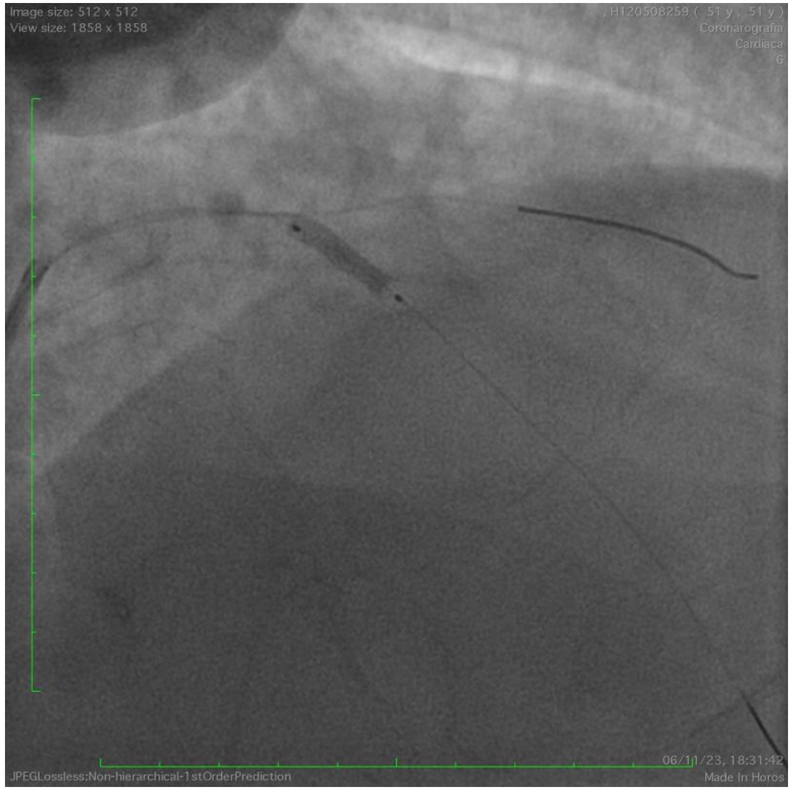
Dual antiplatelet therapy (DAPT) based on **ASA** plus **Ticagrelor** in association with **OAC** was shortly given.

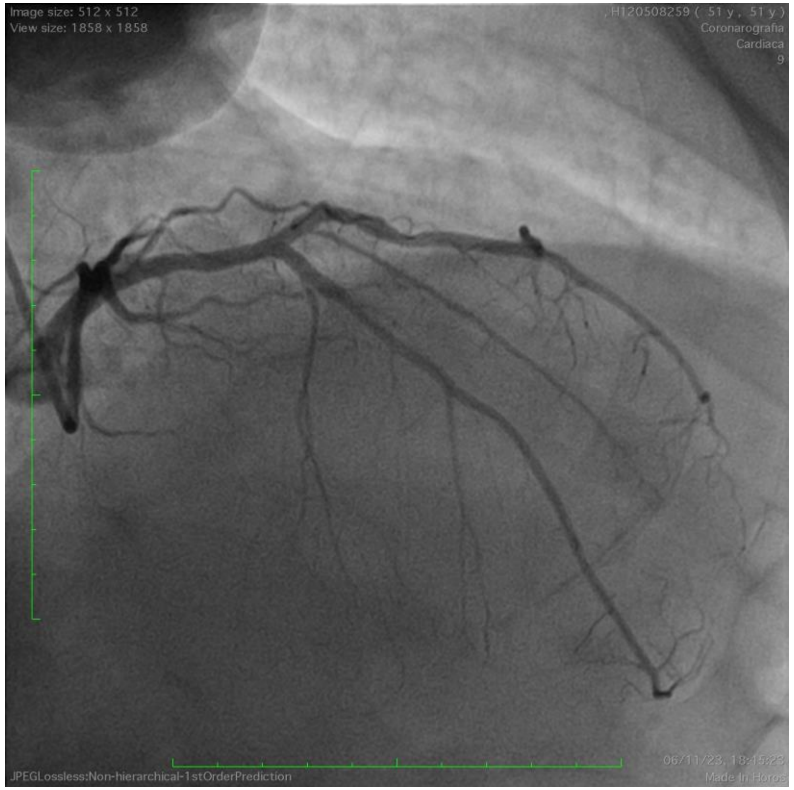


MANAGEMENT

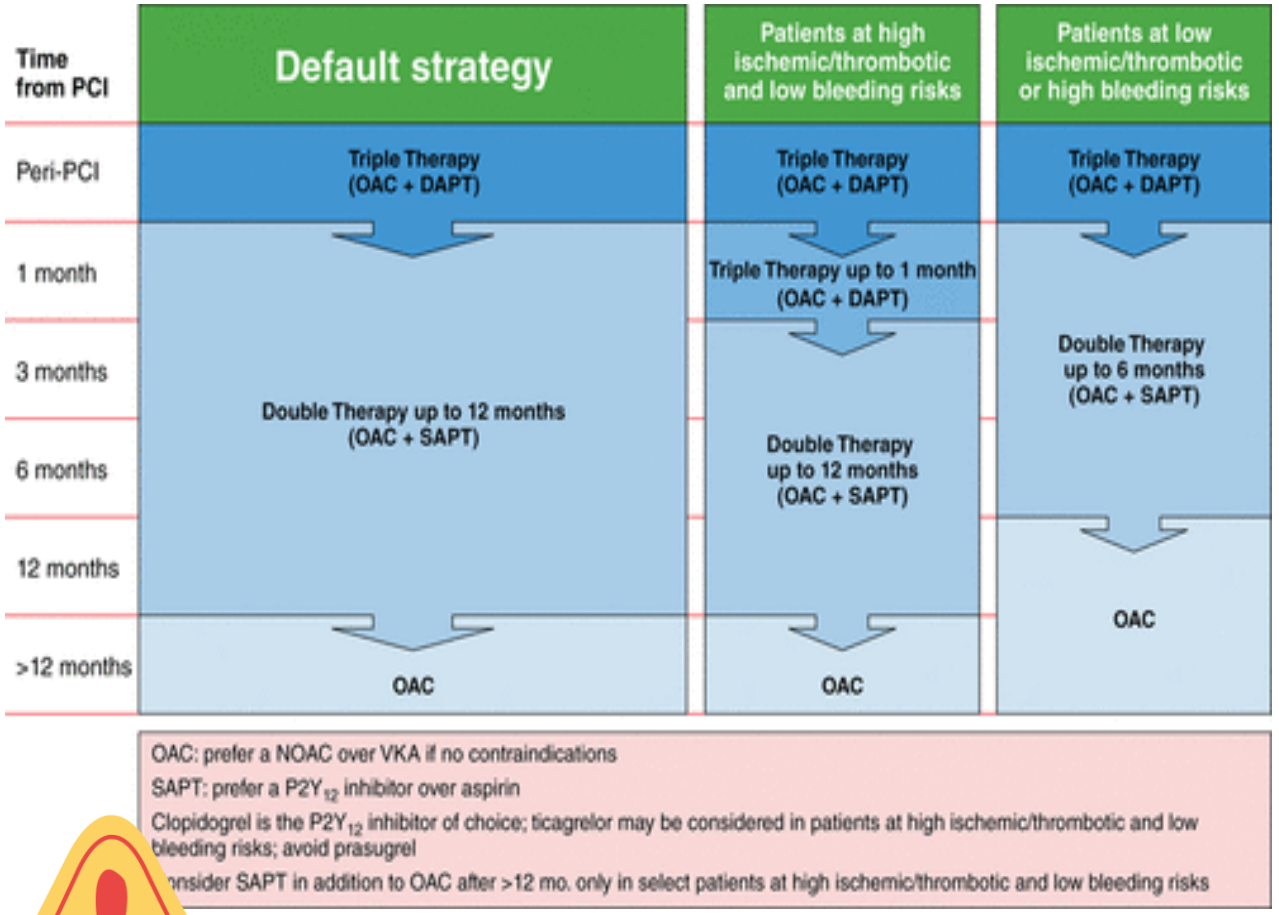
Once the acute phase was over, **intracoronary FFR study** was performed on LAD stenosis showing reduced flow reserve [FFR 0.73]; a second-generation DES was then implanted.







MANAGEMENT



- Antiplatelet therapy was optimized switching **Ticagrelor** to **Clopidogrel** .

- Due to the high thrombotic risk, indication to follow a triple antithrombotic therapy based on **ASA**, **Clopidogrel** and **OAC** for at least 6 months was given.

DISCUSSION

- FVL, is a genetic variant leading to alteration of the inactivation site of factor V, which in turn leads to activated protein C resistance and a **PROTHROMBOTIC STATE.**

- The role of FVL in **ATHEROSCLEROSIS** and **SUBACUTE STENT THROMBOSIS** is controversial, and only few studies have investigated this association[1-3].

- The contribution of thrombosis to atherothrombotic disease may be particularly of major concern in the **YOUNG** where **MULTIPLE RISK FACTORS** are frequently present.

[1] Recurrent Coronary Thrombosis, Factor V Leiden, Primary Antiphospholipid Syndrome, and HIV. JL. Santos, I Cruz, FM Herrero et al. Rev Esp Cardiol 2004;57(10):997-9

[2] Simultaneous subacute coronary drug-eluting stent thrombosis in two different vessels of a patient with factor V Leiden mutation. Eshtehardi P, Eslami M, Moayed DA. J Cardiovasc Med . 2008 Apr;9(4):410

[3] A Peculiar Case of Recurrent Coronary Artery Thrombosis. Nwaobi S, Wood Z, Kalra A, Nguyen S. Cureus. 2022 Sep 20;14(9):e29357.

CLINICAL IMPLICATIONS

- In patients with FVL who undergo coronary percutaneous intervention, indication for stent insertion must be justified not angiographically alone but confirmed with **CORONARY IMAGING** or **FUNCTIONAL INVASIVE TESTING**.

- An **EXTENSIVE ANTITHROMBOTIC REGIMEN** must be considered to ensure stent patency over time.