# Successful management of ostial left main thrombus by systemic thrombolysis 

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A 43-year-old white male, obese (BMI = 43.2), heavy smoker (60 cigarettes/day), arrives at the hospital in a stable non-STsegment elevation myocardial infarction (NSTEMI) setting [chest pain which started 4 h prior to his arrival, electrocardiogram (ECG) showing negative T-waves in inferior leads and flattened T-waves in lateral leads, troponin elevation at $14.70 \mathrm{ng} / \mathrm{mL}$, normal echo]. After he received the standard pharmacological treatment (double antiplatelet treatment, anticoagulant, $\beta$-blocker, nitrates) plus GP $\mathrm{IIb} / \mathrm{Illa}$ inhibitor Tirofiban, the patient became asymptomatic, so we decided to follow an early-invasive strategy.

Coronary angiography showed an ostial left main (LM) thrombus ( $70 \%$ obstruction), normal left anterior descendent artery, circonflex, and right coronary artery (Panel A). Electrocardiogram did not change during and after the intervention and maintained the initial pathological presentation, that of inferolateral ischaemia (Panel B). We switched the GP IIb/llla inhibitor to abciximab.


From this point on we had two possible strategies:

- an interventional strategy (stenting/aspiration with high risk of cerebral and peripheral embolism, which would have exposed the patient to short and long-term risks);
- a pharmacological strategy (continuation of GP IIb/Illa inhibitor or thrombolytic), eventually followed by an interventional strategy.

On the fourth day, thrombus was still in place, as multislice computed tomography also confirmed (Panel C). Biology showed normal cholesterol and negative tests for thrombophylia or platelet dysfunction. We decided to stop abciximab and to perform systemic thrombolysis on the next day, before an eventual angioplasty.

On the following day, the patient underwent the 1 h protocol of alteplase, without complications.
On the sixth day, coronary arteriography showed complete resolution of thrombus with no residual LM stenosis (Panel D).
We present a case of NSTEMI-ostial LM thrombus successfully managed by systemic thrombolysis. We recommend that stable NSTEMI cases involving LM thrombus should initially be managed by systemic thrombolysis or treatment with llb/llla.

We did not perform cardiac magnetic resonance for confirmation of inferior necrosis, which most probably was responsible for the troponin rise and ECG changes, because it was not available at that time. Most probably, the pathophysiological mechanism of necrosis was that of inferior embolization and not due to insufficient blood flow due to massive LM thrombus. However, having the biological, clinical, and ECG criteria for an MI, the patient was dismissed on standard post-MI therapy. He also received specialized help for his smoking dependence.

## Supplementary material

Supplementary material is available at European Heart Journal online.

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