A 56-year-old woman presented with extensive anterior myocardial infarction. One year earlier she underwent full aortic root replacement (biological aortic valve and root, with coronary reimplantation), because of severe aortic stenosis with acent- dend aortic dilatation; three months before admission she had discontinued therapy with oral anticoagulants (OA). Given that primary coronary angioplasty (PTCA) could not be performed immediately she underwent thrombolysis (rtPA-TIMI 14); ventricular fibrillation and severe haemodynamic impairment occurred and required defibrillation, prolonged external cardiac massage, aortic balloon counterpulsation and mechanical ventilation.

Coronarography showed an uncertain image of thrombosis causing not critical left main coronary (LMC) stenosis and abnormal angulation of LMC ostium, not requiring PTCA. CT scan and bronchoscopy showed evidence of pulmonary intraparenchymal hemorrhage, so anticoagulant drugs were discontinued except for low weight heparin.

Transesophageal echocardiography showed extensive anterior akinesis with an ejection fraction of 30%, moderate mitro-tricuspidal regurgitation, yet no evidence of functional impairment of biological prosthetic valve.

Upon awakening, the patient showed right hemiparesis; cranial CT confirmed an extensive ischemic region in the left hemisphere.

Transesophageal echocardiography (TEE) showed the absence of left coronary (LC) cusp systolic excursion, with extensive thrombus extending into the LMC ostium (Fig. 1a), which appeared to be narrowed, partly because of thrombotic apposition, partly because of reduced diameter and slight angulation at insertion (Fig. 1b).
Control TEE after resuming anticoagulation therapy no longer showed thrombus, yet confirmed the LC cuspidal stiffness and the ostial angulation. This is a case of a patient in whom, three months after interrupting OA therapy, intravalvular thrombosis caused massive coronary and cerebral embolization. Long term OA therapy is generally not required in patients with biological prosthesis. We believe that structural impairment to the LC prosthetic cuspidal excursion favoured thrombotic deposition after OA therapy was discontinued and that a role was played by the slight narrowing and angulation of LMC, which was perhaps a consequence of incorrect coronary reimplantation. We were not able to assess the role of a coagulative pathology.

Figure 1  (a) TEE echocardiography. Short-axis plane of closed aortic biological prosthesis showing complete LC cusp obliteration by thrombotic deposition (arrow) and slight narrowing of the LMC ostium. (b) Short-axis plane of open prosthesis. There is clear evidence of the absence of LC cuspidal excursion, which appears to be fixed in closed position, and of thrombus extension to LMC ostium (arrow), which is slightly narrowed and angulated at its insertion.