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Clinical vignette

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Intracoronary thrombus with tissue factor expression heralding acute promyelocytic leukaemia

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A 54-year-old man suddenly experienced severe chest pain at work. Acute inferior myocardial infarction was confirmed and the patient was transferred for primary percutaneous coronary intervention (PCI). No cardiovascular history, risk factors, and no significant comorbidity were reported. At presentation, myocardial necrosis markers were normal, as was routine clinical chemistry. However, blood count showed a pancytopenia with white cells of 640 μL^{-1}, (neutrophiles 160 μL^{-1}), platelets of 112 000μL^{-1}, haemoglobin of 9.1 g/dL, and hematocrit of 25%. Blood smear supported pancytopenia, but otherwise was normal. Coronary angiography showed a subtotal occlusion of the mid-portion of the right coronary artery, where a large, floating thrombus without evidence of a plaque rupture was seen (Panel A; wide arrows). PCI was performed using the PercuSurge Guard-Wire™ system, a balloon-based distal protection device to prevent distal embolization. Large amounts of thrombotic material could be removed. Bone marrow biopsy performed because of persisting pancytopenia revealed acute myeloid leukaemia FAB M3, i.e. acute hypergranular promyelocytic leukaemia (APL). Peripheral blood smear then was showing 28% promyelocytes, containing in 3% Auer rods, and 0.5% were blasts (Panel B; narrow arrows). Histology of the thrombus exhibited a regular pattern with fibrin and platelets, a lot of red blood cells, many neutrophils, and few macrophages. No transformed myeloid cells or blasts were found. However, immunochemistry of the recovered thrombus (Panel C, red colour; arrow heads) detected abundant accumulation of tissue factor, which suggests that this procoagulant plays a crucial role in thrombus formation in APL.