Resolution of intracoronary thrombus with direct thrombin inhibition in a cocaine abuser

A 38 year old black male was admitted with a 10 hour history of central chest pain. He was a cocaine abuser with intake within 24 hours of admission. He was a smoker but had no other identifiable risk factors for coronary disease. ECG showed anterolateral ST elevation with biphasic T waves (upper ECG). Cardiac enzymes were elevated with troponin I concentration of 81 U/ml (reference, < 2 U/ml) and creatine kinase MB concentration of 42 ng/ml (reference, < 16.6 ng/ml). Treatment with aspirin, unfractionated heparin, and nitrates was commenced and coronary angiography was scheduled. Angiography at 48 hours post-admission revealed a large thrombus in the proximal left anterior descending artery but otherwise normal arteries (middle row, left panel).

A diagnosis of cocaine induced coronary thrombosis was made. Atheromatous plaque rupture and thrombosis was considered but deemed less likely as coronary arteries were otherwise normal. Cocaine abuse is a recognised cause of coronary thrombosis, the pathogenic mechanisms being vasoconstriction, platelet activation, endothelial dysfunction, and impaired fibrinolysis.

Percutaneous intervention using distal embolisation protection was considered but dismissed as the risk of device induced embolisation was considered significant. Medical treatment was commenced with tirofiban and low molecular weight heparin. Repeat angiography 48 hours later showed no significant improvement (middle row, middle panel). Heparin and tirofiban were replaced with bivalirudin, a direct thrombin inhibitor (bolus 0.1 mg/kg, followed by 0.25 mg/kg/hour) with the rationale that its unique ability to inhibit both fibrin bound and soluble thrombin, and the absence of effects on platelet activation and aggregation, would enhance endovascular lysis. Repeat angiography 48 hours following bivalirudin showed near total thrombus dissolution (middle row, right panel) with resolution of ECG abnormalities (lower ECG).

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