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Dissolution of a saphenous vein graft thrombus with systemic glycoprotein IIb/IIIa receptor inhibition prior to percutaneous coronary intervention

A 60-year-old man with known coronary artery disease was admitted to the hospital with severe retrosternal pain persisting over two hours. In the week before admission he had suffered from short episodes of chest pain but the symptoms always had resolved spontaneously. The patient had undergone coronary artery bypass grafting of the proximal left anterior descending coronary artery (LAD) and the first diagonal branch 21 years ago, and had suffered from a myocardial infarction ten years ago, but no coronary angiography had been performed at that time. On admission, the ECG showed a non-significant infero-lateral ST segment depression without elevation of the cardiac enzymes. Anti-ischaemic therapy with aspirin, beta-blockers, and a weightadapted abciximab bolus with subsequent continuous infusion was started, and the chest pain resolved. The next day cardiac troponin I was raised to 103 µg/l and creatine kinase to 504 U/l. Due to a sudden recurrence of chest pain the patient was urgently transferred to the cardiac catheterisation laboratory. Coronary angiography revealed severe three-vessel disease without high-grade stenoses of the right coronary and circumflex artery, but a chronic proximal occlusion of the LAD with an open saphenous vein graft (SVG) on the mid part and an occluded SVG on the first diagonal branch with a large thrombus in the distal part (fig. 1A.). Due to the high periprocedural risk of embolisation the intervention was postponed and treatment with abciximab was continued for another 48 hours in combination with half-dose low molecular weight heparin, followed by full-dose low molecular weight heparin for another seven days. The subsequent clinical course was uneventful without recurrence of symptoms. Ten days after admission a repeated coronary angiography revealed a high-grade proximal stenosis of the SVG on the first diagonal branch with a total dissolution of the thrombus (fig. 1B.). Percutaneous transluminal coronary angioplasty of the culprit lesion was performed with insertion of a 3.5/18 mm bare stent (fig. 1C.). Heparin therapy was stopped, and clopidogrel was started for six months. Before discharge, the patient underwent a clinically negative exercise stress test without any significant ECG alterations.

Percutaneous coronary intervention (PCI) is the treatment of choice in acute coronary syndromes. However, PCI in thrombotic SVG occlusions remains an unresolved issue because of technical difficulties, distal embolisation, high restenosis rate, and a poor outcome with low event-free survival rates [1]. The use of distal embolisation protection devices reduces major adverse events after stenting of stenotic SVG [2], and intracoronary application of glycoprotein (GP) IIb/IIIa blockers during angioplasty reduces thrombus burden [3]. GP IIb/IIIa receptor inhibitors prevent platelet aggregation, have a great potential to diminish formation of platelet thrombi in case of platelet activation, and are indicated in patients both undergoing PCI and with acute coronary syndromes not responding to conventional medical therapy when PCI is planned. Our case shows a successful dissolution of a SVG thrombus with GP IIb/IIIa receptor inhibition over 48 hours enabling a subsequent safe and complication-free PCI with a good angiographic result. Due to the high periprocedural risk of PCI in case of a SVG obstruction with concomitant intracoronary thrombus formation a conservative management with attempted dissolution with GP IIb/IIIa receptor inhibitors might be recommended as a first-line therapy as long as the patients are clinically stable.

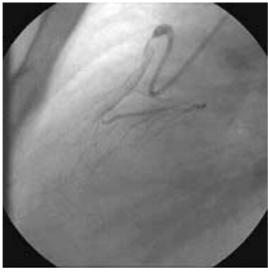
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Figure 1Coronary angiography (lateral view).

- A. Large intracoronary thrombus distal to a high-grade occlusion in the SVG on the first diagonal branch.
- B. Dissolution of the thrombus after treatment with abciximab and lowmolecular heparin.
- C. Good angiographic result after PCI with stenting of the culprit lesion.







References

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