Ventricular septal rupture following abciximab infusion

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Ventricular septal rupture is a rare complication of myocardial infarction. Despite a significant reduction in its incidence with reperfusion therapy, thrombolysis has been implicated in the pathogenesis of septal rupture. There is little information regarding the impact of glycoprotein IIb–IIIa receptor blockers on ventricular septal rupture. We report a case of rupture of the ventricular septum occurring after treatment with the glycoprotein IIb-IIIa receptor blocker abciximab, in the absence of thrombolysis.

KEYWORDS
Ventricular septal defect; Abciximab; Myocardial infarction; Surgical repair

Case report

A 55-year old man presented with a 4-h history of chest pain. He had no history of ischaemic heart disease, was hypertensive and smoked cigarettes. Four years previously, he had suffered a spontaneous intracerebral haemorrhage with a full recovery. The ECG was consistent with an infero-posterior myocardial infarction (MI). Transthoracic echocardiography revealed infero-posterior hypokinesia. He was treated with clopidogrel 300 mg, aspirin 300 mg and intravenous heparin 5000 IU. In view of his prior cerebral haemorrhage, the patient was transferred for coronary angiography. Prior to angiography the patient received a weight adjusted bolus of abciximab (17 mg). Coronary angiography revealed thrombus in the mid-portion of the dominant right coronary artery (Figure 1) and a normal left coronary circulation (Figure 2). A single drug-eluting stent was deployed successfully. Heparin 6000 IU was administered intravenously and an abciximab infusion commenced (0.51 mg per hour for 12 h). At the end of the procedure he developed complete heart block and profound hypotension, requiring temporary pacing and an intra-aortic balloon pump. Following this, the patient had multiple episodes of haemodynamically poorly tolerated VT. The patient remained hypotensive, oliguric and in pulmonary oedema despite inotropic and vasodilator therapy. Transthoracic echocardiography revealed a non-dilated left ventricle with infero-posterior akinesia, a dilated right heart with impaired ventricular function. There was a large ventricular septal defect (VSD) in the mid-inferior septum (Figure 3) with severe left to right shunting (Figure 4). In view of the patient’s condition, he was referred for immediate cardiac surgery. His pre-operative risk using the logistic EuroSCORE was 87%. Surgery was performed under cardio-pulmonary bypass, involving right ventriculotomy through the infarcted tissue and closure of the large, necrotic defect with a bovine pericardial patch. After a 2-week admission to intensive care, he made a good recovery. In view of the extensive myocardial injury and VT, the patient received an implantable cardioverter-defibrillator prior to discharge. At 3 month follow-up he remained well and free of angina.

Discussion

A VSD is a recognised but infrequent complication of ST-elevation MI in current cardiological practice, with a reported incidence of 1–3% without reperfusion therapy, typically occurring in inferior or more frequently, anteroseptal MIs. Recognised risk factors include advanced age, female sex, hypertension, the absence of preceding angina or MI and angiographic total occlusion of the infarct-related artery. Initial therapy includes haemodynamic support with inotropes, vasodilators and an intra-aortic balloon pump. The mortality rate is nearly 100% with medical management and 50% with surgery. Surgery is ideally delayed 3-6 weeks post MI to allow healing of potentially friable tissue. With the advent of reperfusion therapy, the overall incidence of VSD has declined from 1 to 3% in the pre-thrombolytic era to 0.2%. A significant reduction in septal and free wall rupture has been observed both with thrombolysis and percutaneous coronary intervention. Underlying mechanisms include the restoration of vessel patency leading to myocardial salvage and prevention of infarct expansion. However,
the overall reduction in septal rupture with thrombolysis may be partially offset by an increased incidence of early rupture. It has also been suggested that late thrombolysis (12 h after symptom onset) may increase the risk of VSD. Although the impact of reperfusion therapy on subsequent septal rupture has been assessed, there is little information regarding the use of glycoprotein IIb–IIIa receptor blockers. The use of these agents in non-ST elevation MI is well attested and a recent meta-analysis of 11 randomised trials involving 27,115 patients with ST-elevation MI demonstrated a significant mortality benefit of adjunctive abciximab in patients treated with primary angioplasty but not in those receiving thrombolysis. Abciximab was also associated with an increased risk of major bleeding complications with thrombolysis but not with angioplasty. However, there were no specific data on its impact on septal rupture. To date, there are three case reports of mechanical complications in the literature (septal rupture, pulmonary artery rupture and free wall rupture) occurring in patients treated with glycoprotein IIb–IIia receptor blockers. However, in all three cases the outcome was confounded by the concomitant use of thrombolytic agents. This is the first case report of septal rupture occurring with a glycoprotein IIb–IIia receptor blocker in the absence of myocardial haemorrhage. In this case, there was a temporal correlation between the start of the infusion and the onset of haemodynamic instability, which may itself have coincided with septal rupture.

Figure 1 Right coronary angiography demonstrating occlusion of right coronary artery.

Figure 2 Left coronary angiography showing no significant stenoses.

Figure 3 Transthoracic sub-costal 4-chamber echocardiographic findings showing a large ventricular septal defect (VSD). LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Figure 4 Colour flow Doppler in sub-costal 4-chamber view demonstrating left to right systolic flow across the ventricular septal defect.
References