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Case Report

Use of Abciximab for Mediation of Thromboembolic Complications of Endovascular Therapy

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Summary: We describe four cases in which abciximab was used as a thrombolytic rescue agent in the setting of thrombotic events complicating interventional neuroradiologic procedures. After IV administration of abciximab, the thrombus resolved within 30 min in three cases, whereas no thrombolysis occurred in the fourth case despite the addition of intraarterial tissue plasminogen activator. Further evaluation of glycoprotein IIb-IIIa inhibitors in patients with cerebral thromboembolic events is necessary to prove clinical efficacy.

The glycoprotein IIb-IIIa receptor binds to fibrin, which is critical to platelet cross-linking and aggregation. The United States Food and Drug Administration (FDA) has approved glycoprotein IIb-IIIa antagonists for use in the treatment of acute coronary syndromes and in adjunctive therapy to prevent cardiac ischemic complications in patients undergoing percutaneous coronary interventions. We describe four cases in which the glycoprotein IIb-IIIa antagonist abciximab (ReoPro; Centocor, Malvern, PA) was used as a thrombolytic rescue agent in the setting of acute thromboembolic events complicating interventional neuroradiologic procedures.

Case Reports

Case 1

A 67-year-old woman presented for Guglielmi detachable coil (GDC) embolization of an unruptured basilar tip aneurysm. A bolus of 5000 U of IV heparin was administered after placement of a vascular access sheath, after which the activated clotting time (ACT) was 238 s. After placement of the third coil into the aneurysm, thrombus was noted to be present on the coil loops at the aneurysmal neck (Fig 1A). An additional 3000 U of IV heparin was administered immediately, which increased the ACT to 318 s. After 30 min of observation, the appearance of the thrombus remained unchanged. In an attempt to dissolve the intraluminal thrombus, an IV bolus of 0.25 mg/kg of abciximab was administered; this bolus was followed by a maintenance infusion of 10 μ g/min. Angiograms obtained

20 min after the abciximab bolus showed partial resolution of the thrombus. Subsequent angiograms obtained 30 min after abciximab bolus administration revealed complete resolution of the thrombus (Fig 1B). No evidence of distal embolism was depicted on the intracranial angiograms. The abciximab infusion was continued for 12 h. The patient had no neurologic deficits after the procedure.

Case 2

A 43-year-old woman presented with a ruptured basilar artery aneurysm that was treated surgically. Later, symptomatic vasospasm of the left middle cerebral artery developed, and this was treated with balloon angioplasty. She underwent anticoagulant therapy with a 5000-U bolus of heparin immediately after placement of the guiding catheter. After removal of the balloon catheter, angiography revealed embolization to a parietal branch of the left middle cerebral artery (Fig 2A). The ACT was 226 s. An additional 3000 U of heparin was IV administered, after which the ACT was 308 s. During 30 min of subsequent observation, the thromboembolism did not resolve. In an attempt to dissolve the intraluminal thrombus, an IV bolus of 0.25 mg/kg abciximab was administered; this bolus was followed by a maintenance infusion of 10 μ g/min. Angiograms obtained 20 min after the abciximab bolus showed partial recanalization of the thrombus. Subsequent angiograms obtained 30 min after abciximab bolus administration showed complete recanalization of the branches (Fig 2B). The patient had no new neurologic deficits after the procedure. CT scans of the head obtained 72 h after the procedure depicted no evidence of infarction.

Case 3

An 81-year-old woman presented with a ruptured left posterior communicating artery aneurysm. Because of her poor medical condition, the aneurysm was treated with GDCs. Immediately after placement of a 6F guiding catheter in the left internal carotid artery, emboli were noted in the left anterior cerebral and middle cerebral arteries (Fig 3A). A bolus of 5000 U of IV heparin was immediately administered, and this increased the ACT to 294 s. It was decided to complete therapy of the aneurysm before attempting more aggressive therapy of the emboli. After treatment of the aneurysm, no change in the angiographic appearance of the thromboemboli was observed. In an attempt to dissolve the thromboemboli, an IV bolus of 0.25 mg/kg abciximab was administered; this was followed by a maintenance infusion of 10 μ g/min. This bolus was administered 45 min after initial identification of the thromboemboli. Angiography was performed every 10 min for 50 min, during which no change in the angiographic appearance of the thromboemboli was observed (Fig 3B). It was decided to proceed with intraarterial thrombolysis of the middle cerebral artery embolism by using tissue plasminogen activator (t-PA) (Acti-vase; Genentech, South San Francisco, CA). A total of 20 mg of t-PA was administered through a microcatheter into the left middle cerebral artery embolism. Mechanical disruption of the thrombus was also attempted by using the microguidewire, the microcatheter, and an endovascular snare, which restored a

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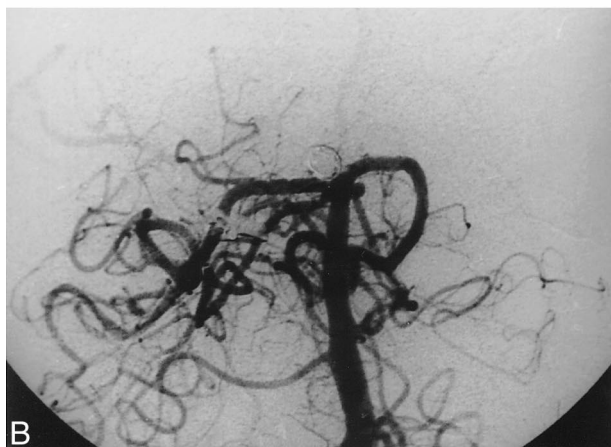


FIG 1. Left vertebral arterial angiograms obtained in a 67-year-old woman who presented for Guglielmi detachable coil (GDC) embolization of an unruptured basilar tip aneurysm.

A, Image shows the thrombus on the coil loops at the aneurysm neck (arrow).

B, Image obtained 30 min after bolus administration of abciximab shows complete resolution of the thrombus.

small amount of flow through the middle cerebral artery. CT of the head performed immediately after the procedure showed no new intracranial hemorrhage. Brain death developed over the next 18 h.

Case 4

A 65-year-old woman presented with a ruptured superior cerebellar artery aneurysm. Because of her poor medical con-

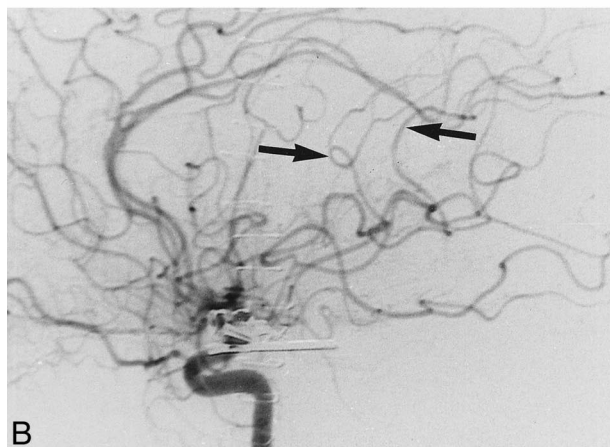
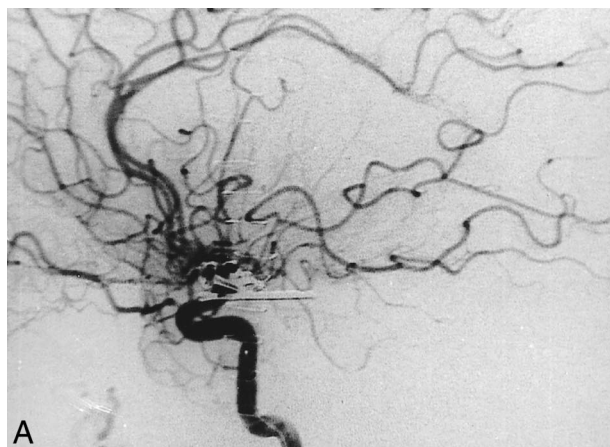


FIG 2. Left internal carotid arterial angiograms obtained in a 43-year-old woman who presented with a ruptured basilar artery aneurysm that was treated surgically.

A, After angioplasty, image shows thromboembolic occlusion of a parietal branch of the left middle cerebral artery.

B, Image obtained 30 min after bolus administration of abciximab depicts recanalization of occluded branches (arrows).

dition, the aneurysm was to be treated with GDCs. A severe stenosis of the right internal carotid artery with intraluminal thrombus extending from atherosclerotic plaque was identified during diagnostic angiography. During angiography, this thrombus embolized to the intracranial internal carotid artery. A decision was made to perform emergency angioplasty and to place a stent in the carotid artery in an attempt to restore perfusion. After emergency stent placement, small branch oc-

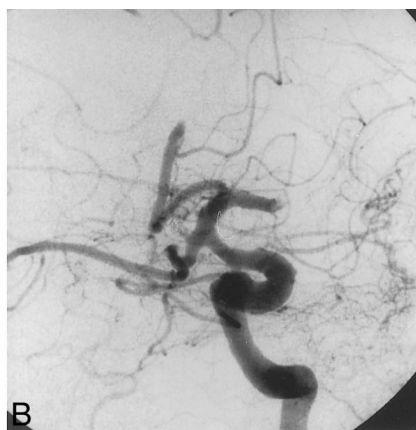


FIG 3. Left internal carotid arterial angiograms obtained in an 81-year-old woman who presented with a ruptured left posterior communicating artery aneurysm.

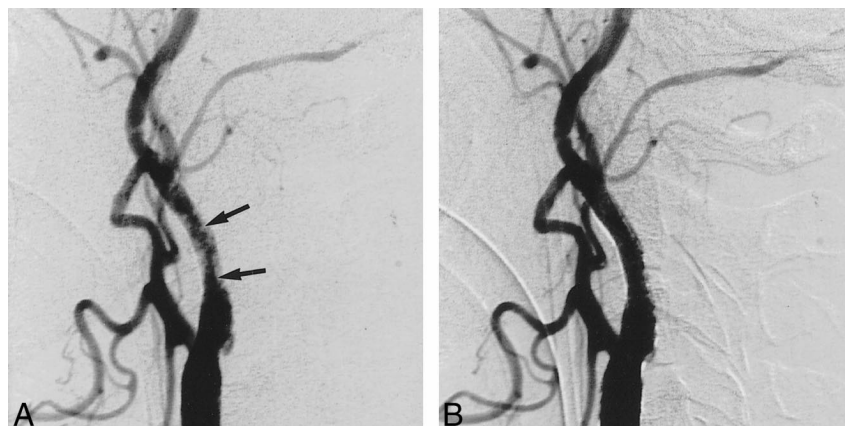
A, Immediately after placement of a guiding catheter in the left internal carotid artery, emboli were noted in the left anterior and middle cerebral arteries (arrows).

B, Image obtained 50 min after bolus administration of abciximab reveals no change in the angiographic appearance of the thromboemboli.

FIG 4. Carotid angiograms obtained in a 65-year-old woman who presented with a ruptured superior cerebellar artery aneurysm.

A, Shaggy thrombus formed on a carotid stent (arrows).

B, Image obtained 30 min after bolus administration of abciximab shows resolution of thrombus.



clusions remained in the carotid territory. It was decided to forgo further interventions to treat these small branch occlusions and to proceed with GDC embolization of the aneurysm. After GDC embolization of the aneurysm, angiography revealed formation of a shaggy thrombus within the lumen of the stented artery (Fig 4A). In an attempt to dissolve the intraluminal thrombus, an IV bolus of 0.25 mg/kg abciximab was administered; this was followed with a maintenance infusion of 10 μ g/min. This bolus was administered 20 min after initial identification of the thromboemboli. Angiography performed 30 min after the bolus administration of abciximab demonstrated complete resolution of the thrombus (Fig 4B). The small intracranial branch occlusions did not resolve with the abciximab infusion. Brain death developed over the next 24 h as a result of her initial subarachnoid hemorrhage.

Discussion

We describe the use of abciximab for the treatment of thromboembolic complications of endovascular procedures. After IV administration of abciximab, the thrombus resolved within 30 min in cases 1, 2, and 4, whereas no thrombolysis occurred in case 3, despite the addition of intraarterial t-PA. In 1997, Wallace et al (1) described the use of abciximab in neurovascular applications to prevent rethrombosis in the basilar artery after balloon angioplasty. In 2000, we reported the successful use of abciximab to treat acute carotid stent thrombosis (2). Another case report (3) recently described the use of IV abciximab in a thrombolytic role as rescue therapy for treatment of acute parent vessel thrombosis during GDC embolization of an intracranial aneurysm. Samuels et al (4) recently presented encouraging preliminary data about the use of abciximab to prevent complications during vertebrobasilar angioplasty.

Experience with glycoprotein IIb-IIIa antagonists for the treatment of acute ischemic stroke currently is limited. In a recently published study (5) designed to evaluate the safety of abciximab in acute ischemic stroke and to obtain pilot efficacy data, abciximab was administered to 54 patients within 24 h of onset of an acute ischemic stroke. These investigators found no increase in hemorrhagic complications, and they noted a trend toward better functional outcome in patients treated with abciximab (5).

Abciximab is approved by the FDA for the prevention of thrombotic complications of percutaneous coronary interventions. An emerging use for abciximab is rescue in patients with acute coronary stent thrombosis (6–9). Abciximab has also been used to facilitate thrombolysis in the setting of acute myocardial infarction (11–14). Two other glycoprotein IIb-IIIa antagonists, eptifibatide and tirofiban, are approved by the FDA for use in the treatment of acute coronary syndromes. The mechanism of enhancement of thrombolysis by glycoprotein IIb-IIIa antagonists is not yet understood. Potential mechanisms include: 1) prevention of platelet aggregation leading to reduction in thrombus mass and the platform for further thrombin generation; 2) inhibition of the release of local inhibitors of thrombolysis by platelets; 3) weakening of the clot structure by decreasing the binding of factor XIII, fibrin, and alpha-2 plasmin inhibitor; and 4) reduction of platelet-mediated clot retraction and reduction of the gel elastic modulus (11).

Abciximab is potentially useful to facilitate thrombolysis in the setting of thromboembolic complications of endovascular procedures. Our experience is currently limited to only four patients, with recanalization occurring in three. Recanalization of the occluded arteries in these patients might have occurred without the administration of abciximab. In patients with middle cerebral artery occlusion treated with heparin alone, the recanalization rate is 18% (15). In a study of patients with myocardial infarction treated with low-dose heparin and IV abciximab, the angiographic recanalization rate was 32% (11). The recanalization rate for patients with acute myocardial infarction treated with combined abciximab and t-PA was 77%, whereas it was 62% for patients treated with t-PA alone (12). The safety of the combined use of heparin, abciximab, and t-PA in the presence of a ruptured cerebral aneurysm or a cerebral infarction or both is unknown. We used this potentially dangerous combination of drugs in only case 3, because we believed that an infarction of the left middle cerebral artery territory would likely be fatal. The reason that the emboli in this patient failed to lyse

despite the administration of abciximab and t-PA is not known with certainty, but these emboli may have been atheroemboli rather than thromboemboli. When the use of abciximab is compared between patients with acute myocardial infarction and patients with cerebral ischemia, it is important to realize that the comparison is not entirely valid, because patients with myocardial infarction typically have atherosclerosis with in situ thrombosis, whereas patients with cerebral ischemia typically have thromboembolism.

Caution should be used with the administration of abciximab in patients with cerebrovascular disease. It is probably wise to optimize the anticoagulation with heparin and to observe the thrombus before initiating treatment of neurovascular complications with glycoprotein IIb/IIIa inhibitors. Abciximab affects platelet function for several days after its administration, and its action cannot be easily reversed. Further evaluation of glycoprotein IIb/IIIa inhibitors in patients with cerebral thromboembolic events is necessary to prove clinical efficacy.

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