Catheter-Directed Intra-Arterial Abciximab Administration for Acute Thrombotic Occlusions during Neurointerventional Procedures

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Summary

Abciximab is one of a new class of platelet aggregation inhibitors that has to date been used mainly in the management of acute coronary ischaemic syndromes or during cardiac intervention for the prevention and treatment of acute vessel occlusion during and after angioplasty or stent placement. More recently, it has begun to play a similar role in neurointerventional work. Its administration during acute stent or vessel occlusions has usually been via systemic intravenous infusion. We describe five cases of acute vessel occlusion during neurointerventional procedures where the abciximab bolus was administered intra-arterially at or close to the site of the occlusion, with rapid complete visual dissolution of the thrombus in four cases and partial dissolution in one, resulting in two patients with no neurological deficits, one with no further neurological deterioration, one with a mild residual thumb paresis and one with a severe neurological deficit.

Introduction

Acute thrombotic or embolic vessel occlusion is a potential complication of any neurointerventional procedure¹. When this occurs the management includes general supportive measures such as the administration of oxygen, in-

travenous fluids, plasma volume expanders and systemic heparin². More direct treatment involves the use of catheter-directed thrombolytic agents such as urokinase and tissue-plasminogen activator³.

More recently, the use of Abciximab has been described for the management of acute vessel occlusion during neurointerventional procedures^{4,5,6,7}. Abciximab is one of a group of drugs that produce selective blockade of the IIb/IIIa glycoprotein receptors on the surface of platelets, thereby inhibiting platelet aggregation. It also has been shown to have a lytic effect on acute thrombus as described in a number of reports. In most of the cases described to date in either cardiology or neurointervention, the abciximab is administered as an initial bolus dose via a peripheral intravenous route, followed thereafter by a 12-hour infusion to maintain the prevention of platelet aggregation.

Our decision to first use catheter-directed intra-arterial abciximab infusion at or close to the site of vessel occlusion was the result of a number of personal anecdotal experiences described to us by a number of cardiologists working in and around our own institution, who had described good success in using catheter-directed abciximab infusion for cases of intra-procedural stent or coronary artery occlusions during cardiac interventions.

Bolstered by the success of our first case utilizing this technique, we have used the same method in four further cases with a total of four undergoing complete visual clot dissolution and one partial dissolution. The purpose of this paper is thus to describe the methods used and results of our catheter-directed intra-arterial abciximab administration experience in acute peri-procedural intracranial vessel occlusion.

Methods

Between January 1995 and June 2001, a total of 207 head and neck and neurointerventional procedures of various kinds had been performed at our institution. During this period we identified a total of six significant thrombotic intraprocedural complications (being acute intraprocedural thrombotic occlusion of an intracranial vessel despite heparinization) that were recognized clinically or angiographically either during or immediately after the procedure. Five of these were managed with intra-arterial abciximab administration. A retrospective review of these cases was performed. In all cases the initial bolus dose of abciximab was calculated according to the known or estimated weight of the patient and then slowly infused over one minute through a microcatheter placed close to if not within the occluded vessel segment. All patients had been heparinized at this stage if not already from the onset of the procedure.

Table 1 shows a summary of the original presenting pathologies and proposed or performed method of treatment, as well as the type of anaesthesia given and use of systemic heparin.

Table 2 is a summary of the vessel that was occluded by the thrombus, the position of the microcatheter during the abciximab infusion, time to response, results and clinical outcome.

Results

Five patients had thrombotic occlusive complications recognized during or immediately after a neurointerventional procedure. In three patients (Cases 1, 2 and 4) this became apparent because of an abrupt change in clinical neurological status and in two patients (Cases 3 and 5) who were fully anaesthetized this was

discovered during check angiography during or immediately after the intervention.

In all patients the microcatheter being used at the time of the intervention was replaced into either the occluded vessel or as superselectively as possible into a position just proximal to the occlusion. The bolus dose of abciximab, calculated according to weight, was administered over a period of about one minute through the microcatheter. Complete angiographic clot lysis was seen in four cases with clot fragmentation and distal embolization in one (Case 4). In all cases there was rapid dissolution of the thrombus within five minutes, often in as little a period of time as it took to remove the syringe containing the abciximab, flush the microcatheter and perform a check arteriogram. In all cases a further continuous 12-hour abciximab infusion was given as an intravenous infusion after the initial bolus.

In two of the cases there was complete clinical recovery with no post-procedural neurological deficits. In one case the patient was left with a mild residual thumb paresis. One patient who had already suffered a hemiparesis and dysphasia following earlier hemorrhage of an arteriovenous malformation suffered no further clinical deterioration resulting from the vessel occlusion and recanalization. In the patient with the epistaxis there was only a partial response to attempted clot dissolution converting a complete distal internal carotid arterial occlusion to a resistant partial middle cerebral and complete posterior cerebral occlusion on the same side, leaving the patient with a residual hemiplegia and homonymous hemianopia.

There were no deaths and no intracranial haemorrhagic complications in any patient. No major systemic haemorrhagic complications occurred in any patient. All patients had their femoral access sheaths removed following cessation of the abciximab and heparin infusions at an activated clotting time of <160 s. No groin haematomas occurred. No other abciximab-related complications were encountered.

Case 1

A 55-year-old woman presented with a severe acute headache but no neurological deficits. A subarachnoid hemorrhage was confirmed at CT. Cerebral Angiography showed a broad-based left middle cerebral bifurcation

aneurysm with no adjacent vessel spasm. A Fas-Tracker 18 microcatheter (Target Therapeutics Inc, Fremont, CA, USA) was used to catheterise the aneurysm lumen. The patient was awake, and was heparinised with an activated clotting time of greater than 300 seconds. Three Guglielmi Detachable coils (GDC) (Target Therapeutics Inc, Fremont, CA, USA) were initially placed into the aneurysm with no complication. However following placement of a fourth coil the patient became stuporous and developed a right sided hemiparesis and aphasia. Angiography showed a filling defect in one of the middle cerebral divisions just distal to the aneurysm base. The microcatheter was withdrawn out of the aneurysm lumen and

placed just proximal to the thrombus. A bolus of 15 mg of abciximab (ReoPro, Centocor BV, Netherlands) was given immediately through the microcatheter. Within a minute of the bolus injection she regained full consciousness with normal speech but with some residual weakness of the right arm. Angiography showed lysis of the thrombus with restoration of flow through the occluded vessel. A continuous overnight infusion of abciximab was given at a dose of 0.01 mg/kg/minute. An MR scan done the following day showed a small area of ischaemia in the left parietal lobe. She was discharged two days later. A slight paresis of her right thumb was present at a three month follow-up examination.

Table 1

Patient No	Age and sex	Presenting	Goal of Pathology	Anaesthesia treatment	Systemic heparinization
1	55 y F	L / MCA bifurcation Aneurysm	GDC embolization	Local	Yes
2	70 y F	Type I Dural AVF	External carotid branch embolization	Local	No (flushes only)
3	33 y M	L / insular AVM	Pre-operative embolization	General	Yes
4	68 y M	Epistaxis	Embolization	Local	No (flushes only) given later in procedure
5	48 y F	R / frontal AVM	Embolization	General	Yes

Table 2

Patient No	Vessel occluded	Position of microcatheter for abciximab infusion	Time to clot lysis / clinical response	Result	MR post complication	Clinical Outcome
1	M2 / Post MCA bifurcation branch	M1 segment	2 minutes	Complete angiographic	Small infarct L/ Parietal lobe lysis	Mild paresis of R thumb at 3 months
2	MCA Bifurcation	M1 segment	2 minutes	Complete angiographic lysis	-	No neurological deficits
3	M1 segment L/ MCA	M1 segment	< 5 minutes	Complete angiographic lysis	-	No further neurological deterioration
4	Terminal ICA	Terminal ICA	< 10 minutes	Clot fragmentation and migration into MCA and PCA	Large infarct mid and posterior R/ cerebrum	Hemiplegia and homonymous hemianopia
5	A1 and M1 segment	Terminal ICA	< 5 minutes	Complete angiographic lysis	-	No neurological deficits

Case 2

A 70-year-old woman was referred for management of a left-sided Type 1A dural arteriovenous fistula (DAVF) supplied by occipital, posterior auricular and middle meningeal branches of the left external carotid artery (ECA). Treatment was planned due to an intolerable bruit.

Catheterisation of the external carotid artery proved very difficult due to highly tortuous vessels.

A 5F Headhunter Type 1 catheter (Cook, Bloomington, USA) was placed into the ECA as a guiding catheter through which a Tracker-18 Unibody micro-catheter (Target Therapeutics Inc, Fremont, CA, USA) was used to selectively catheterize the occipital artery, the largest of the feeders to the DAVF. This was embolised with cyanoacrylate (Histoacryl, B. Braun). The remaining feeding vessels were considered too small to catheterize at the same sitting and all catheters were withdrawn and the access sheath removed. She had not been heparinised during the procedure although heparinised flushes (1000 u in 1000 ml of 0.9% saline) had been used. The patient had remained awake and fully conversant during the procedure, requiring only light sedation with a benzodiazepine agent at the onset. The procedure had taken almost two hours. From about 30 minutes into the procedure we had noted episodic involuntary jerking of the right leg for which no explanation was initially available. During external compression of the femoral artery at the end of the procedure she became acutely unrousable with a right sided hemiparesis. Access was gained immediately via the left groin and repeat angiography showed acute occlusion of the left middle cerebral artery (MCA) at its bifurcation. A micro-catheter was placed into the M1 segment and a bolus dose (of 15 mg) of abciximab was administered at this site.

5000 u of Heparin was also given intravenously at this stage. Within 2 minutes the patient regained consciousness. She had no recall of the end of the embolisation procedure but was otherwise fully orientated. No residual neurological deficits were noted with complete resolution of the right hemiparesis. Complete lysis of the occluding thrombus was noted angiographically with re-establishment of normal flow through the MCA.

A review of the earlier diagnostic runs demonstrated that the MCA thrombus had been present prior to the embolisation but was not occlusive at that stage and had not initially been noticed as all attention had been focussed on the ECA and we had no clinical suspicion then of any abnormality. In retrospect, however, the involuntary intermittent spasms of the right leg had probably been heralding the development of the stroke. She was maintained on an overnight infusion of abciximab and heparin and made a full clinical recovery with no residual or recurrent neurological deficits.

Case 3

A 33-year-old male presented with an acute left intra cerebral hemorrhage. Clinically he had a right-sided hemiplegia and expressive aphasia. Angiography was performed showing an arteriovenous malformation (AVM) in the left insular region. His neurological deficits improved progressively over the next two weeks to a milder hemiparesis and dysphasia. He was scheduled for pre-operative embolisation. This was done under general anaesthesia. After the initial diagnostic angiographic survey and during preparation for microcatheter insertion an acute occlusion of the M1 segment of the left middle cerebral artery (MCA) was noted. A FasTracker microcatheter (Target Therapeutic's Inc, Fremont, CA, USA) was then placed into the MCA. Systemic Heparin (5000 u) was given and a bolus dose of 17.5 mg of abciximab was infused through the microcatheter. Within minutes flow was re-established in the MCA. All catheters were withdrawn. The patient was maintained on a overnight infusion of abciximab. His clinical condition remained unchanged with no deterioration after the procedure. Two weeks later the AVM was excised and he was discharged one week later for stroke rehabilitation still with some residual right-sided hemiparesis and dysphasia.

Case 4

A 68-year-old male patient was referred for endovascular management of acute uncontrollable epistaxis which was ongoing despite anterior and posterior nasal packing. Clinically the bleeding was from the left nasal cavity. Associated clinical conditions included hypertension

Figure 1 Case 1: A) Selective left middle cerebral arteriogram showing coils within the bifurcation aneurysm. A filling defect indicating the acute thrombus formation is seen in one of the M2 trunks (arrow). B) After abciximab infusion through the microcatheter placed in the M1 segment there is total lysis of the thrombus with normal flow in the same vessel.

and emphysema. He had been badly assaulted six weeks prior to the epistaxis including a number of blows to the face and head but had not consulted any doctor at that stage. Upon admission his haemoglobin was 19.2 g/dl. The platelet count was normal. He had been injected with Tranexamic Acid (Cyklokapron, Pharmacia and Upjohn) for clot stabilisation by the referring Ear, Nose and Throat specialist. Angiography was performed from the right groin. Systemic Heparin was not used initially. Selective catheterisation of all vessels proved difficult due to severe arterial tortuosity. The first vessel catheterized was the left common carotid artery (CCA) using a 5F Glidecath (Terumo Corporation, Japan) placed just proximal to the carotid bifurcation.

After excluding any abnormalities of the left internal carotid artery (ICA), the left internal maxillary artery (IMAX) was catheterised using a Mass Transit microcatheter (Cordis, Johnson & Johnson, USA) and embolised with Embospheres (Biosphere Medical, Louvres, France) 500 -700 μ m and 700 - 900 μ m in size. With the microcatheter still in situ the nasal packing was removed. The hemorrhage continued. The right external carotid artery (ECA) was then catheterized with the same 5F Glidecath and the right IMAX was catheterized with the same microcatheter and also embolised.

The bleeding persisted and the left facial artery was then catheterized and embolised using a 5F Simmonds 2 catheter as a guiding catheter for greater stability. The bleeding still persisted and we decided to assess the right facial artery. During the second catheterisation of the right CCA it was noted that there was now an acute occlusion of the terminal right ICA. At this stage the patient became stuporose and developed an acute left hemiplegia. He was then anticoagulated with 5000 u of heparin intravenously. A Tracker-18 Unibody microcatheter (Target Therapeutics Inc, Fremont, CA, USA) was placed into the distal ICA. An occlusion at and distal to the level of the ophthalmic origin was seen. A bolus dose of 18 mg of abciximab was given immediately at this site. By the end of the bolus administration flow was seen in the ophthalmic artery but not in the terminal ICA. A check of the cross-over supply showed only partial left-to-right filling of the right anterior cerebral artery (ACA). No back-to-front cross filling was seen and no flow in the right posterior cerebral artery (PCA) was seen. The right CCA was again catheterized. The occluding ICA thrombus had fragmented and flow was now seen in the distal ICA, ACA and in part of the distal MCA territory. Residual clot was seen in the lower division of the MCA bifurcation as well as in the dominant right posterior

communicating artery. All catheters were withdrawn at this stage. The nose was re-packed in the angiography suite due to continued slow bleeding. An infusion of abciximab was then commenced at a dose of 0.01 mg/kg/minute. After consultation with the resident neurologist and ear, nose and throat specialist we decided against the use of alteplase (Actilyse, Ingram Pharmacies) as an adjunct to the abciximab in light of the ongoing epistaxis.

An MR scan later showed the presence of a large infarct in the right middle and posterior cerebral territories including the right thalamus, with sparing of the anterior cerebral territory and parts of the frontal and anterior temporal lobes. At present the patient has a left hemiplegia and homonymous hemianopia and is currently undergoing stroke rehabilitation.

Case 5

A 48-year-old woman presented with a history of multiple previous surgical procedures performed for a large right sided frontoparietal arteriovenous malformation which had been diagnosed 15 years before. She now presented with retro-orbital headaches and progressively worsening paresis of the left arm. Cerebral angiography showed areas of residual nidus with other dilated vessels suggestive of probable secondary angiogenesis in and around the surgical site. She was presented for partial targeted embolisation in order to prevent further neurological deterioration. During this procedure she was placed under full general anaesthesia and heparinized systemically with an activated clotting time (ACT) of around 300 seconds. A Nimodipine infusion was started at 7.5 ml/hour. Initially a Spinnaker flow-directed catheter (Target Therapeutics Inc, Fremont, CA, USA) was used in an attempt to reach the target area through very tortuous vessels. During this attempt spasm also developed in the terminal ICA with a reduction of flow in both the middle and anterior cerebral arteries. This was followed almost immediately by occlusion of the ipsilateral A1 vessel. The ACT at this stage was 270 seconds. A FasTracker microcatheter (Target Therapeutics Inc, Fremont, CA, USA) was placed into the terminal ICA. During attempted recatheterisation of the A1 vessel occlusion of the M1 vessel occurred. A bolus dose of 15 mg of Abciximab was given

through the microcatheter followed by a 60 mg bolus of Papaverine. The occlusion in the A1 and M1 segments resolved completely with some residual focal spasm in the terminal ICA. The procedure was terminated at this point.

The patient was awakened and returned to Intensive Care Unit with further overnight infusion of Abciximab. No neurological defects were detected at any stage after this procedure, and she was discharged two days later.

Discussion

Abciximab (c7E3 Fab) is the fragment antigen-binding (Fab) fragment of the chimeric monoclonal antibody 7E3. It is directed against the glycoprotein IIb/IIIa (GP IIb/IIIa) receptors located on the surface of human platelets. Once bound to the receptors it inhibits platelet aggregation by preventing the binding of fibrinogen, Von-Willebrand-factor and other adhesive ligand plasma proteins to the GP IIb/IIIa receptor sites on activated platelets. It is this protein binding to the receptors that mediates the recruitment of additional platelets into a platelet aggregation that is later stabilized by Fibrin^{8,9}. Abciximab has also been shown to bind to other receptors including the $\alpha v \beta_3$ or Vitronectin receptor¹⁰. This receptor is found not only on platelets but on other cells including smooth muscle and endothelial cells, and plays a role in cell adhesion, proliferation and migration, and in particular in angiogenesis and intimal hyperplasia.

Abciximab is one of several similar anti-platelet aggregation agents currently in use. Others include Eptifibatide (Integrilin, Schering-Plough) and Tirofiban (Aggrastet, MSD) which also act via inhibition of the GP IIb/IIIa receptors. To date, the use of these agents has mainly related to the prevention of thrombotic occlusion during percutaneous cardiac intervention and for the treatment of acute coronary ischaemic syndromes^{11,12,13}. A number of reports also describe its use in the management of acute thrombotic occlusions including during and immediately after angioplasty or stent placement in coronary vessels¹⁴.

The successful usage of abciximab for salvage after acute vessel occlusion during and after neurointerventional procedures has also been described more recently^{4,5,6,7}. In all cases the abciximab bolus was administered intra-

A

B

Figure 2 Case 2:

A) Selective left common carotid arteriogram showing the Type 1 dural arteriovenous fistula. One of the post-bifurcation M2 trunks is occluded at this stage, but this is missed initially only becoming evident at the end of the procedure by a sudden change in clinical status.

B) Lateral carotid arteriogram following microcatheter-directed abciximab administration into the ipsilateral M1 vessel. Flow has been re-established in the previously occluded vessel within minutes.

venously. In cases where abciximab was the sole agent used and a good response was seen the clot lysis occurred within 10 to 30 minutes after onset of the infusion of the abciximab. In our four cases of total thrombus dissolution this occurred completely within minutes of the bolus administration, sometimes in the time taken to disconnect the syringe that contained the abciximab, flush the microcatheter, reconnect a syringe full of contrast and perform a check angiogram. Even in cases following coronary intervention much of the literature to date involves the systemic intravenous administra-

tion of abciximab in the acute occlusive setting. Two reports in the recent cardiological literature involve the administration of abciximab directly at the site of occlusion, suggesting that there may be greater degree of benefit by this direct administration with more rapid clot lysis^{15,16}. These, together with our own observations suggest that the rate of clot dissolution is more rapid when this technique is used.

We elected to use abciximab rather than the more usual fibrinolytic agents in the setting of acute intracranial vessel occlusion for a number of reasons. Firstly we had had a number of

anecdotal reports from cardiologists in and around our institution who had already successfully used catheter-directed abciximab delivery for acute vessel or stent occlusions following coronary interventions. Secondly, as we in South Africa perform general intervention in addition to neurointervention we have had good experience in peripheral arterial thrombolysis, mainly with tissue-plasminogen activator, with mixed results. Thirdly, we feel that as thrombus that develops during an interventional procedure is hyperacute and platelet-rich, consisting of platelet aggregates not yet fully stabilized by fibrin that it would make sense to use an agent that acts on platelets rather than a fibrinolytic one.

Abciximab was originally intended to be a prophylactic agent aimed at the prevention of clot formation during intervention or propagation of clot in acute coronary ischaemia. It has since been shown to be capable of dissolving acute thrombus through a number of mechanisms not yet completely understood.

Abciximab has been shown to work synergistically with other thrombolytic agents as has been reported in a number of trials such as the TIMI14 trial¹⁷. The use of combinations of abciximab and other thrombolytics has been shown to be more beneficial in certain cardiovascular trials, but their combined use in neurointervention has not been established. We briefly considered the adjunctive use of tPA in case 4 where there was evidence of occlusive material that was resistant to the abciximab, but with ongoing epistaxis despite seemingly adequate internal maxillary and facial arterial embolization together with any unknown possible sequelae of the facial trauma 6 weeks prior to presentation we elected not to use any agent that could further aggravate the bleeding, although the 12-hour abciximab infusion was given after the initial bolus. The reason for the apparent further resistance to lysis after an initial response is unknown. We have speculated a number of possible reasons including the displacement of organized thrombus from a plaque in the tortuous extracranial carotid artery, atheroembolism, or embolization of an intimal fragment that resulted from the initial difficult catheterisation attempts. Based on more recent unpublished personal experience mechanical clot removal may have proven useful in this setting, although we did not have this

experience at that stage. A similar technique has been described by Chopko, et Al¹⁸.

In case 5 the occlusions were probably due to a combination of spasm and thrombosis and arguably the papaverine that was infused probably played a significant role in re-establishing flow through the MCA and terminal ICA, but the A1 vessel was clearly thrombosed and thus responded to the abciximab.

No significant haemorrhagic complications occurred in any of our cases. This included no haemorrhagic conversion of the cerebral infarcts demonstrated by magnetic resonance imaging in cases 1 and 4. Two recent meta-analysis looking at the risk of intracranial hemorrhage in patients treated with abciximab for acute coronary ischaemic syndromes or during coronary interventions both concluded independently that the use of abciximab and heparin does not cause a statistically significant increase in the incidence of intracranial hemorrhage than the use of heparin alone^{19,20}. The incidence of intracranial haemorrhage with abciximab treatment and adjunctive heparin (or aspirin) is around 0,12% to 0,16%.

Other potential complications that may occur with abciximab treatment include non-intracranial bleeding complications (1-4%), pulmonary alveolar hemorrhage (0.27%)²¹, and thrombocytopenia (0.3% - 1%). Anaphylaxis can occur with re-administration within weeks after the initial bolus. Being itself an antibody fragment the development of human-antichimeric antibodies to the C7E3 Fab has been observed in up to 6,6% of patients receiving abciximab. This can result in the development of hypersensitivity reactions when abciximab is re-administered to patients who have become sensitized to this product. The risk of such a reaction to be greatest if abciximab is re-administered within two weeks²².

Eptifibatide and Tirofiban are both synthetic peptides with none of the immunogenic problems associated with abciximab.

Other side effects of abciximab include hypotension, nausea, vomiting, bradycardia, and fever. The standard recommended dosage of abciximab consists of an initial single bolus of 0.25 mg/kg, usually administered intravenously, followed by a further continuous intravenous infusion of 0,01 mg/kg/minute for a further 12 to 96 hours. At these doses there is generally an 80% or greater degree of GPIIb/IIIa receptor

Figure 3 Case 4: A) Selective right internal carotid arteriogram, right anterior oblique projection, showing occlusion of the internal carotid artery at the level of, and including, the ophthalmic origin. B) After intra-arterial abciximab administration flow is re-established in and beyond the distal ICA. There is persistent occlusion of the ipsilateral dominant posterior communicating artery and posterior cerebral artery and the posterior branches of the middle cerebral artery. C) Axial T2 weighted magnetic resonance scan done 48 hours after the procedure shows infarction within the MCA and PCA territories.

A

B

C

blockade. The initial phase half-life following bolus administration is less than ten minutes during which time the infusion should be started. Upon cessation of the infusion, platelet function recovers over the next 24 to 48 hours. The effects of abciximab can only be reversed by the administration of fresh platelets. When abciximab is used in conjunction with heparin,

the activated clotting time should be maintained at around 200 seconds. Abciximab is supplied in powdered form and must first be dissolved in sterile water and filtered prior to administration. Eptifibatide and Tirofiban are available already in solution and can be administered immediately without the need for any preparation or filtering.

Conclusions

Our small series has again confirmed the efficacy of abciximab in the lysis of acute thrombus formation during neurointerventional procedures. Our results using the catheter-directed intra-arterial administration at or close to the site of occlusion suggests a more rapid clot lysis than in cases where the abciximab bolus is given intravenously. The use of a platelet-aggregation inhibitor seems to make more sense in the hyperacute situation where the occluding

thrombus probably consists mainly of aggregated platelets not yet fully stabilized by fibrin. Further evidence indicates that there is no increased risk of intracranial hemorrhage when abciximab is used with heparin than with heparin alone (although its use in the setting of acute aneurysmal rupture has not yet been clarified). These two facts thus suggest that abciximab is a useful and relatively safe agent to use in acute peri-procedural vessel occlusion, and that the rate of clot lysis may be faster if administered trans-arterially.

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