

## Contemporary Management of Postcatheterization Pseudoaneurysms

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A pseudoaneurysm (PSA) is a contained rupture; there is a disruption in all 3 layers of the arterial wall (Figure 1). PSAs may occur under 4 circumstances: (1) after catheterization (Figure 2); (2) at the site of native artery and synthetic graft anastomosis (eg, aortofemoral bypass graft); (3) trauma; and (4) infection (eg, mycotic PSA) (Figure 3). This review will focus on PSAs that occur after cardiac and peripheral endovascular procedures. PSAs occur when an arterial puncture site does not adequately seal. Pulsatile blood tracks into the perivascular space and is contained by the perivascular structures, which then take on the appearance of a sac. Hematoma and the surrounding tissue form the wall of the PSA.

Postcatheterization PSA is one of the most common vascular complications of cardiac and peripheral angiographic procedures. The incidence of PSA after diagnostic catheterization ranges from 0.05% to 2%.<sup>1</sup> When coronary or peripheral intervention is performed, the incidence increases to 2% to 6%. In 1 series where diagnostic ultrasound was performed on 536 consecutive patients who underwent catheterization, the incidence of PSA was 7.7%, with 83% of the PSAs associated with interventional procedures.<sup>2</sup> Despite a low incidence, PSAs are commonly encountered when more complex coronary and peripheral interventions are performed, especially with the use of potent antithrombotic and antiplatelet therapy. Since 1996, the number of peripheral interventions has more than doubled to an estimated 750 000 procedures in 2005.<sup>3</sup> In 2003, the Centers for Disease Control/National Center for Health Statistics estimated 1.4 million inpatient diagnostic cardiac catheterization procedures, and 1.2 million angioplasties were performed in the United States.<sup>4</sup>

It has been suggested that PSAs may thrombose spontaneously. In 1 study, spontaneous thrombosis occurred in 72 of 82 patients with PSA <3 cm at a mean of 23 days,<sup>5</sup> whereas in another prospective study only 9 of 16 patients had spontaneous thrombosis at a mean of 22 days. Failure to thrombose was associated with size >1.8 cm and concomitant use of anticoagulation or antiplatelet agents.<sup>6</sup> Most of the studies that suggested observation occurred prior to the era of aggressive antithrombotic and dual antiplatelet therapy. The

rate of spontaneous thrombosis in patients who take aspirin+clopidogrel or warfarin is really not known. In the absence of severe pain, observation of small PSAs (<2.0 cm) is reasonable. However, if the patient has severe pain, treatment is indicated.

The most catastrophic complication of PSA is rupture. Although the exact rate is unknown, the risk of spontaneous rupture of PSA is related to size >3 cm, presence of symptoms, large hematoma, or continued growth of the sac.<sup>5,7,8</sup> Although most postcatheterization PSAs are sterile, infection of a PSA significantly increases the risk of rupture as well as septic emboli.<sup>9</sup>

### Factors Associated With Pseudoaneurysm Formation

Several patient and procedural factors may contribute to the formation of PSA as shown in Table 1.<sup>10</sup> Of particular importance is the increased incidence of femoral artery PSAs when the puncture site is not in the common femoral artery, but rather is located in the superficial or deep femoral artery or the external iliac artery.<sup>11</sup> The complexity of interventions such as coronary stenting, atherectomy, intraprocedural thrombolytic therapy, and repeat coronary angioplasty has also been shown to increase the risk of vascular complications.<sup>12,13</sup> In a meta-analysis of randomized trials, there was no increased vascular complication rate with Angio-Seal (St. Jude Medical, St. Paul, Minn) or Perclose (Abbott Vascular Devices, Redwood City, Calif) for patients who underwent either diagnostic catheterization or percutaneous coronary intervention.<sup>14</sup> There was an increase in the vascular complication rate with VasoSeal (Datascope Corp, Montvale, NJ) (odds ratio, 2.25; 95% CI, 1.07 to 4.71). This study did not report the PSA rate, only the total vascular complication rate, a composite of PSA, arteriovenous fistula, retroperitoneal hematoma, femoral artery thrombosis, surgical vascular repair, access site infection, and blood transfusion.<sup>14</sup> In a meta-analysis that was performed by Korenny and associates and involved 30 trials and up to 4000 patients, closure devices showed a relative risk of PSA formation of 1.19 (95% CI, 0.75 to 1.88;  $P=0.46$ ). However, in the 2 studies that reported intention-to-treat analysis, the occurrence of PSA formation

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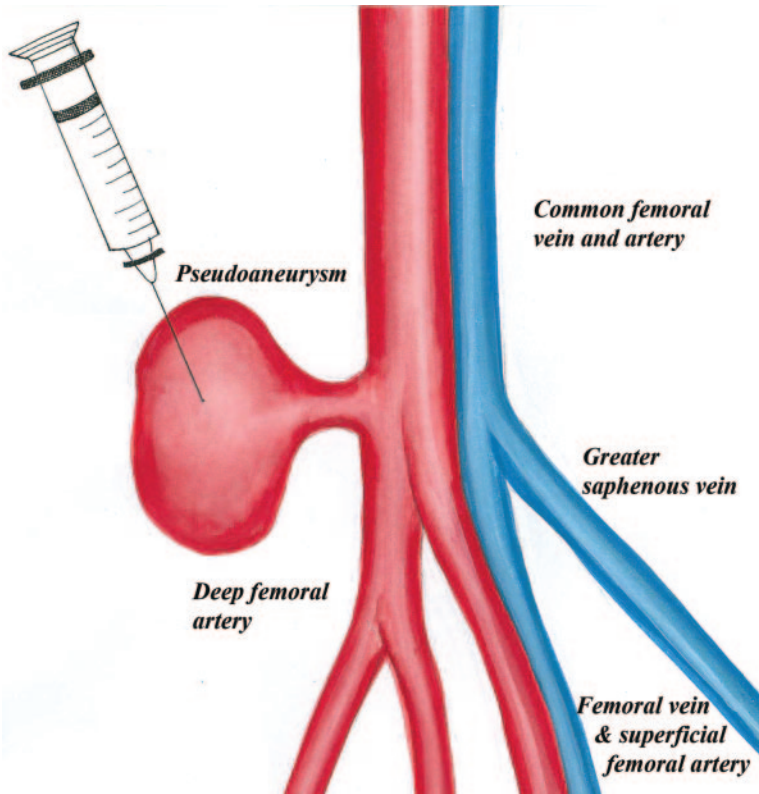
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**Figure 1.** Artist's rendition of a postcatheterization PSA with proper placement of the needle for thrombin injection. There is a disruption in all 3 layers of the arterial wall. In essence, this represents a contained rupture.

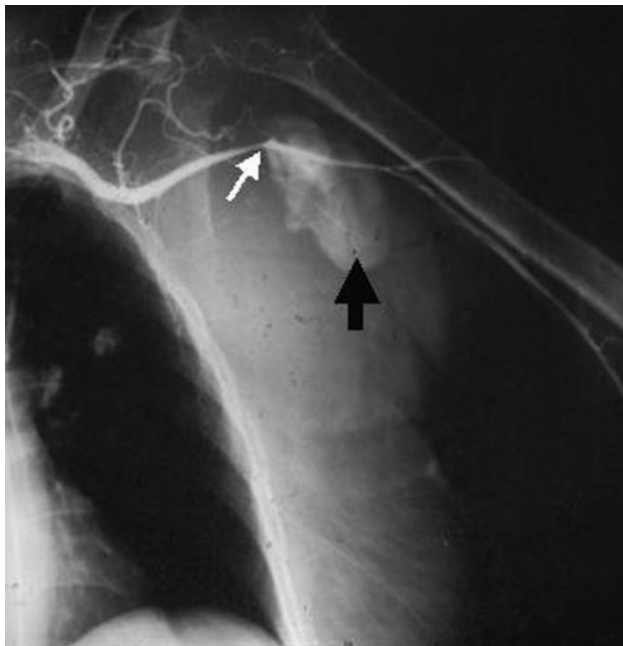
was 5.4 times greater with the use of closure devices compared with manual compression.<sup>15</sup> There are no good prospective data to predict who will develop a PSA. However, it makes intuitive sense that the accuracy of the initial puncture and the expertise and duration of compression of the

puncture site after the sheath is removed may be important factors in the development of PSAs. Vascular complications are less common when the interventionalist uses ultrasound<sup>16</sup> or fluoroscopy with localization of the femoral head<sup>17</sup> to puncture the femoral artery in the correct location and with the first attempt (eg, no posterior wall puncture, especially in obese patients and those with weak pulses).<sup>18</sup>

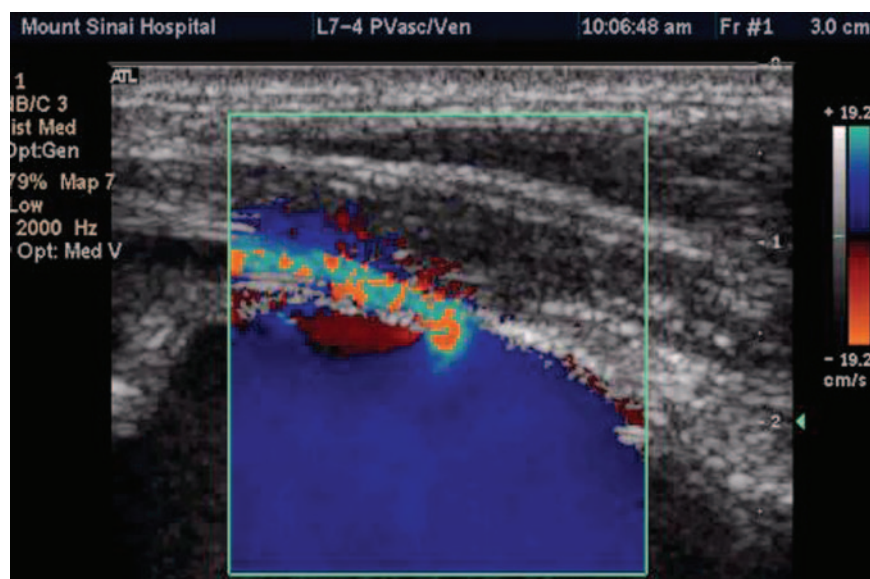
### Diagnosis

The presence of pain or swelling in the groin after catheterization is the most common presentation of a PSA. Swelling from a large PSA or hematoma may also lead to compression of nerves and vessels with associated neuropathy, venous thrombosis, claudication, or, rarely, critical limb ischemia. Local ischemia of the skin may lead to necrosis and infection. On physical examination, there may be a palpable pulsatile mass or the presence of a systolic bruit. However, it should be noted that none of these aforementioned physical findings may be present. Any patient who experiences pain that is disproportionate to that expected after a percutaneous procedure should undergo an ultrasound examination to exclude the presence of a PSA regardless of the presence of a bruit.

The diagnostic examination of choice is duplex ultrasound with a 5- to 7-MHz linear transducer. If the puncture site is high (eg, external iliac artery) or there is extensive hematoma, a lower frequency transducer (curved linear or sector array) may be required to image deeper structures. B-mode scanning alone is unable to differentiate PSA from hematoma (Figure 4). Color Doppler enhances the diagnostic accuracy of ultrasound by identification of pulsatile flow within the sac



**Figure 2.** Arteriogram that demonstrates an axillary artery PSA after a diagnostic arteriographic procedure. Note the disruption in the vessel wall (white arrow) and the larger PSA sac (black arrow).



**Figure 3.** Ultrasound of a spontaneous PSA in the left brachial artery. Note the absence of a tract. The PSA chamber is directly connected to the brachial artery. This type of PSA must be treated surgically. This patient had infective endocarditis.

(Figure 5A).<sup>19</sup> The sensitivity of duplex ultrasound to identify a PSA is 94% with a specificity of 97%.<sup>20</sup>

The typical appearance of a PSA on ultrasound is demonstrated in Figures 4 and 5. The B-mode image shows an echolucent sac that expands and contracts with cardiac contraction (Figure 4). On color Doppler, there is a swirling flow pattern with turbulence in the chamber(s), there may be 1 or more chambers noted. A tract connects the PSA chamber to the feeding vessel (most commonly the common femoral artery) (Figure 5A). When a pulsed wave Doppler is placed within the track, a “to-and-fro” signal is obtained, which signifies that this is in fact a PSA (Figure 5B). In addition to identification of the number, size, and depth of the chambers of the PSA, the depth, width, and length of the tract that connects the artery to the PSA should be identified. It is also important to clearly identify the vessel that feeds the PSA in both transverse and long-axis views as well as record flow in the vessel proximal and distal to the PSA. The venous structures should be clearly identified to rule out the presence of a deep venous thrombosis secondary to prolonged compression on the groin or pressure from an expanding hematoma.

There are an increasing number of procedures performed from the arm, which thus increases the likelihood of PSAs in this location. The same principles apply to diagnosis and treatment of PSAs in the arm and leg.

One common mistake during ultrasound examination is to image too superficially. It is important to increase the depth on the ultrasound machine so that deep PSAs (>4 cm from the skin) are not overlooked. A complete examination should include imaging of the mid and distal external iliac artery, common femoral artery, and proximal portions of the superficial femoral and profunda femoral arteries.

## Treatment

Until the early 1990s, the only treatment available for PSA was surgery. Since that time, ultrasound-guided compression (USGC) repair, ultrasound-guided thrombin injection (UGTI), and a whole host of other treatment modalities such as FemStop compression devices,<sup>21</sup> coil insertion,<sup>22</sup> fibrin adhesives,<sup>23</sup> or balloon occlusion have been used with variable success. The 3 most common treatment strategies are discussed below in more detail.

## Surgical Management

Surgical management of PSA is still an important and necessary management strategy in a minority of patients with PSA (Table 2). Any PSA that occurs at the site of a vascular anastomosis (eg, aortobifemoral bypass) should be repaired surgically because it results from a disruption at the suture site and may be caused by infection. Likewise, spontaneously occurring PSAs are often mycotic (Figure 3) and should be repaired surgically. Compression on underlying structures by an expanding pulsatile mass, which causes claudication, neuropathy, or critical limb ischemia, requires urgent surgical decompression and resection of the PSA. Rarely, the PSA is so large that it has or will cause skin necrosis. In this situation it is imperative to decompress this area surgically.<sup>5,10,24</sup> However, surgery is rarely employed to treat the usual postcatheterization PSA. The disadvantages of surgery for the

**TABLE 1. Factors Associated With the Formation of Pseudoaneurysm**

Antiplatelet agents (often aspirin and clopidogrel)
Anticoagulation
Large sheath size, >8F
Age >65 years
Obesity
Poor postprocedural compression
Simultaneous artery and vein catheterization
Hypertension
Peripheral arterial disease
Hemodialysis
Complex interventions
Low or high puncture sites





**Figure 4.** B-mode ultrasound of PSA with echolucent sac. The only way to tell if this is a PSA or a hematoma is to use color Doppler. If flow is present, it is a PSA. If flow is absent, it is consistent with a hematoma.

treatment of PSA are that it requires anesthesia and an incision usually in the groin, an area known to become infected easily after a surgical procedure. Lumsden and colleagues reported a surgical complication rate of 20% after PSA repair. Complications included bleeding, infection, neuralgia, prolonged hospital stay, perioperative myocardial infarction, and, rarely, death.<sup>25</sup>

### Ultrasound-Guided Compression

In 1991, Fellmeth and associates introduced a safe and noninvasive method to treat PSA: USGC.<sup>26</sup> USGC has been shown to have a success rate of 75% to 98%.<sup>27–29</sup> With the use of ultrasound to identify the chamber and tract of the PSA, the ultrasound transducer is positioned and pressure is applied to compress the chamber and tract while flow in the native artery is allowed. Direct ultrasound visualization confirms cessation of flow into the PSA. Compression is usually held for cycles of 10 minutes. This can be repeated until success or until a discretionary failure time. Although it would be advantageous to use manual compression devices, the vertical angle created by the device does not allow selective compression of the PSA chamber and tract. Nonselective compression leads to longer compression times, more discomfort to the patient, and a lower success rate, in addition to an increase in the likelihood of complications such as deep venous thrombosis.<sup>29</sup> Body habitus, size, depth, and number of chambers, as well as concurrent anticoagulation may limit the success of USGC.

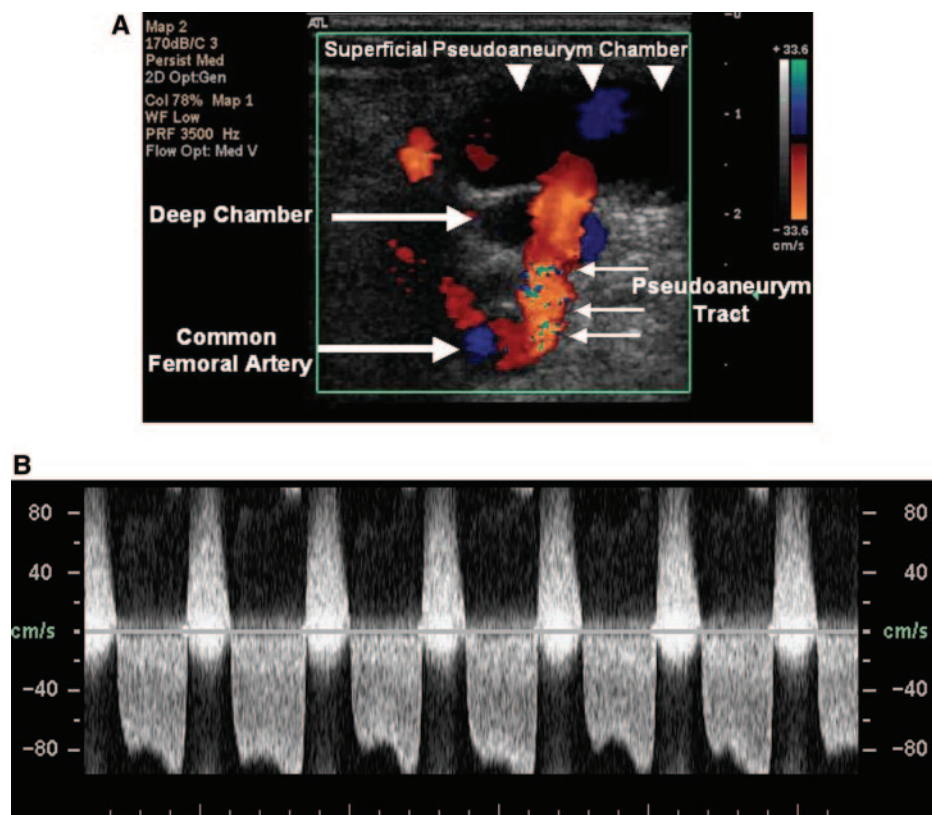
In patients on anticoagulation, the success has been reported to be in the range of 30% to 73%.<sup>27,30</sup> Two series from the same institution evaluated the role of USGC of postcatheterization PSAs in patients who did not receive anticoagu-

lation and in those who did.<sup>30,31</sup> In the first 100 cases of postcatheterization PSA, USGC was immediately successful in 94 patients (94%), which included 30 (86%) of 35 patients who received anticoagulation and 64 (98%) of 65 patients who were not on anticoagulation. There were recurrences in a total of 10 patients who subsequently underwent repeat USGC<sup>8</sup> or surgery.<sup>2</sup> We later demonstrated that USGC was successful in 56 (73%) of 77 patients who received anticoagulation. Seven (12.5%) of these patients required 2 or 3 compression attempts to induce sustained thrombosis.<sup>30</sup> There were no significant complications reported in either of these studies.

The main limiting factor in USGC is the time it takes to induce sustained thrombosis. The average compression time to achieve occlusion was 33 minutes with a range of 10 to 120 minutes.<sup>31</sup> This is a very painful procedure for the patients, and they often have to be pretreated with narcotic analgesia. In addition, it is hard work for the physician or ultrasound technologist who must apply constant hard pressure over the groin. It is difficult to maintain pressure in the correct position for prolonged periods of time. Rare complications have been reported such as vasovagal reactions, PSA rupture, skin necrosis, and deep vein thrombosis, although we have not experienced this in our large series.<sup>30,31</sup>

### Ultrasound-Guided Thrombin Injection

UGTI has become the treatment of choice for postcatheterization PSAs.<sup>32–34</sup> Percutaneous coagulation of a PSA was first reported by Cope and Zeit in 1986.<sup>35</sup> The procedure was later adapted to utilize ultrasound guidance with the injection of thrombin.<sup>36</sup> It should be noted that this is off-label use of thrombin. There is a warning on the thrombin package that



**Figure 5.** A, Color ultrasound of PSA. There are 2 chambers and a long track that connects the artery to each of the chambers. There is marked turbulence (not visualized on this still image) within the track and the chamber. B, When a pulsed-wave Doppler is placed in the PSA tract, one can see the typical “to and fro” signal that indicates that blood flows (often at a very high speed) both into and out of the PSA chamber. This is diagnostic of a PSA.

states “for topical use only, not for injection”. The principle of thrombin injection into the PSA chamber is based on the fact that thrombin is important in the conversion of fibrinogen to fibrin. Thus a fibrin clot is formed instantaneously (even in the presence of antiplatelet therapy or anticoagulation therapy) with UGTI, whereas it may take up to several hours with USGC. Success ranges from 91% to 100% in large series.<sup>32,37–49</sup> More than 45 individual series have been published on the safety and efficacy of this therapy. The cumulative overall success rate in 1329 PSA injections was 97%. Table 3 reports the success and complications in the largest series.<sup>32,37–49</sup>

Bovine thrombin, manufactured by GenTrac, Inc (Middleton, Wis) and distributed by Jones Pharma Inc (Bristol, Va), is available in a commercial kit, and it is generally reconsti-

tuted in normal saline to a concentration of 1000 U/mL. Several authors have advocated lower concentrations of thrombin (eg, 100 U/mL)<sup>50,51</sup> or the use of human thrombin because of the theoretical concept that it may produce a lower likelihood of allergic reactions.<sup>44,49</sup> In >400 thrombin injections, we have not experienced an allergic reaction to bovine thrombin.

#### Technique of Ultrasound-Guided Thrombin Injection

After informed consent is obtained, duplex ultrasound is performed as previously described. Distal pulses are confirmed manually and documented. The injection site is prepared in a sterile fashion, and local anesthesia is used with 1% to 2% lidocaine infiltrated into the skin and subcutaneous tissue.

Several injection techniques have been reported in the literature, but the technique that we use is the 3-way stopcock technique (Figure 6). This technique alleviates the need to change syringes during the procedure (Table 4).<sup>32</sup> Normal saline is placed into a 5-mL syringe via a 22-gauge needle. Three milliliters of bovine thrombin is placed in the other syringe, and they are connected to the 3-way stopcock. For most injections, a 1.5-inch 22-gauge needle is used. If the PSA is deeper than 3.75 cm, a 21 gauge  $\times$  9 cm Echotip (Cook Inc., Bloomington, Ind) needle that can be visualized under ultrasound will be used. With the thrombin “off” and the saline “on,” the needle is advanced under ultrasound

**TABLE 2. Indications for Surgical Repair of Pseudoaneurysm**

Infected pseudoaneurysm
Rapid expansion
Failure of other therapies
Skin necrosis
Compressive syndromes
Neuropathy
Claudication
Critical limb ischemia

**TABLE 3. Summary of Ultrasound-Guided Thrombin Injection Series**

First Author	Year	No.	Bedrest (Hours)	Percent Success	Dose (Units)	Recurrence	Complications	Embolic	Description of Complications
Kruger <sup>43</sup>	2003	50	12	100	357 vs 638	4	0	0	...
Calton <sup>37</sup>	2001	52	...	94	...	2	1	1	Thrombotic complication of native artery
Sheiman <sup>47</sup>	2001	54	1	93	1000+	4	0	0	...
Sheiman <sup>48</sup>	2003	54	3	91	703 vs 1510	5	0	0	...
Etemad <sup>38</sup>	2003	61	...	100	435.0	0	1	0	Vasovagal
La Perna <sup>32</sup>	2000	70	2	94	1150.0	3	1	0	Soleal deep vein thrombosis on follow-up with calf pain
Kang <sup>36</sup>	2000	83	1	99	820.0	7	1	1	Brachial artery thrombosis that resolved spontaneously
Mohler <sup>45</sup>	2001	91	NR	98	500 to 1000	2	1	0	Pulmonary embolus unrelated to procedure
Vasquez <sup>49</sup>	2005	96	6	99	1000 vs 300	0	3	0	Local erythema at injection site (bovine); groin pain×2 (human)
Maleux <sup>44</sup>	2003	101	...	98	200.0	2	0	0	...
Paulson <sup>46</sup>	2001	114	6	96	306.0	0	4	2	Infected abscess; blue toe; pain in leg and buttocks; anaphylaxis
Khoury <sup>41</sup>	2002	131	4	96	NR	9	3	2	2 intra-arterial thrombin injections with critical limb ischemia; PSA rupture after thrombosis
Grewe <sup>39</sup>	2004	132	0	99	669.0	0	0	0	...
Krueger <sup>42</sup>	2005	240	6 to 24	100	425.0	6	2	1	Allergic reaction (fever); decreased posterior and anterior tibial artery Doppler velocity
Overall		1329	...	97.5	...	44	17	7 (0.5%)	...

guidance. The needle and transducer are positioned in parallel to puncture the PSA chamber as superficial and far from the PSA tract as possible. Although the needle tip may sometimes be visualized on B-mode imaging, confirmation of proper needle placement is made with aspiration of arterial blood and small injections of saline to produce a flash of color within the chamber and allow direct visualization of the needle tip. Once appropriately placed, the thrombin port is

turned “on” and the saline port “off”. Aliquots of 0.2 mL are injected into the PSA chamber and visualized with color Doppler until no flow is observed. This usually occurs within seconds (Figure 7). The goal is to produce complete obliteration of flow in the chamber and tract. However, if tract flow continues, it usually disappears by the time the ultrasound is performed the next day. Under no circumstances should an injection be made directly into the tract, as this will increase the likelihood of thrombosis of the native artery. There is some controversy about how to approach patients with >1 chamber. Injection into the deeper chamber will almost certainly cause all other chambers to thrombose. However, the needle placement is closer to the native artery, and the potential for complications may be higher. We prefer to inject into the most superficial chamber first. This usually results in thrombosis of all chambers. However, if flow is still present in the deeper chambers, a second injection may need to be performed. Less than 1 mL of thrombin is used in virtually all cases, and many PSAs can be completely thrombosed with as little as 0.2 to 0.4 mL. Steps utilized in the 3-way stopcock technique are summarized in Table 4.

#### Results of Thrombin Injection

The primary success rate is very high at 91% to 100% (Table 3). In our early series of 70 consecutive patients who underwent UGTI, the PSA arose from the distal external iliac



**Figure 6.** Three-way stopcock for UGTI.



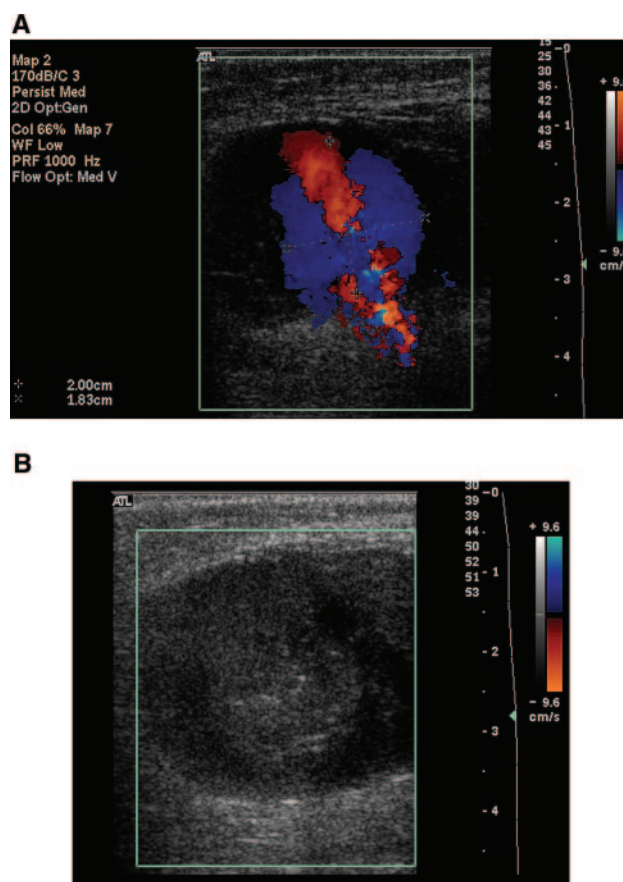
**TABLE 4. Three-Way Stopcock Technique**

1. Measure the distance from the skin to the most superficial portion of the pseudoaneurysm cavity and the distance to the pseudoaneurysm track.
2. Infiltrate the skin and subcutaneous tissue with 1% to 2% lidocaine.
3. With the 3-way stopcock assembled as described in the text (Figure 6), insert the needle to the depth measured in step 1 at the most superficial portion of the most superficial cavity. Apply negative pressure on the syringe that contains saline and when arterial blood appears in the syringe, inject saline to document that the tip is in the proper location. Be certain that the injection is not near the track itself. If it is, remove and start over. Be certain you are in the pseudoaneurysm cavity and not the artery or the vein.
4. Turn the stopcock and gently inject 0.2 mL of topical thrombin. Inject 0.2 mL aliquots until the pseudoaneurysm and tract are thrombosed or until 1.0 mL of thrombin is used.
5. If there are 2 or more cavities, inject the most superficial cavity first. Most of the time all cavities will thrombose. If not, go after the deeper cavities.
6. If the tract persists, follow the patient. It will usually thrombose by the next day. Never inject directly into the tract.
7. After successful thrombosis, image (pulsed wave and color Doppler) the external iliac, common femoral, superficial femoral, and profunda femoral arteries and their associated veins to document normal flow and no evidence of deep venous thrombosis.
8. This procedure may be performed as an outpatient. The patient is sent home immediately after injection.
9. Repeat the ultrasound in 24 to 72 hours to confirm continued thrombosis. If the pseudoaneurysm reopens, reinject on follow-up visit.

artery in 11%, common femoral artery in 65%, superficial femoral artery in 16%, profunda femoral artery in 4%, and brachial artery in 3%. A total of 66 of 70 patients (94%) had complete thrombosis of the PSA with thrombin injection; 20 of 21 anticoagulated patients (95%) had successful thrombin injections.<sup>32</sup> In this early series, 2 of the failures were in patients after aortic stent graft insertion, and the PSA tract was short and wide. The third failure was the first patient in the series and was likely a technical failure as a result of inexperience with the technique. Occasionally, the majority of the PSA thromboses but a small portion remains patent. In these situations, we use compression immediately after thrombin injection to manually disperse the thrombin into the open portion of the PSA.<sup>52</sup>

After cessation of the flow in the PSA, preservation of distal pulses should be documented if they were present prior to injection. Pulsed-wave and color Doppler flow should be documented in the artery and vein proximal and distal to the PSA and compared with images recorded prior to the intervention. Although bedrest is not required after injection, it is advisable to recommend avoidance of strenuous activity for 24 hours. We treat many of these patients as outpatients and let them ambulate immediately after the procedure. There is a recurrence rate of 0% to 9% when imaged the next day (Table 3). A follow-up duplex ultrasound is obtained within 24 to 72 hours after thrombin injection. Recurrence of PSA can be treated with a second (or third) injection with excellent results.

The most serious complications associated with thrombin injection are the development of deep venous thrombosis (if



**Figure 7.** A, PSA that arises from the common femoral artery. There is a thrombus in the PSA (black area around the color), and the color represents flow ( $2.0 \times 1.8$  cm) into the PSA. B, Image after UGTI. Note the thrombus present. There is no flow within the chamber.

the thrombin is inadvertently injected into the vein), pulmonary embolism,<sup>45</sup> or thrombosis of the artery (if thrombin is injected into the PSA tract or the artery itself). This is most likely to occur if an injection is made directly into the neck of the PSA.<sup>36,41</sup> Although some clinicians take the patient immediately to surgery should this complication occur, other patients have been observed while anticoagulated and reported spontaneous resolution with no significant clinical sequelae.<sup>46,53,54</sup> Although it has been suggested that short wide tracts may increase the risk of thrombin injection, we have not altered our indications on the basis of the tract anatomy and have not experienced an increased complication rate in our large series.

Allergic reactions and anaphylaxis have been reported in patients previously exposed to bovine thrombin.<sup>42,55,56</sup> Pope and Johnston recommend skin testing in patients with prior exposure to bovine thrombin;<sup>55</sup> this has not been observed in most reports and rarely skin testing is performed. There have been previous reports of antibodies that form against bovine factor V contamination of thrombin preparations. This has potential for serious hemorrhagic complications in humans; however, it has not been observed in any of the series of patients treated with UGTI. The overall complication rate from UGTI is 1.3% with an embolic rate of 0.5% (Table 3).

There is a definite learning curve, and the reported embolic complications are quite rare as one gains more experience with the technique of injection.

### Contraindications to Ultrasound-Guided Thrombin Injection

Thrombin injections should only be performed in patients who develop PSA secondary to a catheterization procedure. If a PSA occurs spontaneously, a mycotic PSA (Figure 3) should be suspected, and UGTI should not be undertaken. Additionally, a PSA that occurs at the anastomosis of a synthetic graft and native artery should be treated surgically and not with UGTI. The size of the PSA chamber is not, in of itself, a contraindication to UGTI. However, if the PSA is large enough to cause skin necrosis or compression of nerves or blood vessels, then surgery should be performed instead of thrombin injection (Table 2).

### Special Circumstances

Occasionally a PSA is noted on the completion angiogram, prior to removal of the sheath. If this occurs, there are several techniques that can be utilized to correct the situation at that time. Covered stents and coils have been utilized with good results.<sup>57,58</sup>

### Prevention

There are no prospective studies that have addressed measures to prevent the formation of PSA. More complex procedures and more potent antithrombotic therapy have led to the occurrence of more frequent PSA formation. The most important strategies to prevent PSA formation are:

- Assure a needle puncture in the proper location with the use of either fluoroscopy or ultrasound, thus achieve vascular access on the first puncture without access through the posterior wall.
- Appropriate groin compression after sheath removal. Adherence to the first makes this accomplishment easier.

### Conclusion

PSAs are common after catheterization procedures. Duplex ultrasound is the diagnostic method of choice because it is noninvasive, accurate, and cost-effective. Surgical repair of PSAs is rare but is indicated in patients who exhibit rapid expansion, infection, compression syndrome, or failure of UGTI. UGTI has an excellent primary success rate of 97% and a low complication rate of  $\leq 1.3\%$ . Proper technique with adequate ultrasound visualization of thrombin injected into the PSA will minimize embolic complications. Although USGC is safe and successful, procedural limitations have made UGTI the technique of choice for first-line management of PSAs that occur after percutaneous catheterization procedures.

### Disclosures

None.

### References

- Hessel SJ, Adams DF, Abrams HL. Complications of angiography. *Radiology*. 1981;138:273–281.
- Katzenschlager R, Ugurluoglu A, Ahmadi A, Hulsman M, Koppensteiner R, Larch E, Maca T, Minar E, Stumpfien A, Ehringer H. Incidence of pseudoaneurysm after diagnostic and therapeutic angiography. *Radiology*. 1995;195:463–466.
- Turf wars intensify over endovascular training. *CXVascular Interventional News*. 2005;20.
- Thom T, Haase N, Rosamond W, Howard VJ, Rumsfeld J, Manolio T, Zheng ZJ, Flegal K, O'Donnell C, Kittner S, Lloyd-Jones D, Goff DC Jr, Hong Y, Adams R, Friday G, Furie K, Gorelick P, Kissela B, Marler J, Meigs J, Roger V, Sidney S, Sorlie P, Steinberger J, Wasserthiel-Smolter S, Wilson M, Wolf P; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2006 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2006;113:e85–e151.
- Toursarkissian B, Allen BT, Petrinc D, Thompson RW, Rubin BG, Reilly JM, Anderson CB, Flye MW, Sicard GA, Sullivan TM. Spontaneous closure of selected iatrogenic pseudoaneurysms and arteriovenous fistulae. *J Vasc Surg*. 1997;25:803–809.
- Kent KC, McArdle CR, Kennedy B, Baim DS, Anninos E, Skillman JJ, Perry MO, Smith RB, Cox GS, McCann RL, Kresowik TF, Hye RJ. A prospective study of the clinical outcome of femoral pseudoaneurysms and arteriovenous fistulas induced by arterial puncture. *J Vasc Surg*. 1993;17:125–133.
- Graham AN, Wilson CM, Hood JM, Barros D'Sa AA. Risk of rupture of postangiographic femoral false aneurysm. *Br J Surg*. 1992;79:1022–1025.
- Kresowik TF, Khoury MD, Miller BV, Winniford MD, Shamma AR, Sharp WJ, Blecha MB, Corson JD. A prospective study of the incidence and natural history of femoral vascular complications after percutaneous transluminal coronary angioplasty. *J Vasc Surg*. 1991;13:328–336.
- Oweida SW, Roubin GS, Smith II RB, Salam AA. Postcatheterization vascular complications associated with percutaneous transluminal coronary angioplasty. *J Vasc Surg*. 1990;12:310–315.
- Morgan R, Belli AM. Current treatment methods for postcatheterization pseudoaneurysms. *J Vasc Interv Radiol*. 2003;14:697–710.
- Rapoport S, Sniderman KW, Morse SS, Proto MH, Ross GR. Pseudoaneurysm: a complication of faulty technique in femoral arterial puncture. *Radiology*. 1985;154:529–530.
- Muller DWM, Shamir KJ, Ellis SG, Topol EJ. Peripheral vascular complications after conventional and complex percutaneous coronary interventional procedures. *Am J Cardiol*. 1992;69:63–68.
- Popma JJ, Satler LF, Pichard AD, Kent KM, Campbell A, Chuang YC, Clark C, Merritt AJ, Bucher TA, Leon MB. Vascular complications after balloon and new device angioplasty. *Circulation*. 1993;88:1569–1578.
- Nikolsky E, Mehran R, Halkin A, Aymong ED, Mintz GS, Lasic Z, Negoita M, Fahy M, Krieger S, Moussa I, Moses JW, Stone GW, Leon MB, Pocock SJ, Dangas G. Vascular complications associated with arteriotomy closure devices in patients undergoing percutaneous coronary procedures: a meta-analysis. *J Am Coll Cardiol*. 2004;44:1200–1209.
- Koreny M, Riedmuller E, Nikfardjam M, Siostrzonek P, Mullner M. Arterial puncture closing devices compared with standard manual compression after cardiac catheterization: systematic review and meta-analysis. *JAMA*. 2004;291:350–357.
- Blank R, Rupprecht HJ, Schorrlepp M, Kopp H, Rahmani R. Clinical value of Doppler ultrasound controlled puncture of the inguinal vessels with the “smart needle” within the scope of heart catheter examination [in German]. *Z Kardiol*. 1997;86:608–614.
- Garrett PD, Eckart RE, Bauch TD, Thompson CM, Stajduhar KC. Fluoroscopic localization of the femoral head as a landmark for common femoral artery cannulation. *Catheter Cardiovasc Interv*. 2005;65:205–207.
- Dudek O, Teichgraber U, Podrabsky P, Lopez Haenninen E, Soerensen R, Ricke J. A randomized trial assessing the value of ultrasound-guided puncture of the femoral artery for interventional investigations. *Int J Cardiovasc Imaging*. 2004;20:363–368.
- Paulson EK, Kliever MA, Hertzberg BS, O'Malley CM, Washington R, Carroll BA. Color Doppler sonography of groin complications following femoral artery catheterization. *AJR Am J Roentgenol*. 1995;165:439–444.
- Coughlin BF, Paushter DM. Peripheral pseudoaneurysms: evaluation with duplex US. *Radiology*. 1988;168:339–342.
- Dangas G, Mehran R, Duvvuri S, Ambrose JA, Sharma SK. Use of a pneumatic compression system (FemoStop) as a treatment option for femoral artery pseudoaneurysms after percutaneous cardiac procedures. *Cathet Cardiovasc Diagn*. 1996;39:138–142.



22. Pan M, Medina A, Suarez de Lezo J, Romero M, Hernandez E, Segura J, Melian F, Wanguemert F, Landin M, Benitez F, Amat M. Obliteration of femoral pseudoaneurysm complicating coronary intervention by direct puncture and permanent or removable coil insertion. *Am J Cardiol*. 1997;80:786–788.
23. Loose HW, Haslam PJ. The management of peripheral arterial aneurysms using percutaneous injection of fibrin adhesive. *Br J Radiol*. 1998;71:1255–1259.
24. Saad NE, Saad WE, Davies MG, Waldman DL, Fultz PJ, Rubens DJ. Pseudoaneurysms and the role of minimally invasive techniques in their management. *Radiographics*. 2005;25:S173–S189.
25. Lumsden AB, Miller JM, Kosinski AS, Allen RC, Dodson TF, Salam AA, Smith RB 3rd. A prospective evaluation of surgically treated groin complications following percutaneous cardiac procedures. *Am Surgeon*. 1994;60:132–137.
26. Fellmeth BD, Roberts AC, Bookstein JJ, Freischlag JA, Forsythe JR, Buckner NK, Hye RJ. Postangiographic femoral artery injuries: non-surgical repair with US-guided compression. *Radiology*. 1991;178:671–675.
27. Eisenberg L, Paulson EK, Kliever MA, Hudson MP, DeLong DM, Carroll BA. Sonographically guided compression repair of pseudoaneurysms: further experience from a single institution. *AJR Am J Roentgenol*. 1999;173:1567–1573.
28. Coley BD, Roberts AC, Fellmeth BD, Valji K, Bookstein JJ, Hye RJ. Postangiographic femoral artery pseudoaneurysms: further experience with US-guided compression repair. *Radiology*. 1995;194:307–311.
29. Steinkamp HJ, Werk M, Felix R. Treatment of postinterventional pseudoaneurysms by ultrasound-guided compression. *Invest Radiol*. 2000;35:186–192.
30. Dean SM, Olin JW, Piedmonte M, Grubb M, Young JR. Ultrasound-guided compression closure of postcatheterization pseudoaneurysms during concurrent anticoagulation: a review of seventy-seven patients. *J Vasc Surg*. 1996;23:28–34.
31. Cox GS, Young JR, Gray BR, Grubb MW, Hertzner NR. Ultrasound-guided compression repair of postcatheterization pseudoaneurysms: results of treatment in one hundred cases. *J Vasc Surg*. 1994;19:683–686.
32. La Perna L, Olin JW, Goines D, Childs MB, Ouriel K. Ultrasound-guided thrombin injection for the treatment of postcatheterization pseudoaneurysms. *Circulation*. 2000;102:2391–2395.
33. Taylor BS, Rhee RY, Muluk S, Trachtenberg J, Walters D, Steed DL, Makaroun MS. Thrombin injection versus compression of femoral artery pseudoaneurysms. *J Vasc Surg*. 1999;30:1052–1059.
34. Weinmann EE, Chayen D, Kobzantsev ZV, Zaretsky M, Bass A. Treatment of postcatheterisation false aneurysms: ultrasound-guided compression vs ultrasound-guided thrombin injection. *Eur J Vasc Endovasc Surg*. 2002;23:68–72.
35. Cope C, Zeit R. Coagulation of aneurysms by direct percutaneous thrombin injection. *Am J Roentgenol*. 1986;147:383–387.
36. Kang SS, Labropoulos N, Mansour MA, Michelini M, Filliung D, Baubly MP, Baker WH. Expanded indications for ultrasound-guided thrombin injection of pseudoaneurysms. *J Vasc Surg*. 2000;31:289–298.
37. Calton WC Jr, Franklin DP, Elmore JR, Han DC. Ultrasound-guided thrombin injection is a safe and durable treatment for femoral pseudoaneurysms. *Vasc Surg*. 2001;35:379–383.
38. Etemad-Rezai R, Peck DJ. Ultrasound-guided thrombin injection of femoral artery pseudoaneurysms. *Can Assoc Radiol J*. 2003;54:118–120.
39. Grewe PH, Mugge A, Germin A, Harrer E, Baberg H, Hanefeld C, Deneke T. Occlusion of pseudoaneurysms using human or bovine thrombin using contrast-enhanced ultrasound guidance. *Am J Cardiol*. 2004;93:1540–1542.
40. Kang SS. Thrombin injection for pseudoaneurysm occlusion: technical considerations [comment]. *J Endovasc Surg*. 2002;9:36–37.
41. Khouiry M, Rebecca A, Greene K, Rama K, Colaiuta E, Flynn L, Berg R. Duplex scanning-guided thrombin injection for the treatment of iatrogenic pseudoaneurysms. *J Vasc Surg*. 2002;35:517–521.
42. Krueger K, Zaehring M, Strohe D, Stuetzer H, Boecker J, Lackner K. Postcatheterization pseudoaneurysm: results of US-guided percutaneous thrombin injection in 240 patients. *Radiology*. 2005;236:1104–1110.
43. Kruger K, Zaehring M, Sohngen FD, Gossmann A, Schulte O, Feldmann C, Strohe D, Lackner K. Femoral pseudoaneurysms: management with percutaneous thrombin injections—success rates and effects on systemic coagulation. *Radiology*. 2003;226:452–458.
44. Maleux G, Hendrickx S, Vaninbrouckx J, Lacroix H, Thijs M, Desmet W, Nevelsteen A, Marchal G. Percutaneous injection of human thrombin to treat iatrogenic femoral pseudoaneurysms: short- and midterm ultrasound follow-up. *Eur Radiol*. 2003;13:209–212.
45. Mohler ER 3rd, Mitchell ME, Carpenter JP, Strandness DE Jr, Jaff MR, Beckman JA, Gerhard-Herman M. Therapeutic thrombin injection of pseudoaneurysms: a multicenter experience. *Vasc Med*. 2001;6:241–244.
46. Paulson EK, Nelson RC, Mayes CE, Sheafor DH, Sketch MH Jr, Kliever MA. Sonographically guided thrombin injection of iatrogenic femoral pseudoaneurysms: further experience of a single institution. *AJR Am J Roentgenol*. 2001;177:309–316.
47. Sheiman RG, Brophy DP. Treatment of iatrogenic femoral pseudoaneurysms with percutaneous thrombin injection: experience in 54 patients [see comment]. *Radiology*. 2001;219:123–127.
48. Sheiman RG, Mastromatteo M. Iatrogenic femoral pseudoaneurysms that are unresponsive to percutaneous thrombin injection: potential causes. *AJR Am J Roentgenol*. 2003;181:1301–1304.
49. Vazquez V, Reus M, Pinero A, Abellan D, Canteras M, Espinosa de Rueda M, Morales D, Parrilla P. Human thrombin for treatment of pseudoaneurysms: comparison of bovine and human thrombin sonogram-guided injection. *AJR Am J Roentgenol*. 2005;184:1665–1671.
50. Olsen DM, Rodriguez JA, Vranic M, Ramaiah V, Ravi R, Diethrich EB. A prospective study of ultrasound scan-guided thrombin injection of femoral pseudoaneurysm: a trend toward minimal medication. *J Vasc Surg*. 2002;36:779–782.
51. Reeder SB, Widlus DM, Lazinger M. Low-dose thrombin injection to treat iatrogenic femoral artery pseudoaneurysms. *AJR Am J Roentgenol*. 2001;177:595–598.
52. Paulson EK, Sheafor DH, Kliever MA, Nelson RC, Eisenberg LB, Sebastian MW, Sketch MH Jr. Treatment of iatrogenic femoral arterial pseudoaneurysms: comparison of US-guided thrombin injection with compression repair. *Radiology*. 2000;215:403–408.
53. Ferguson JD, Whatling PJ, Martin V, Walton J, Banning AP. Ultrasound guided percutaneous thrombin injection of iatrogenic femoral artery pseudoaneurysms after coronary angiography and intervention. *Heart*. 2001;85:e5.
54. Friedman SG, Pellerito JS, Scher L, Faust G, Burke B, Safa T. Ultrasound-guided thrombin injection is the treatment of choice for femoral pseudoaneurysms. *Arch Surg*. 2002;137:462–464.
55. Pope M, Johnston KW. Anaphylaxis after thrombin injection of a femoral pseudoaneurysm: recommendations for prevention. *J Vasc Surg*. 2000;32:190–191.
56. Sheldon PJ, Oglevie SB, Kaplan LA. Prolonged generalized urticarial reaction after percutaneous thrombin injection for treatment of a femoral artery pseudoaneurysm. *J Vasc Interv Radiol*. 2000;11:759–761.
57. Thalhammer C, Kirchherr AS, Uhlich F, Waigand J, Gross CM. Postcatheterization pseudoaneurysms and arteriovenous fistulas: repair with percutaneous implantation of endovascular covered stents. *Radiology*. 2000;214:127–131.
58. Kobeiter H, Lapeyre M, Becquemin JP, Mathieu D, Melliere D, Desgranges P. Percutaneous coil embolization of postcatheterization arterial femoral pseudoaneurysms. *J Vasc Surg*. 2002;36:127–131.

KEY WORDS: aneurysm ■ arteries ■ imaging ■ peripheral vascular disease ■ thrombin ■ thrombosis ■ vasculature

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