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Case Report

A Case of Puncture-Site Giant Pseudoaneurysm Following Recanalization Therapy for Acute Ischemic Stroke: Marked Growth and Rupture of a Femoral Artery Pseudoaneurysm

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Objective: We report a case of the marked growth and rupture of a giant femoral artery pseudoaneurysm at the puncture site that developed after recanalization therapy for acute basilar artery occlusion

Case Presentation: A 79-year-old woman developed acute ischemic stroke due to atherosclerotic basilar artery occlusion. Endovascular intervention was performed and recanalization of the affected vessel was achieved. However, she developed brainstem infarction and consciousness disturbance persisted. The femoral access site was treated using a vascular closure device at the end of the procedure. A right femoral artery pseudoaneurysm of approximately 5 cm in size was found 2 weeks after onset during the examination for deep venous thrombosis with right lower extremity edema. Manual compression did not achieve thrombotic occlusion of the aneurysm due to obesity and leg edema. Considering the severe neurological status of the patient, the pseudoaneurysm was followed up without surgical treatment. Dual antiplatelet therapy and direct oral anticoagulant agents were administered. Four weeks after onset, the pseudoaneurysm presented rapid growth, and on the 35th day after onset, it exceeded 15 cm in size and ruptured, causing hemorrhagic shock with massive femoral hematoma. Pseudoaneurysm resection and hematoma removal were performed surgically, and the patient recovered. However, improvement of neurological manifestations was poor and the modified Rankin Scale at 90 days after onset was 5.

Conclusion: A case of giant femoral artery pseudoaneurysm following recanalization therapy for acute ischemic stroke was reported. Pseudoaneurysms at the puncture site can rupture after significant growth. Curative treatment is required without delay.

Keywords ▶ pseudoaneurysm, femoral artery, puncture-site complication, acute ischemic stroke, recanalization therapy

Introduction

Mechanical thrombectomy for acute large vessel occlusion was demonstrated to be effective and has become widely performed, increasing the number of treated cases. Antiplatelet agents/anticoagulant agents are used as measures against the perioperative thromboembolic events of thrombectomy. Thrombectomy may also be applied after intravenous thrombolysis with recombinant tissue plasminogen activator (t-PA). As intensive antithrombotic therapy is required as described above, several complications of puncture-site vessels are of concern. Puncture-site bleeding, retroperitoneal hemorrhage, arterial occlusion, and arteriovenous fistula have been reported as complications involving puncture-site vessels after catheter intervention,[1,2] including pseudoaneurysms developing in the puncture-site vessel due to insufficient hemostasis.[3–5] We report a patient with a giant femoral artery pseudoaneurysm at the puncture site that developed after recanalization therapy for acute basilar artery occlusion and ruptured after marked enlargement.

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Case Presentation

Course of illness

The patient was a 79-year-old woman. She had past medical histories of hypertension, diabetes, and hyperlipidemia. The body weight was 64 kg, height was 151 cm, and BMI was 28.1. She had common cold symptoms from 3 days before visiting the hospital, and anorexia, general malaise, and nausea developed one day earlier. As dysarthria, left paresis, and right facial palsy were noted upon wakening, the patient was transported to our hospital by ambulance. Her condition upon arrival at the emergency service was Japan Coma Scale (JCS) 1, but it aggravated during clinical examination and consciousness deteriorated to JCS200. Ataxic respiration was observed and quadriplegia developed. Subsequently, imaging examination was performed.

Imaging findings

No finding suggesting hemorrhage was noted on head CT. On diffusion-weighted MRI of the head, high-intensity areas were present on the right side of the pons and in the left cerebellar hemisphere. On head MRA, basilar artery occlusion was noted. The basilar artery tip was visualized through the right posterior communicating artery (**Fig. 1A** and **1B**). The patient was diagnosed with progressively aggravated acute basilar arterial occlusion. t-PA was not used because the accurate first onset time was unclear. Emergency endovascular recanalization therapy was performed.

Neuroendovascular intervention

Neuroendovascular intervention was performed under local anesthesia. The arterial puncture time was 93 minutes after the condition deteriorated. A 9Fr sheath was inserted into the right femoral artery. The puncture site of the femoral artery from the skin surface was deep because of obesity, but arterial puncture was possible. To use the 9Fr Optimo (Tokai Medical Products, Aichi, Japan) as a guiding catheter, approaches to the bilateral vertebral arteries were tried, but access to either vertebral artery was difficult due to vascular tortuosity at the aortic arch and origin of the vertebral arteries. Thus, it was changed to an approach through the right brachial artery. A 6Fr sheath was inserted into the right brachial artery and guided to the right vertebral artery using 6Fr Cerulean (Medikit, Tokyo, Japan) as a guiding catheter. A region with defective contrast was noted in the vertebral artery union on the right vertebral arteriography, and blood flow peripheral to this region was delayed (**Fig. 2A**). Recanalization therapy was performed using a Trevo XP 4 mm-20 mm (Stryker Neurovascular, Fremont, CA, USA). A stenosis lesion considered an arteriosclerotic lesion remained, but delayed blood flow of the basilar artery improved and thrombolysis in cerebral infarction (TICI) 2b recanalization was acquired (**Fig. 2B**). The time from puncture to recanalization was 92 minutes. Hemostasis of the femoral arterial puncture site was performed using an 8Fr AngioSeal STS Plus (Terumo, Tokyo, Japan). On femoral arteriography through the sheath, the sheath insertion site was located at the level of the inferior margin of the femoral head and it was confirmed to be slightly proximal to the bifurcation, not to the superficial femoral artery (**Fig. 2C**). There was no procedural problem with AngioSeal for the hemostasis device. Subcutaneous fat was thick, but collagen sponge insertion was possible using a tamper tube and the tamping marker was confirmed. The brachial arterial puncture site was managed by manual compression after the natural reversal of heparin.

Course after neuroendovascular intervention

After intervention, the condition was evaluated as acute basilar arterial occlusion with a background arteriosclerotic stenosis lesion, and argatroban drip infusion and tube administration of aspirin and clopidogrel were performed as acute-phase antithrombotic therapy. Dual antiplatelet therapy with aspirin and clopidogrel was continued from 1 week after onset. On diffusion-weighted MRI performed on the day following treatment, a high-intensity area was observed in the midbrain and the pons. In addition, small scattered high-intensity areas were observed in the left thalamus, left cerebellar hemisphere, and cerebellum. On MRA, the basilar artery was recanalized and stenosis was noted at the union of vertebral arteries (**Fig. 1C** and **1D**). Clinically, the neurological manifestations did not improve after endovascular treatment, and her condition of brainstem infarction-induced symptoms: consciousness disturbance with JCS200 and quadriplegia remained unchanged.

Clinical findings suggesting a pseudoaneurysm at the femoral arterial puncture site, such as subcutaneous hematoma and pulsatile mass, were unclear. The D-dimer level was 65 μg/mL, being markedly high, on blood testing at 2 weeks after onset and edema was noted in the right lower limb, for which lower limb vascular ultrasonography was performed. A pseudoaneurysm with a size of 4.8 cm and deep venous thrombosis were observed at the right femoral arterial puncture site (**Fig. 3A** and **3B**). It was considered to be deep venous thrombosis accompanying compression of the femoral vein by the pseudoaneurysm. In addition,

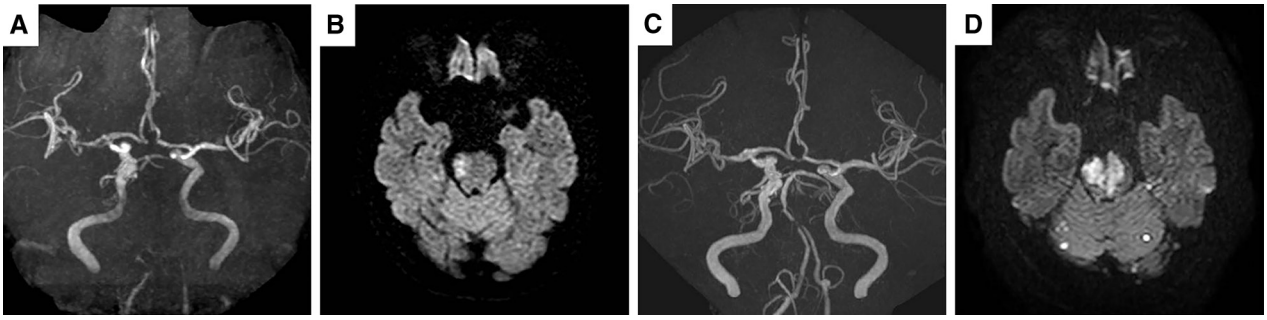

Fig. 1 (**A**) MRA at onset: Bilateral vertebral arteries over the trunk of the basilar artery were not visualized. The basilar artery tip was visualized through the right posterior communicating artery. (**B**) Diffusion-weighted MRI at onset: A high-intensity area was present on the right side of the midbrain. (**C**) MRA on the day following onset after recanalization therapy: Severe stenosis was noted in the union of vertebral arteries, but recanalization of the basilar artery was acquired. (**D**) Diffusion-weighted MRI on the day following onset: A transverse high-intensity area was noted in the brainstem.

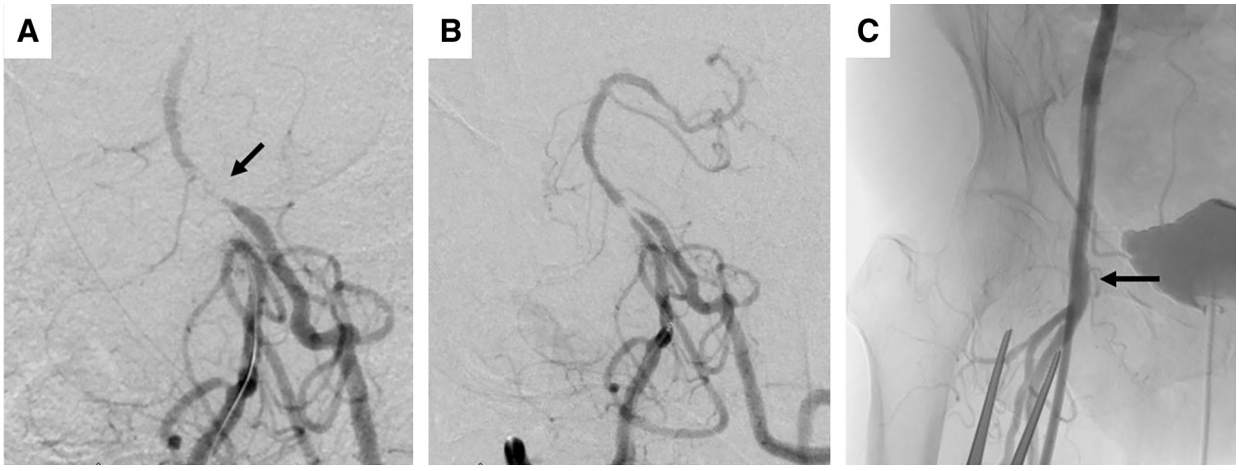

Fig. 2 Images of neuroendovascular intervention (recanalization therapy). (**A**) Right vertebral arteriography: A contrast-defective region suggesting severe stenosis and thrombus was present in the union of vertebral arteries over the proximal basilar artery, delaying antegrade blood flow. (**B**) Vertebral arteriography after thrombectomy using a stent retriever: A severe stenotic lesion remained in the union of vertebral arteries, but antegrade blood flow of the basilar artery improved. (**C**) Right femoral arteriography through the sheath: The arterial puncture site was located at the level of the inferior margin of the femoral head (arrow), suggesting a site slightly proximal to the femoral arterial bifurcation.

mild pulmonary arterial embolism was observed on contrast-enhanced chest CT.

For the pseudoaneurysm, treatment by manual compression was tried, but it was difficult because of obesity and lower limb edema. Surgical treatment was considered, but invasive treatment was not performed and course observation was selected in consideration of the current general condition and neurological outcome of the patient and family's request. For pulmonary arterial thrombosis and deep venous thrombosis, anticoagulant therapy was initiated. After heparin drip infusion, the drug was changed to apixaban. The patient's general condition stabilized thereafter and neurological manifestations slightly improved. At 3 weeks after onset, the patient became able to partially communicate through opening and closing the eyes, being in a condition close to locked-in syndrome.

The pseudoaneurysm located at the right femoral arterial puncture site slightly enlarged and then markedly increased in size 4 weeks after onset. It reached 10.6 cm at 5 weeks after onset (**Fig. 3C** and **3D**). Surgery for the pseudoaneurysm was planned, but her blood pressure suddenly decreased, leading to a shock state 37 days after onset. The right thigh was markedly swollen and tense. The hemoglobin level was reduced to 5.7 g/dL. Features of a large hematoma were noted in the right thigh on CT and the pseudoaneurysm size was larger than 15 cm (**Fig. 4**), suggesting ruptured pseudoaneurysm-induced thigh hematoma and simultaneous hemorrhagic shock. There was no intraabdominal bleeding or retroperitoneal hemorrhage. Surgery was performed after stabilizing the vital signs by fluid replacement and blood transfusion.

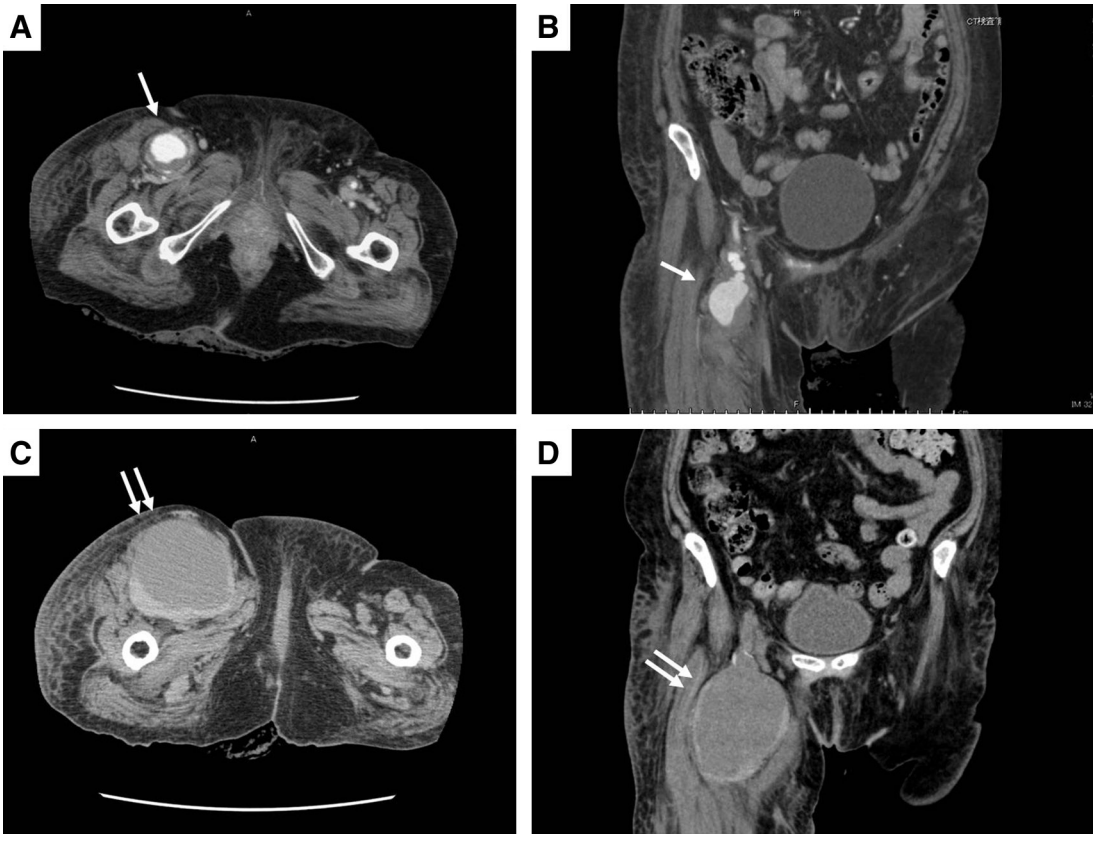

Fig. 3 Horizontal view (**A**) and coronal view (**B**) on contrast-enhanced CT of the lower limb at 2 weeks after onset: Pseudoaneurysm with a maximum size of approximately 5 cm (arrow) was present at the arterial puncture site of the right thigh. Horizontal view (**C**) and coronal view (**D**) on plain CT of the lower limb at 5 weeks after onset: The pseudoaneurysm enlarged and its maximum diameter increased to approximately 11 cm (double arrow).

Surgery for the femoral arterial puncture-site pseudoaneurysm

Emergency surgery for the femoral arterial puncture-site pseudoaneurysm (resection of the pseudoaneurysm, removal of hematoma, and vascular repair) was performed under general anesthesia. The right inguinal region was incised, the common femoral artery right below the inguinal ligament was secured, the pseudoaneurysm was incised under temporary clamp of the common femoral artery, and the hematoma was removed, after which back flow from the perforated site was confirmed. The region around the perforated site was temporarily closed with sutures and a patch, and clamp of the common femoral artery was released. The hematoma filling the right thigh was removed by curettage within the visible range as much as possible. No finding suggesting infectious aneurysm was observed. The femoral artery around the perforated site was then dissected, and the superficial and deep femoral arteries were exposed. The common femoral artery was clamped again, and the bifurcation from the common femoral artery to the superficial and deep femoral arteries was observed. The vascular perforated site was located almost right above the superficial/deep femoral arterial bifurcation and an approximately 4-mm hole was present. Adhesion considered to be formed by AngioSeal was observed around the perforated site. The perforated site was closed by sutures (polyvinylidene thread) and clamp of the common femoral artery was released. Surgery was completed without intraoperative complications. The patient recovered from a fatal condition.

Course after surgery for the pseudoaneurysm

After surgery, neither recurrence of puncture-site aneurysm nor circulation disorder of the lower extremity developed, but the wound on the right thigh remained after surgery and skin grafting was required 3 months after thigh repair surgery. Improvement of neurological manifestations was poor and the modified Rankin Scale at 90 days after onset was 5.

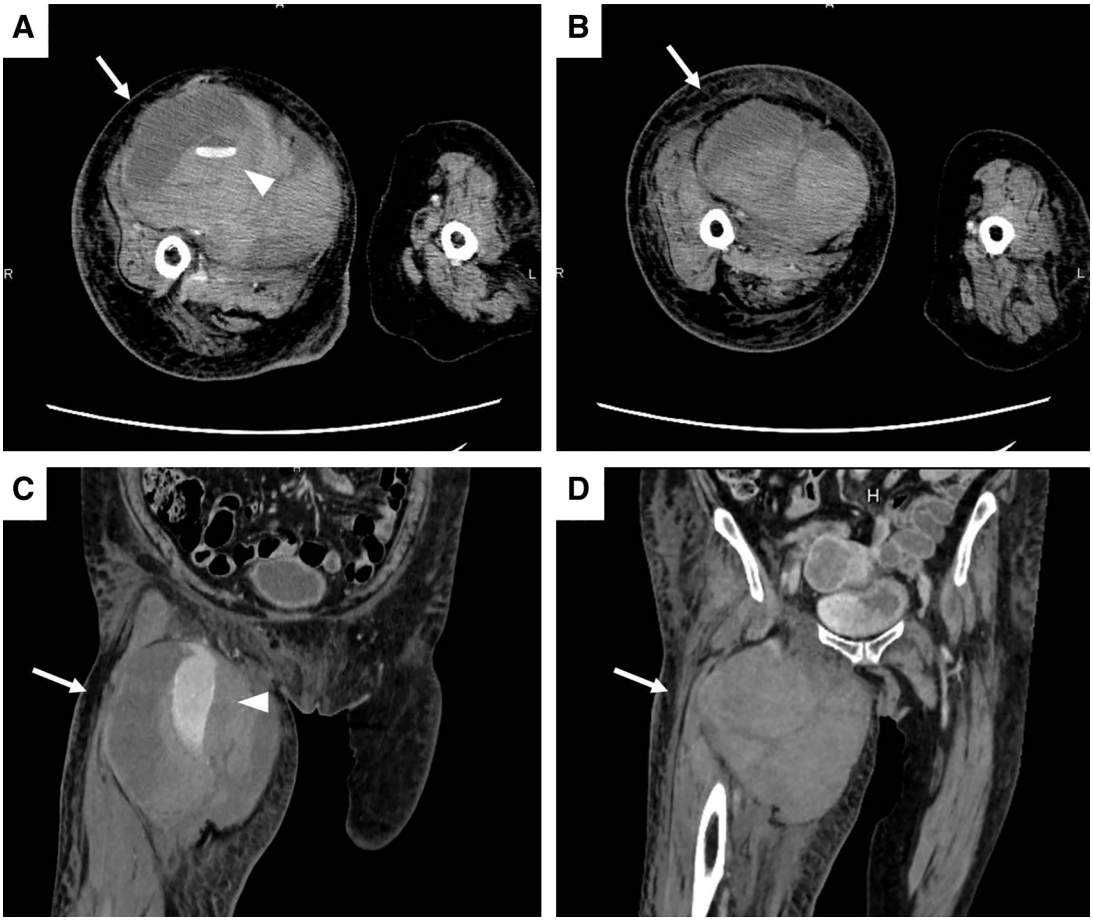

Fig. 4 Horizontal view (**A**, **B**) and coronal view (**C**, **D**) on contrast-enhanced CT of the lower limb when the pseudoaneurysm ruptured at 35 days after onset: A giant pseudoaneurysm accompanied by right thigh hematoma was observed (arrow). Leakage of contrast medium was noted in a part of the pseudoaneurysm (arrowhead).

Discussion

Puncture-site complications are one of the complications of catheter intervention. Puncture-site complications are reported mainly from the cardiology and vascular surgery fields. On the other hand, they have been recently reported from the neuroendovascular intervention following the spread of thrombectomy for large vessel occlusion and use of stents for cerebral aneurysm treatment.[1,2,6] In a study by Shapiro et al. in which 339 cases of 7 prospective studies on mechanical thrombectomy were reviewed, the incidence of all puncture-site complications was 4.59% and that of major puncture-site complications was 1.67%.[2] In a review of studies on neuroendovascular treatment by Oneissi et al. (16 randomized control trials [RCTs] and 17 non-RCTs), the overall incidence of puncture-site complications was 5.13% in RCT and 2.78% in non-RCT. The incidence of pseudoaneurysm was 0.23%–2.04% (mean: 0.61%) in RCT and 0.03–3.23% (mean: 0.19%) in non-RCT.[1] Among puncture-site complications after endovascular treatment, pseudoaneurysm is the second most frequent complication following puncture-site hematoma and bleeding.

Pseudoaneurysms may cause rupture-induced massive hemorrhage[7] or serve as a source of thrombus inducing peripheral vascular occlusion. In this patient, the ruptured pseudoaneurysm caused hemorrhagic shock. Pseudoaneurysms are large and grow, possibly causing local symptoms, peripheral neuropathy, and lower limb venous congestion as a space-occupying lesion.[8] Deep venous thrombosis may be caused by puncture-site subcutaneous hematoma alone,[9] but in this patient, femoral vein compression by pseudoaneurysm caused lower limb edema and deep venous thrombosis. In addition, venous thrombosis-induced pulmonary arterial embolism was noted.

Puncture-site pseudoaneurysms are discovered upon the development of clinical symptoms, such as pulsatile mass, bruit, and pain, in many cases. In a series reported by Koza et al., puncture-site pseudoaneurysm was diagnosed

8.3 days (2–21 days) after intervention on average.[10] In our patient, the lesion was discovered upon the development of deep venous thrombosis 2 weeks after onset because observation of the puncture-site vessel was difficult due to obesity and there were no complaints of puncture-site symptoms from the patient due to consciousness disturbance. For screening of pseudoaneurysms, evaluation by vascular ultrasonography is desirable. When hematoma is present at the puncture site or observation is difficult due to obesity, it is better to confirm the puncture-site vessel by echo, which can be easily performed at the bedside. Hoke et al.[11] reported that an increased D-dimer level (D-dimer >0.67 μg/mL) and a decrease in the platelet count (<200000/L) are useful serological markers of puncture-site pseudoaneurysm. In the present patient, no decrease in the platelet count was noted at the time of discovery, whereas the D-dimer level was being markedly increased, but this may have been due to venous thrombosis.

Risk factors of pseudoaneurysms include the following: patient-derived factors include advanced age, female, obesity, hypertension, diabetes, peripheral vascular disease, coronary arterial disease, hemodialysis, and reduction of platelets. Procedural and treatment-derived factors include perioperative anticoagulant therapy and antiplatelet therapy, left femoral artery puncture, use of a sheath with a large diameter, puncture at a low level (puncture at a site peripheral to the common femoral artery), puncture at a high level, and insufficient hemostasis.[4,5] This patient had many risk factors, such as advanced age, female , obesity, hypertension, diabetes, anticoagulant therapy + antiplatelet therapy, sheath with a large diameter, and puncture at a low level.

To acquire hemostasis of the puncture site by manual compression in patients undergoing perioperative antithrombotic therapy, such as thrombectomy, a considerable amount of time is necessary. Puncture-site hemostasis can be acquired within a short time using a hemostatic device such as AngioSeal. It also shortens the bed rest and hospitalization periods, being advantageous for not only medical care workers but also patients.[12,13] In this patient, hemostasis was applied to the 9Fr sheath insertion site using AngioSeal for the 8Fr sheath. Janssen et al.[14] reported that complications did not increase even though 8Fr AngioSeal was used for hemostasis of a 9Fr or larger sheath insertion site; however, the use of a device with a size smaller than the sheath size may have played a role in the development of the pseudoaneurysm. In addition, this patient was not highly obese and the tamper tube length reached the lesion, but the collagen sponge may not have achieved compression bonding to the hole of the vascular puncture site due to the subcutaneous fat thickness. As observation was difficult due to the hematoma and crushed soft tissue, and the time point was during the collagen absorption period, it was difficult to judge whether the collagen sponge of AngioSeal was placed at an appropriate position based on the intraoperative findings. The location of the perforated site and puncture at a low level may have been causes. On the other hand, no signs of infection were noted around the puncture site nor were there signs suggesting infectious aneurysm. However, puncture-site complications develop at a specific rate even though appropriate cases were selected and AngioSeal was correctly used,[12] suggesting that early discovery and appropriate treatment are important for puncture-site complications, including pseudoaneurysms.

The characteristic of this case was the marked enlargement of the puncture-site pseudoaneurysm to a size larger than 15 cm. The first cause was the hesitation in performing curative surgery for the pseudoaneurysm because severe disturbance of consciousness remained due to brainstem infarction and the neurological outcome was poor. Moreover, deep vein thrombosis and pulmonary embolism were observed, for which an anticoagulant agent was administered in addition to two antiplatelet agents, being another possible cause. Furthermore, lower limb edema caused by pseudoaneurysm compression-induced venous circulation disorder may have influenced pseudoaneurysm enlargement.

Small puncture-site pseudoaneurysms are asymptomatic, but those with a large size often enlarge and may cause problems in addition to rupture, as described above, requiring therapeutic management. Stone et al.[5] reported that pseudoaneurysms with a size of 3 cm or smaller are relatively likely to spontaneously thrombose and they recommended following the course for 1–2 weeks by ultrasonography. On the other hand, treatment is necessary for large pseudoaneurysms and those continuing to enlarge, being symptomatic and not thrombosing during course observation.[5] Pseudoaneurysm treatment methods include echo-guided compression, echo-guided percutaneous thrombin injection, and surgical repair.[3–5] Echo-guided compression and echo-guided percutaneous thrombin injection have been reported to be minimally invasive and capable of achieving a high success rate.[3–5] In our patient, echo-guided manual compression was tried, but occlusion of the pseudoaneurysm was difficult because of obesity and crural edema. Percutaneous thrombin injection was not performed because of a lack of experience, but it should have been tried. An aneurysm-thrombosing echo-guided compression method by injecting

saline into the pseudoaneurysm neck has been proposed[15] and it will be investigated. When symptoms are caused by compression by a markedly enlarged hematoma, surgical vascular repair including hematoma removal is necessary. Skin necrosis requiring repair and cases accompanied by local infection are also indications of surgical treatment. However, the complication rate of surgical treatment for such pseudoaneurysms is high.[10,16,17] The main complications of surgical treatment include infection and suture failure. This patient exhibited wound suture failure and required repair surgery. Surgical treatment becomes difficult as the size of the pseudoaneurysm increases, but curative treatment should be investigated before it becomes large. The pseudoaneurysm was approximately 5 cm in size at the time of discovery in this patient, for which urgent treatment may have been desirable.

Conclusion

A patient with a femoral arterial giant pseudoaneurysm that developed after recanalization therapy for acute ischemic stroke was reported. As femoral arterial puncture-site pseudoaneurysms may rupture due to marked enlargement, they should be treated without delay.

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Disclosure Statement

The authors declare no conflict of interest.

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