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Case Report

Fatal Femoral Access-Site Pseudoaneurysm Following Multimodal Endovascular Stroke Reperfusion Therapy: A Case Report

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ABSTRACT

Mechanical thrombectomy is an established treatment for acute ischemic stroke due to large-vessel occlusion, and transfemoral access is generally safe. However, access-site complications, though uncommon, can be severe. We report a fatal femoral access-site pseudoaneurysm occurring after multimodal endovascular stroke reperfusion therapy. A 69-year-old woman presented with acute left hemiparesis and was found to have a right M1 occlusion. She received intravenous tenecteplase followed by emergent mechanical thrombectomy via right femoral access, with complete reperfusion achieved. The procedure also required carotid angioplasty, closure-device deployment, and periprocedural eptifibatide. Within hours, she developed an enlarging groin hematoma and early livedo reticularis of the thigh. Duplex ultrasound revealed a large femoral pseudoaneurysm, later localized angiographically to the profunda femoris–superficial femoral artery bifurcation. Despite urgent endovascular exclusion with a covered stent graft, she arrived in hemorrhagic shock and progressed to profound metabolic acidosis, multiorgan failure, and death from hypovolemic shock. This case highlights how the combination of complex aortoiliac anatomy, large-bore transfemoral access, closure-device use, and potent antithrombotic exposure can predispose to rapid pseudoaneurysm expansion and severe hemorrhage. The early appearance of livedo reticularis represents a potentially important cutaneous warning sign of evolving vascular injury. Although limited to a single case, these observations underscore the need for careful access planning in anatomically challenging patients and prompt evaluation of early groin changes or unexplained clinical decline following endovascular stroke therapy.

1. Introduction

Mechanical thrombectomy is the standard treatment for acute ischemic stroke due to large vessel occlusion and is associated with high rates of reperfusion and improved functional outcomes [1]. As the use of endovascular therapy expands, increasing attention has been directed toward complications related not only to thrombectomy itself but to the broader spectrum of multimodal stroke interventions. The common femoral artery remains the most frequently used vascular access-site for neuroendovascular procedures because of its anatomical accessibility and ability to accommodate large bore catheters [2, 3]. Although the overall safety profile of transfemoral access is favorable, access site complications occur in 2 – 6% of patients undergoing neuroendovascular procedures [2, 4].

Common femoral artery pseudoaneurysm is a clinically significant access site complication that can lead to hemorrhage, limb ischemia, and hemodynamic instability [2, 5]. Pseudoaneurysm formation results from inadequate hemostasis at the arterial puncture site, allowing blood to dissect into surrounding tissue while maintaining communication with the arterial lumen [5]. The reported incidence of femoral pseudoaneurysm varies by procedure type. After diagnostic angiography, rates range from 0.05 – 2.2%. Following therapeutic endovascular procedures, particularly those requiring larger sheaths or prolonged catheter manipulation, the incidence increases to 2 – 6% [6, 7]. Data specific to neurointerventional stroke thrombectomy are more limited, but available series suggest that access site complications remain uncommon and generally fall within the broader range reported for large bore transfemoral interventions.

With the growing use of multimodal endovascular stroke reperfusion strategies, including intravenous thrombolysis, mechanical thrombectomy, carotid angioplasty or stenting, vascular closure devices, and adjunctive antiplatelet therapy, the cumulative bleeding risk warrants careful consideration. We present a case of a fatal femoral access site pseudoaneurysm following multimodal endovascular stroke reperfusion therapy.

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Table 1: Chronological Timeline of Clinical Events

Event	Date	Clock Time
Symptom onset	7/20	19:30
ED arrival	7/20	20:35
Initial CT/CTA	7/20	20:43
Tenecteplase administered	7/20	21:11
Right femoral artery puncture	7/20	22:26
First device pass	7/20	22:52
Complete reperfusion (TICI 3)	7/20	22:56
Sheath removal	7/20	23:44
Angio-Seal deployment	7/20	23:55
First documented groin swelling	7/21	07:30
First skin discoloration / livedo reticularis	7/21	07:30
Duplex ultrasound confirming pseudoaneurysm	7/21	18:04
Vascular surgery consult	7/21	18:25
Transfer to OR	7/21	22:30
Start of transfusion	7/21	22:45
Stent-graft deployment (Viabahn)	7/22	02:30
Time of death	7/22	12:15

ED, Emergency Department; CT, Computed Tomography; CTA, Computed Tomography Angiography; TICI, Thrombolysis in Cerebral Infarction; OR, Operating Room.

2. Case Presentation

A 69 year old woman with a remote history of deep vein thrombosis and no routine medical care presented to the emergency department with acute onset left leg weakness, left lower facial droop, and slurred speech. Symptoms began approximately one hour prior to arrival. Her husband reported that she initially retained some movement in the left arm, but by the time of evaluation she exhibited complete left upper extremity paralysis. She denied fever, chest pain, dyspnea, or constitutional symptoms. She was a daily smoker and was not taking anticoagulants or antiplatelet agents.

On arrival, she was hypertensive to 239/130 mmHg, with HR 86, RR 18, oxygen saturation 96% on room air, and temperature 98.4°F. Neurological examination revealed an NIHSS of 5. Laboratory studies showed PT 13.5 sec, INR 1.07, PTT 28.7 sec, BUN/Cr 40/1.57 mg/dL, and eGFR 33 mL/min/1.73m². CT angiography of the head and neck demonstrated a right M1 occlusion **(Figure 1)** and 50% narrowing of the proximal right ICA. Thirty minutes after arrival, she received intravenous tenecteplase 21 mg once. Although her initial NIHSS was 5, the patient's deficits were functionally disabling, including complete left upper extremity paralysis, left leg weakness impairing ambulation, dysarthria, and facial droop. She also demonstrated rapid neurological progression, with NIHSS worsening to 14 after tenecteplase administration. These clinical features provided a clear rationale for escalation to mechanical thrombectomy and adjunctive carotid angioplasty, with the anticipated neurological benefit judged to outweigh procedural risk. Chronological timeline of clinical events are shown in **(Table 1)**.

One hour after thrombolysis, she underwent emergent mechanical thrombectomy under general anesthesia. Right common femoral artery access was obtained under real time ultrasound guidance with a single puncture attempt above the femoral bifurcation, using a micropuncture system that was then upsized to an 8 French arterial sheath. Mechanical thrombectomy was performed using an aspiration catheter system with adjunctive techniques. No intraprocedural heparin was administered due to recent systemic thrombolysis. Diagnostic angiography confirmed near occlusive cervical ICA stenosis (≈ 99%) with a focal area of non visualization of contrast and markedly reduced antegrade flow, as well as right M1 occlusion **(Figs. 2 and 3)**. This discrepancy was attributed to differences in imaging modality and dynamic flow conditions in the hyperacute stroke setting, rather than interval disease progression. The cervical ICA lesion was considered hemodynamically significant and flow limiting, contributing to impaired distal perfusion in the setting of an intracranial large vessel occlusion. In conjunction with the patient's rapidly worsening neurological deficits, this was treated as a tandem lesion stroke. Following successful reperfusion (TICI 3), the patient underwent balloon angioplasty of the right internal carotid artery. Because of residual stenosis and the risk of acute thrombosis after angioplasty, she was started on eptifibatide at 0.5 mcg/kg/min for 2 hours, without a bolus and without additional intraprocedural antiplatelet loading. Approximately 30% residual stenosis remained after angioplasty. She was extubated post procedure, awake, following commands, and not receiving sedative medications.

Approximately seven and a half hours later, she developed a right groin hematoma with violaceous, reticulated discoloration of the ipsilateral thigh consistent with livedo reticularis **(Figure 4)**. Serial vascular examinations were performed approximately every hour. Bilateral femoral pulses were palpable, and bilateral popliteal Doppler signals were present. On the right, a posterior tibial Doppler signal and pulse were appreciated, but a dorsalis pedis pulse or Doppler signal could not be obtained. Both lower extremities were cool to touch with cool feet bilaterally. Sensory examination remained intact, and no tense compartments were noted. These findings raised concern for evolving access site vascular injury rather than acute limb ischemia or compartment syndrome. At the time of initial recognition, the patient remained hemodynamically stable without hypotension or tachycardia, and distal perfusion appeared largely preserved based on serial pulse and Doppler examinations. The findings were therefore initially interpreted as a localized access site hematoma without clear evidence of active arterial bleeding or acute limb ischemia. Given this clinical stability, close bedside monitoring with serial examinations was pursued rather than immediate intervention. At the time of hematoma recognition, the patient had completed the planned 2 hour eptifibatide infusion earlier in the post procedural period, and no further antiplatelet infusion was ongoing. The access site was marked and monitored for interval expansion. Manual compression was deferred due to concern for potential retroperitoneal extension and the absence of a discrete, compressible puncture site on examination. The patient remained on continuous hemodynamic monitoring with frequent reassessment of vital signs, serial vascular examinations, and trending of hemoglobin and coagulation parameters. Given preserved hemodynamics without hypotension or tachycardia, transfusion was initially deferred. Progressive enlargement of the hematoma, declining hemoglobin levels, and evolving metabolic abnormalities prompted duplex ultrasonography. Following confirmation of a large pseudoaneurysm with active flow, vascular surgery consultation was obtained. During continued observation, objective clinical deterioration became increasingly apparent. Serial laboratory evaluation demonstrated progressive anemia and coagulopathy, followed by worsening metabolic acidosis with elevated serum lactate and declining renal function. Although the patient initially

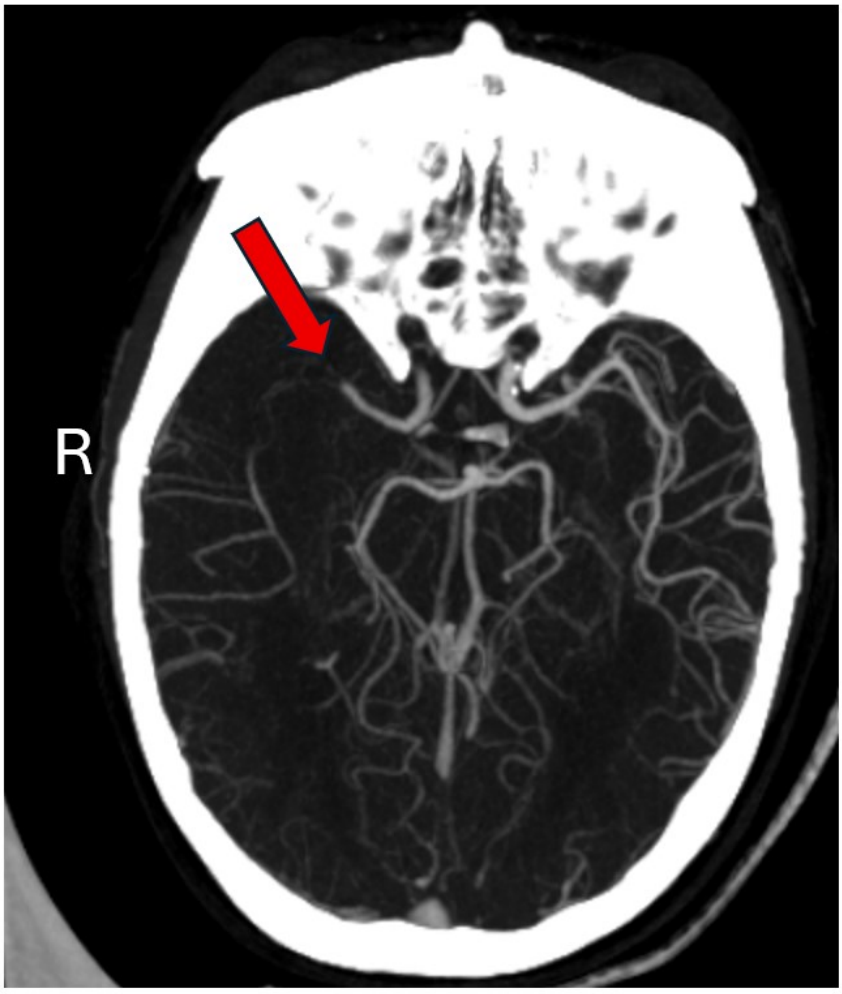

Figure 1: CT angiography of the head and neck showing M1 occlusion (Arrow).

Table 2: Serial hematologic and coagulation parameters during the period of clinical deterioration

Date	Time	Hgb (g/dL)	Platelets (k/μL)	PT (sec)	INR	aPTT (sec)	Fibrinogen (mg/dL)
7/20	20:53	13.0	251	13.5	1.07	28.9	-
7/21	01:48	11.4	211	16.9	1.43	45.2	-
7/21	13:47	10.9	161	-	-	-	-
7/21	20:00	8.3	127	-	-	-	-
7/22	00:00	6.5	133	26.9	2.59	195.3	136
7/22	03:00	5.7	150	-	-	-	-
7/22	05:55	6.9	147	37.7	3.98	43.9	-
7/22	11:30	7.3	66	-	-	-	-

RR, Reference Ranges.

remained normotensive, she subsequently developed hypotension with signs of impaired end organ perfusion, including oliguria. These objective findings, in conjunction with ongoing hematoma expansion, supported the diagnosis of evolving hemorrhagic shock and prompted escalation to definitive vascular intervention and blood product resuscitation.

Serial limb examinations were continued throughout this period. The absent right dorsalis pedis Doppler signal was not known to be present prior to the procedure and was considered a new finding, while posterior tibial and popliteal signals remained consistently detectable. There was no progression to pulselessness, worsening sensory deficit, motor weakness, or development of tense compartments on repeated examinations. Although the differential

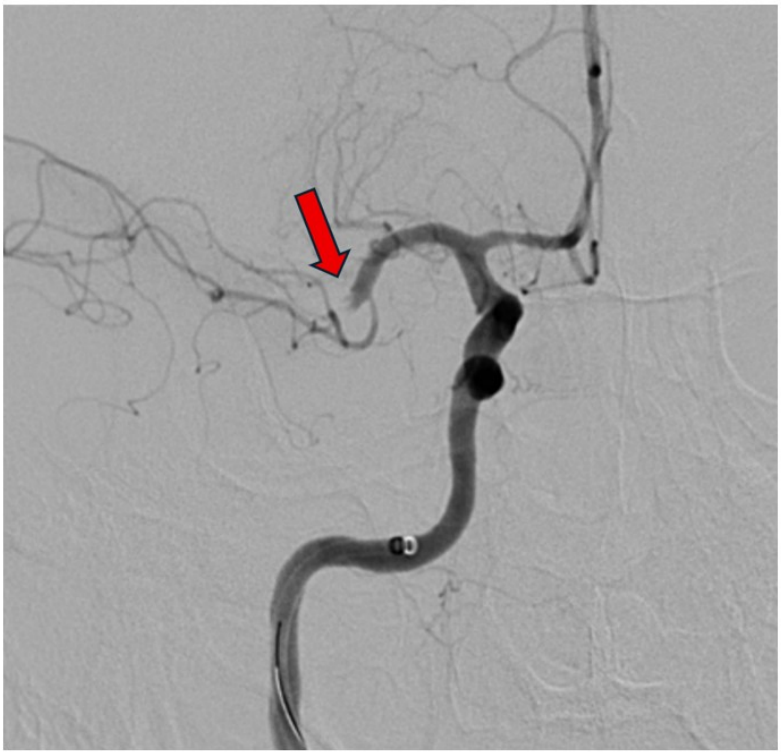

Figure 2: Right M1 occlusion (Arrow) during angiography.

Table 3: Hemodynamic trends during the evolution of hemorrhagic shock

Date	Time	BP (mmHg)	MAP
7/21	19:30	81/58	66
7/21	20:00	137/78	97
7/22	00:15	189/89	122
7/22	00:30	153/72	99
7/22	01:05	142/67	92
7/22	01:30	161/75	103
7/22	03:00	136/63	87
7/22	05:00	110/59	76
7/22	09:00	108/59	75
7/22	10:00	96/56	69
7/22	11:00	87/55	66

BP, blood pressure; MAP, mean arterial pressure.

diagnosis included acute limb ischemia, distal embolization, and compartment syndrome, the persistence of proximal arterial signals, intact sensation, and lack of compartment firmness reduced concern for these entities and supported evolving access site vascular injury as the primary process.

Her neurological status worsened, with NIHSS increasing from 14 to 21. A repeat non contrast head CT obtained the following morning demonstrated an evolving right MCA territory infarct without definitive evidence of hemorrhagic transformation. No repeat CTA or MRI was performed due to rapidly worsening hemodynamic instability. The neurological deterioration occurred in close temporal association with the expanding access site hematoma and evolving hemorrhagic shock, and in the absence of intracranial hemorrhage or sedative confounders, the decline was attributed to systemic hypoperfusion from the femoral pseudoaneurysm.

Duplex ultrasound demonstrated a large right groin pseudoaneurysm measuring 6.5 × 3.6 cm with active flow **(Figure 5)**. The right external iliac artery, common femoral artery, profunda femoris artery, and superficial femoral artery were all patent. Although duplex characterized the lesion as arising from the common femoral region, the precise site of arterial wall disruption could not be definitively localized due to hematoma and pseudoaneurysm mass effect. She was urgently taken for vascular intervention. On arrival, she was in hemorrhagic shock with mottled extremities, no urine output, and profound hypovolemia. She was intubated, resuscitated with 6 units of packed red blood cells, 6 units of fresh frozen plasma, and 6 units of platelets, and required vasopressor support. Serial hematologic and coagulation parameters during the period of clinical deterioration are shown in **(Table 2)**, and Hemodynamic trends during the evolution of hemorrhagic shock are shown in **(Table 3)**. Intraoperative angiography demonstrated severe iliac tortuosity and active contrast extravasation at the bifurcation of the profunda femoris and superficial femoral arteries, clarifying the exact site of arterial injury. A Viabahn 8 × 10 cm covered stent graft was deployed across the bifurcation with successful endovascular exclusion of the pseudoaneurysm.

Despite restoration of arterial integrity, the patient remained profoundly acidotic, hypothermic, and hemodynamically unstable. She was transferred to the surgical ICU in critical condition with multisystem organ failure. Over the next several hours, she continued to deteriorate despite maximal resuscitative efforts. Approximately twelve hours after surgery, she suffered cardiopulmonary arrest and

Table 4: Metabolic and acid–base trends, including lactic acid

Date	Time	Lactate (mmol/L)	pH	HCO_3 (mmol/L)	CO_2 (mmol/L)	Creatinine (mg/dL)
7/20	20:53	-	-	-	-	1.57
7/21	01:48	-	-	-	-	1.54
7/21	22:00	-	7.02	8.8	34	-
7/22	00:00	15.0	7.00	11.3	46	3.30
7/22	03:00	-	7.17	15.3	42	3.16
7/22	05:55	17.3	7.17	15.3	35	-
7/22	09:00	-	7.19	14.0	34	-
7/22	11:30	-	7.16	12.1	30	-

RR, Reference Ranges.

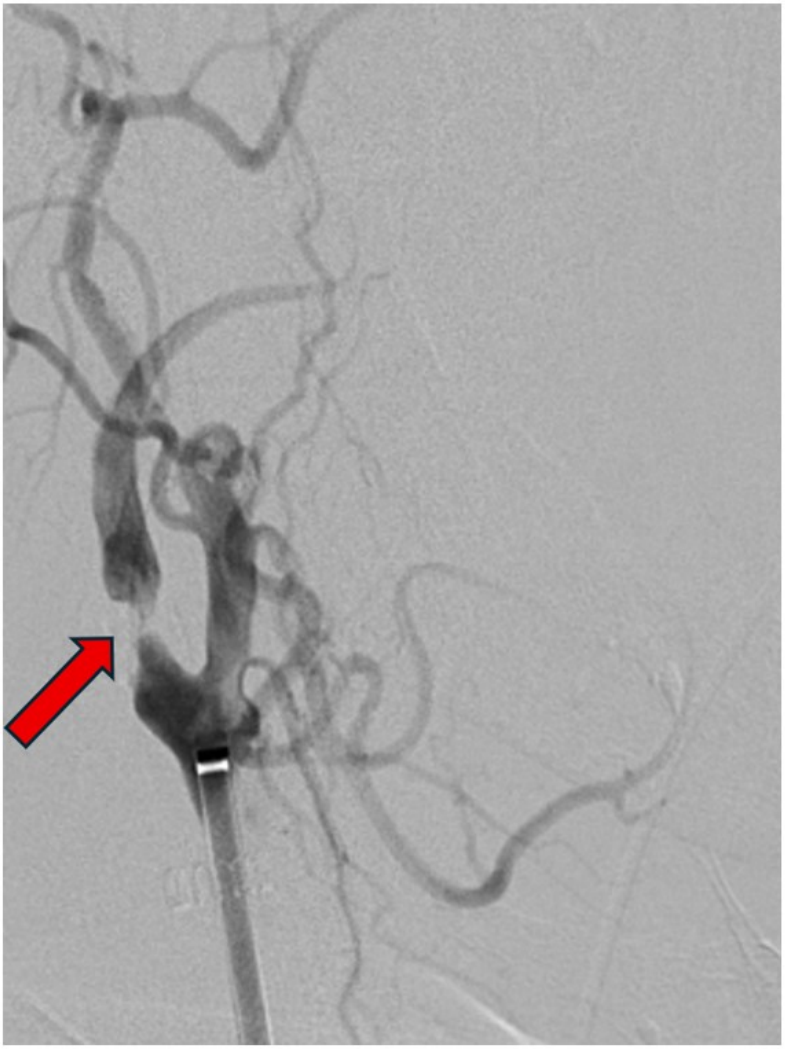

Figure 3: Angiography showing occlusion (Arrow) of the right internal carotid artery.

could not be revived. The cause of death was determined to be hypovolemic shock leading to multiorgan failure. Metabolic and acid – base trends, including lactic acid, are summarized in **(Table 4)**.

3. Discussion

This case represents a rare yet catastrophic complication at the femoral access site after a multimodal endovascular stroke reperfusion therapy, including intravenous thrombolysis, mechanical thrombectomy, carotid angioplasty, closure device deployment, and

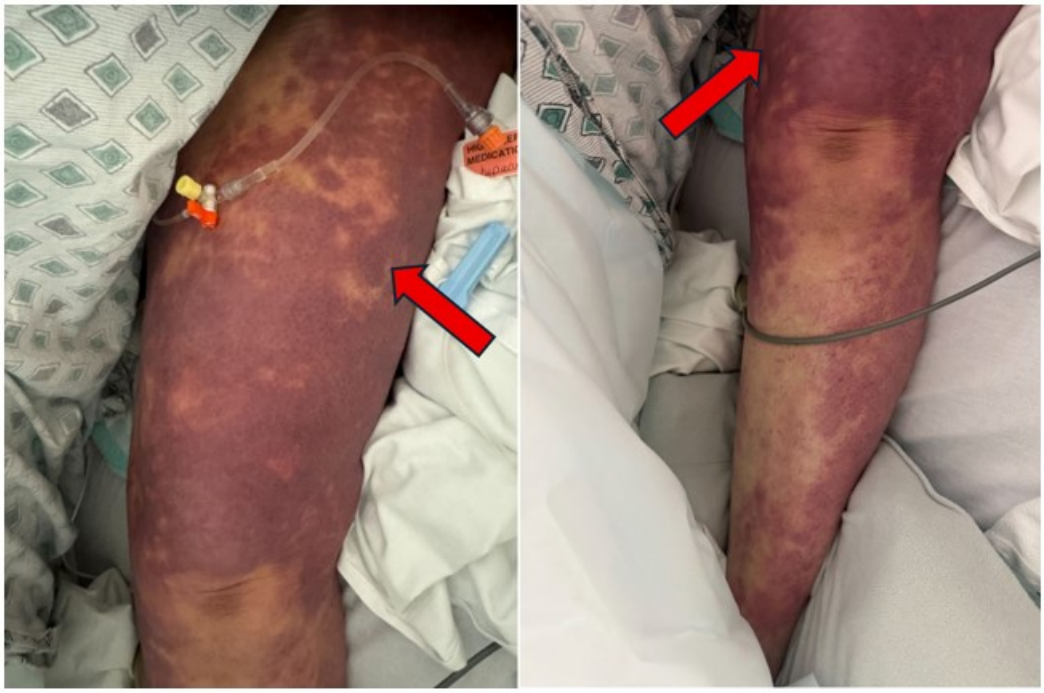

Figure 4: Clinical photograph of the right thigh showing violaceous, reticulated discoloration consistent with livedo reticularis (arrow), appearing early in the course of hematoma expansion.

Table 5: Risk factors for access-site complications and clinical deterioration in the reported case

Category	Factors Present in This Case
Patient factors	Active smoking; chronic kidney disease (eGFR 33); infrarenal abdominal aortic aneurysm; severe iliac tortuosity
Stroke therapy factors	IV tenecteplase; mechanical thrombectomy; carotid angioplasty
Access factors	Large-bore (8F) transfemoral access; closure-device deployment
Antithrombotic factors	Tenecteplase; eptifibatide infusion
Hemodynamic factors	Severe hypertension on arrival; peri-procedural BP fluctuations

IV, intravenous; eGFR, estimated glomerular filtration rate; BP, blood pressure; 8F, 8 French size.

periprocedural eptifibatide. Although most femoral artery pseudoaneurysms have been successfully managed with conservative, endovascular, or surgical approaches, there have been reports of severe hemorrhagic complications with fatal outcomes, particularly when there was a significant delay in diagnosis or definitive hemostasis [2, 8]. In contrast to most prior descriptions, this case was marked by a fatal hemorrhagic course despite early recognition and technically successful endovascular exclusion. Importantly, the case illustrates how initial hemodynamic stability and preserved distal perfusion may delay escalation of care, underscoring the need for heightened vigilance when cutaneous changes or progressive hematoma are present in high risk patients.

Several patient- or procedure-related factors that have been documented in the literature likely contributed to the development of this pseudoaneurysm with significant clinical consequences as shown in **(Table 5)**. For instance, large-bore transfemoral approaches with closure devices have been known to increase the risk of access site complications, particularly with potent antiplatelet therapy [2, 5]. Glycoprotein IIb/IIIa inhibitors, such as eptifibatide, are known to increase the risk of access site bleeding or pseudoaneurysm enlargement with femoral arterial puncture [5, 7]. Significant fluctuations in blood pressure levels, particularly in the peri- or post-procedure period, may increase stress on the arteriotomy site, thereby compromising hemostasis [5].

This case also suggests practical considerations for future risk mitigation strategies. Severe aortoiliac tortuosity and the presence of an infrarenal abdominal aortic aneurysm may warrant careful pre procedural review of aortoiliac imaging when available, with particular attention to vessel geometry and access feasibility. In such patients, early consideration of alternative access routes, including radial, brachial, or direct carotid approaches, may reduce femoral access – site risk when technically appropriate. When transfemoral access is pursued in the setting of challenging anatomy, meticulous puncture technique, cautious selection and deployment of closure devices, and a lower threshold for prolonged manual compression or adjunctive hemostasis strategies may be reasonable. Additionally, patients with complex access anatomy and high cumulative antithrombotic exposure may benefit from heightened post procedural surveillance, including more frequent access site examinations or early adjunctive imaging if concerning clinical signs emerge. These recommendations should be interpreted as pragmatic considerations derived from retrospective insight in a single case, rather than prescriptive guidance. Livedo reticularis is a nonspecific cutaneous finding and may be seen in a range of systemic, vascular, and embolic conditions; however, in this case, its appearance in conjunction with an enlarging groin hematoma and evolving anemia served as an early warning sign of significant access site vascular injury.

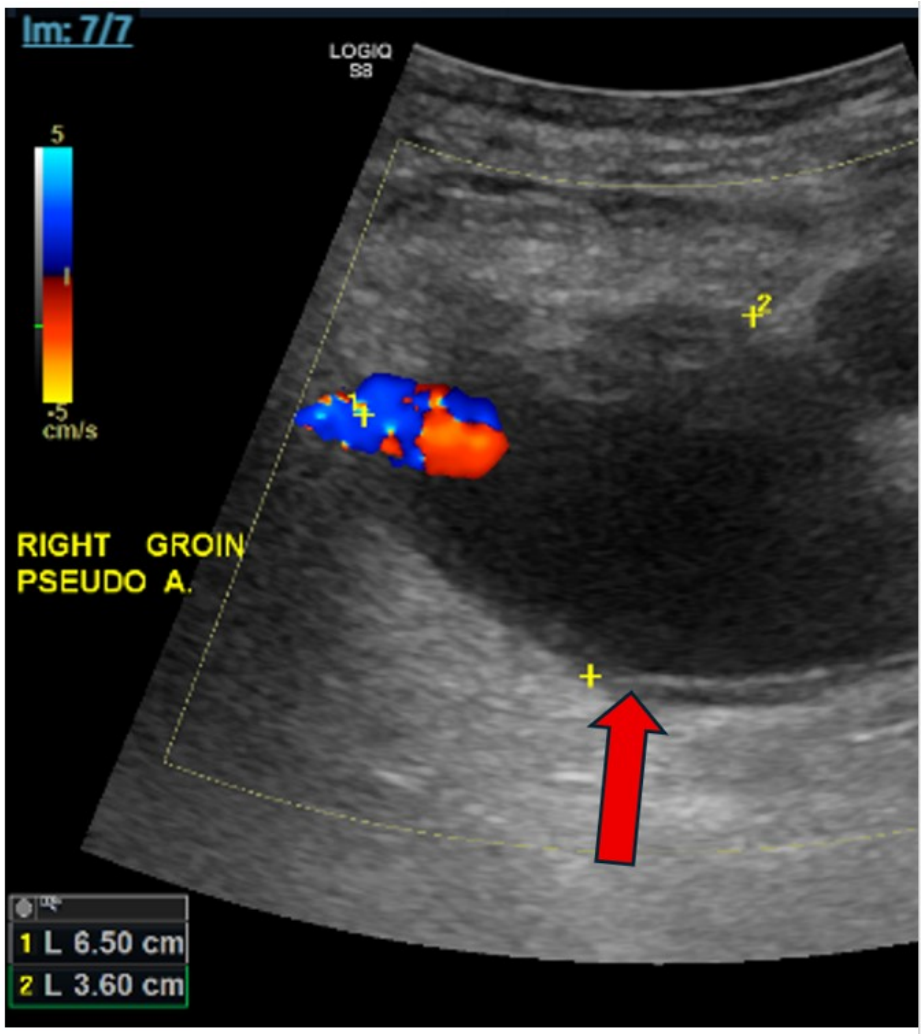

Figure 5: Arterial duplex ultrasound showing a large right groin pseudoaneurysm (Arrow) measuring 6.5 × 3.6 cm.

Femoral access site complications following mechanical thrombectomy are relatively uncommon in reported series; however, published rates vary widely depending on whether cohorts include diagnostic angiography, mixed neuroendovascular procedures, or thrombectomy specific populations. Femoral artery pseudoaneurysms form a small subset of these complications [3, 4]. A study comparing radial and femoral access for mechanical thrombectomy procedures using a meta-analysis demonstrated lower access site complication rates for radial access procedures. This further points to the risk associated with transfemoral access procedures [4]. Recent reviews of neurointerventional procedures continue to identify access site hemorrhage as an important contributor to morbidity and mortality despite overall technical advances [9].

In the present case, endovascular exclusion of the pseudoaneurysm with a stent graft was technically successful; however, the patient subsequently developed refractory hemorrhagic shock with profound metabolic acidosis, hypothermia, and organ failure. As suggested by the prior literature, the cause of death due to severe femoral access site complications is thought to be related more to the physiological consequences of blood loss rather than the failure of vascular repair itself [2, 5, 8]. This case again emphasizes the need for increased post-procedural surveillance following mechanical thrombectomy, especially when using larger femoral access sites, when patients are on potent antithrombotic agents, or when the anatomy is complex. The clinical presentation of the patient with early signs of groin swelling, skin discoloration, unexplained anemia, neurological decline, or cardiovascular instability should prompt immediate investigation with imaging and vascular consultation as emphasized in the management of neurointerventional complications themselves [2, 3, 5, 9]. Although the use of ultrasound and modern closure devices significantly decreases the incidence of complications, the risk of severe or lethal femoral access site complications persists [7, 10].

4. Conclusion

This case demonstrates that severe femoral access site hemorrhage can progress rapidly even after technically successful multimodal endovascular stroke reperfusion therapy and prompt pseudoaneurysm exclusion, particularly in patients with complex aortoiliac anatomy and substantial antithrombotic exposure. The early appearance of livedo reticularis highlights a potentially important, but nonspecific cutaneous warning sign of evolving vascular injury. Although limited to a single case, these observations reinforce the need for careful access planning in anatomically challenging patients and immediate evaluation of early groin changes or unexplained clinical decline to prevent catastrophic outcomes.

Conflicts of Interest

The authors declare no competing interests that could have influenced the objectivity or outcome of this research.

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None.

Informed Consent

Written informed consent for publication of this case was obtained from the patient's Next of Kin.

Large Language Model

None.

Author Contributions

SS contributed to conceptualization, reviewing, data collection, and writing original draft preparation. ZA contributed to supervision, editing, reviewing, writing original draft preparation, validation of diagnostic reasoning, and critical revision. FN contributed to conceptualization, writing of the discussion section, reviewing, and image collection. AIDJ contributed to literature review and writing. TZ contributed to detailed literature review and writing of the introduction section. SA contributed to editing, reviewing, and literature review. AR contributed to literature review and writing of the draft manuscript. JN contributed to supervision, critical revision, and final approval of the manuscript version to be published.

Data Availability

All relevant clinical data supporting the findings of this case report are included within the article, including the clinical timeline, laboratory trends, hemodynamic data, imaging findings, and procedural details. No additional datasets were generated or analyzed. Additional patient-level clinical records are not publicly available because they contain protected health information and are subject to patient confidentiality requirements. De-identified information may be made available from the corresponding author upon reasonable request and subject to institutional approval and applicable privacy regulations.

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