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CASE REPORT

Treatment of a patient diagnosed with cerebral hemorrhage complicated by atypical pulmonary embolism in the rehabilitation department: Case report and anatomical factors analysis.

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Abstract

OBJECTIVE: This report documents the occurrence, clinical management, and outcome of a patient diagnosed with an atypical pulmonary embolism (PE) complicating subacute cerebral hemorrhage in a rehabilitation setting. By integrating the relevant literature, this study aimed to provide a clinical reference for the diagnosis and treatment of atypical PE in patients undergoing post-hemorrhagic rehabilitation.

CASE: The medical history and imaging data from a patient diagnosed with atypical PE during the subacute phase after intracerebral hemorrhage in a rehabilitation setting were analyzed. After diagnostic confirmation, anticoagulation and symptomatic therapies were initiated. A literature review exploring anatomical factors underlying PE in this clinical context was performed. After multidisciplinary treatment, the patient's PE lesions disappeared and indicators, such as blood pressure and heart rate, returned to normal.

CONCLUSION: Intracerebral hemorrhage complicated by atypical PE is a relatively uncommon occurrence in the rehabilitation ward. In this case, conservative low-dose nadroparin calcium anticoagulation was associated with marked biochemical improvement and complete bilateral pulmonary artery clearance on follow-up CTA, without recurrent intracranial hemorrhage. Although no domestic or international guidelines currently address intracerebral hemorrhage complicated by atypical PE, our experience suggests that early prevention, timely diagnosis, individualized anticoagulation, and multidisciplinary collaboration can enable safe and effective management in carefully selected patients.

INTRODUCTION

Pulmonary thromboembolism (PTE) is a common form of pulmonary embolism (PE). PE comprises a group of clinical syndromes characterized by obstruction of the pulmonary arterial system by various emboli, including PTE, fat embolism syndrome, amniotic fluid embolism, and air embolism. After stroke and myocardial infarction, PTE is a common clinical emergency and the third leading cause of cardiovascular mortality globally (Konstantinides *et al.* 2020). Patients with cerebral hemorrhage are prone to venous thrombosis due to factors such as impaired consciousness, cognitive dysfunction, hemiplegia, and prolonged bed rest after onset (Steiner *et al.* 2014).

Notably, PE in the rehabilitation ward setting — distinct from acute neurology wards or ICUs — presents unique diagnostic challenges: patients are ambulatory in structured training programs, vital signs are intermittently monitored, and the classic PE presentation may be masked by baseline neurological deficits. No current domestic or international guidelines address PE management in this specific context, and published cases from rehabilitation departments remain scarce. Herein, we report a case of atypical PE arising during post-ICH rehabilitation and describe the multidisciplinary treatment (MDT) approach that achieved complete radiological resolution without recurrent hemorrhage.

In this report, we use the term atypical PE to describe a presentation lacking the classic triad of chest pain, hemoptysis, and dyspnea, instead manifesting as isolated hypotension and oxygen desaturation, as in the present case. Despite the absence of classic symptoms, early recognition and prompt workup led to timely diagnosis and successful treatment.

CASE PRESENTATION

The patient was a 63-year-old male, who on October 20, 2024, experienced weakness in the left limb while playing poker, accompanied by dizziness and headache, with no nausea, vomiting or other discomforts. The patient presented to the emergency department with a blood pressure (BP) of 193/122 mmHg, while other vital signs were within normal limits. Urgent computed tomography (CT) revealed an intracerebral hemorrhage in the right parieto-occipital lobe. After admission, comprehensive examinations were completed. The patient underwent symptomatic treatment including hemostasis, neurotrophic support, and cerebral protection. This regimen was continued until the cerebral edema was substantially absorbed. After treatment, the patient continued to experience residual left limb motor dysfunction and speech impairment. To seek further rehabilitation treatment, the patient was admitted to the department of rehabilitation medicine at the authors' hospital on November 6, 2024.

Physical examination on admission revealed the following: body temperature, 36.4°C; heart rate (HR), 86 beats/min; respiratory rate, 18 breaths/min; and BP, 112/86 mmHg. Specialty physical examination revealed that the patient was alert and oriented but appeared lethargic, with slurred speech. Auditory comprehension was intact, and responses were partially relevant. Orientation, calculation, and memory were impaired, with a Mini-Mental State Examination score of 19 (indicating moderate cognitive impairment). Both pupils were equal and round, approximately 3 mm in diameter, with prompt light reflexes. Extraocular movements were intact. Bilateral forehead wrinkles were symmetrical. There was left-sided flattening of the nasolabial fold, tongue deviation to the left, and

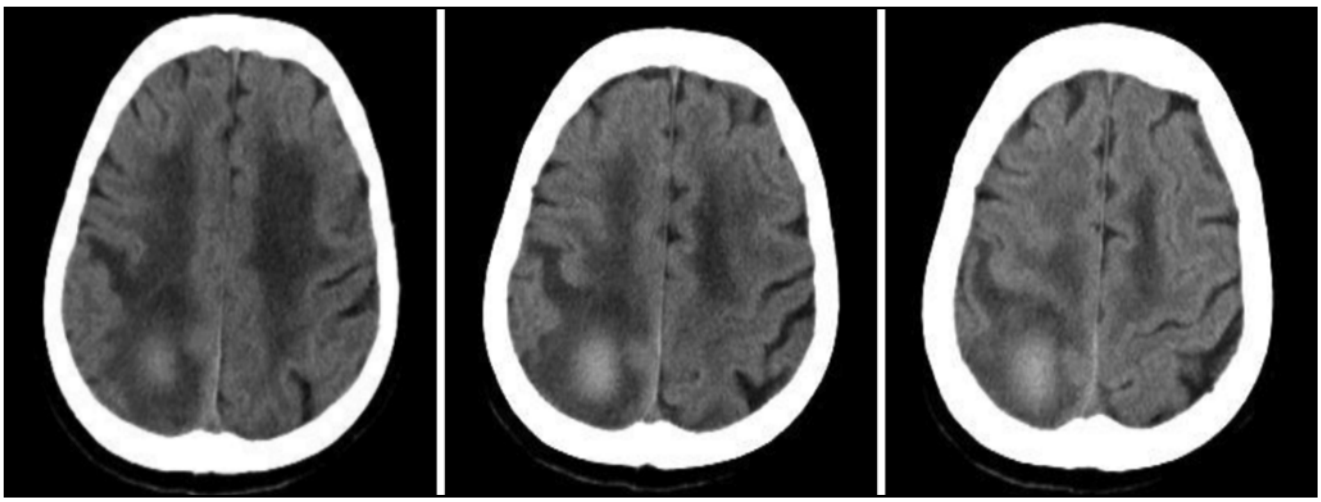

Fig. 1. Cranial computed tomography (CT) on rehabilitation admission (November 6, 2024), 17 days after onset of intracerebral hemorrhage. A hyperdense area is visible in the right parieto-occipital lobe (approximately 24 × 17 mm), consistent with a hematoma in the absorption phase. No midline shift or new hemorrhagic expansion was identified. This imaging confirmed the presence of residual hemorrhage and served as the baseline reference for assessing anticoagulation safety throughout subsequent treatment.

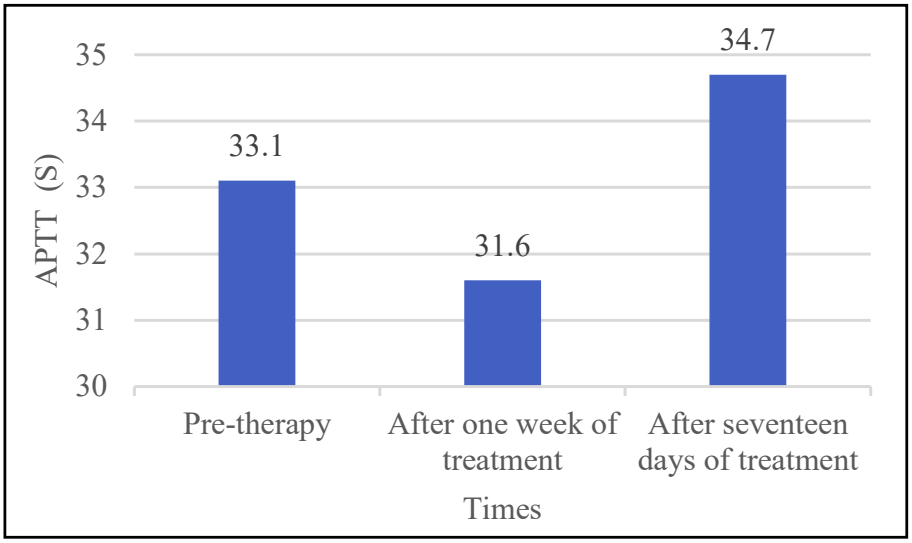

Fig. 2. Serial activated partial thromboplastin time (APTT) values before and after anticoagulation therapy. Baseline APTT on ICU admission was 33.1 s. After 7 days of low-dose nadroparin calcium (2050 IU once daily), APTT was 31.6 s; after a further 17 days at double dose (2050 IU twice daily), APTT was 34.7 s. Because a low-molecular-weight heparin was utilized, all values remained near the baseline level rather than reaching the conventional 1.5–2.5× normal reference range targeted with unfractionated heparin. The stable APTT trajectory confirmed that no over-anticoagulation occurred, and bleeding risk remained controllable throughout the treatment course.

drooping of the left corner of the mouth. Performance on puffing cheeks, showing teeth, and whistling was poor. Other findings included the following: Muscle strength (MMT), Left UE (shoulder-elbow-wrist-fingers): 3-3-4-4; Left LE (hip-knee-ankle-toes), 3-3-3-3; Modified Ashworth Scale (MAS), left knee extensors, grade 1; Brunnstrom Stage, left side, IV-V-IV; Sensation, left-sided superficial sensation was decreased, with position sense and two-point discrimination intact; left tendon reflexes (+++); Babinski sign (–);unable to sit unsupported; and Barthel Index, 30 (severe functional impairment in the activities of daily living [ADL]).

Medical history as per family revealed no routine BP monitoring. Pertinent history included smoking and alcohol consumption. The patient denied a history of diabetes or coronary artery disease. Diagnoses included medical (cerebral hemorrhage, absorption phase /hypertension stage 3 [very high risk]) and rehabilitation (left hemiparesis/cognitive dysfunction/speech impairment/severe limitations in ADL).

After admission, relevant examinations were completed, including cranial CT, electrocardiogram, echocardiogram, lower limb vascular ultrasound, routine blood tests, biochemical tests, myocardial enzymes, brain natriuretic peptide, etc. Symptomatic supportive treatment for underlying diseases was initiated. A hyperdense area was evident in the right parietal lobe, measuring approximately 24 × 17 mm, suggesting changes during the absorption phase of the cerebral hemorrhage (Figure 1). Coagulation function tests revealed a D-dimer level of 16.25 mg/L (normal range, 0–0.55 mg/L). Vascular ultrasonography of both the lower limbs revealed no thrombosis. Moreover, the patient had no chief complaints of discomfort, and vital signs were stable. Accordingly, comprehensive training for the hemiplegic limb was initiated to improve muscle strength on the affected side, joint mobilization was applied to enhance limb joint mobility, and lower-limb cycle ergometer training along with medium-frequency pulse electrical stimulation was used to improve motor control of the hemiplegic limb. Concurrently, cognitive and speech function training was provided to enhance the patient's cognitive and speech abilities while vital signs were closely monitored.

At 07:00 on day 4 of hospitalization, the patient suddenly developed hypotension (BP, 82/45 mmHg), with an HR of 93 beats/min, and a peripheral oxygen saturation of 91%. Low-flow oxygen therapy (2–3 L/min) was administered, and re-measurement revealed a BP of 89/63 mmHg and peripheral oxygen saturation of 95%. At 07:40, BP was 121/40 mmHg, and electrocardiographic monitoring and volume expansion with 500 ml of glucose-saline solution were initiated. Electrocardiography revealed sinus rhythm with narrowing of the T waves in some leads. Re-examination of the cardiac enzyme spectrum and brain natriuretic peptide revealed no significant abnormalities. Subsequently, further CT angiography (CTA) of the pulmonary arteries was performed, demonstrating multiple pulmonary emboli in both pulmonary arteries (as indicated by Figure 5A and 5C). Due to the critical condition, the patient was transferred to the intensive care unit (ICU) on the same day for further treatment.

Upon admission to the ICU, bilateral lower limb vascular ultrasound performed the following day revealed atherosclerotic plaque formation in the right common femoral artery, as well as thrombosis in the intermuscular veins of both calves. This presentation represented hemodynamically unstable straddling type acute PE. The pulmonary artery embolism severity score was 2 points, indicating moderate risk with a significant impact on pulmonary artery blood flow, a constellation associated with high mortality. Subsequently, a multidisciplinary treatment mode was initiated. After evaluation, the department of respiratory medicine concluded that the patient had developed

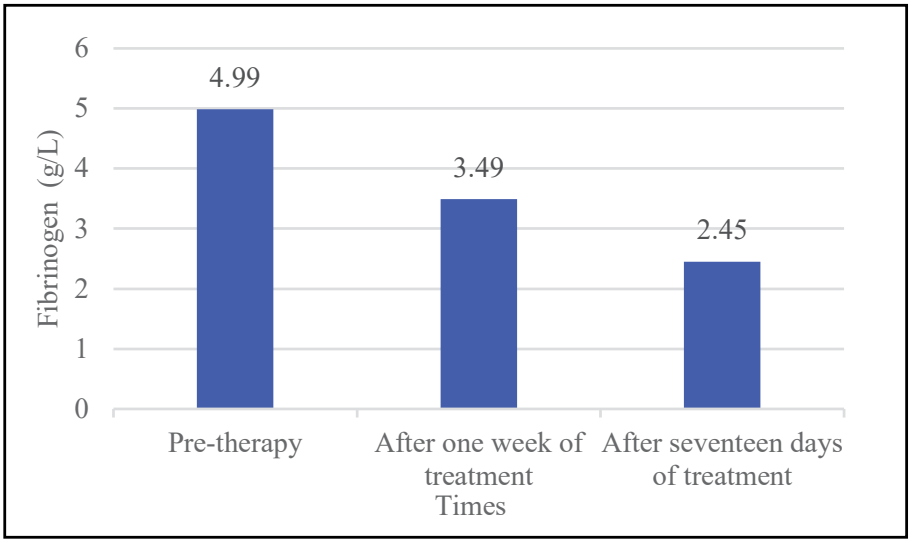

Fig. 3. Serial plasma fibrinogen levels before and after anticoagulation therapy. Fibrinogen at baseline was 4.99 g/L. After 7 days of nadroparin calcium (2050 IU once daily), it decreased to 3.49 g/L; after 17 additional days at double dose (2050 IU twice daily), it fell to 2.45 g/L — remaining within the normal range throughout. As the primary coagulation substrate consumed during endogenous fibrinolysis, the progressive decline in fibrinogen provided laboratory evidence that anticoagulation alone was sufficient to sustain effective clot dissolution without exogenous thrombolytic agents.

straddling-type pulmonary artery thrombosis with hemodynamic instability. Given that the patient was 17 days post-hemorrhage and had clear contraindications to thrombolysis, cautious low-dose anticoagulation was recommended. The department of cardiology suggested attempting local alteplase (rt-PA) administration via right heart catheterization but noted through risk assessment that novel anticoagulants offered no significant safety advantage. It was recommended to initiate nadroparin calcium anticoagulation first, followed by warfarin. Considering the patient's cerebral hemorrhage and clear contraindications for intravenous thrombolysis, the department of neurology advised neutral treatment. The department of neurosurgery considered that the etiology of the PE and intracranial hemorrhage remained unclear, warranting vigilance for possible recurrent intracranial hemorrhage and, thus, also recommended neutral treatment—more specifically, conservative anticoagulation therapy. Based on the above multidisciplinary consultations and after discussing the risks and benefits with the patient's family, low-dose nadroparin calcium anticoagulation therapy was initiated.

On admission, the patient's activated partial thromboplastin time (APTT) was 33.1 s, and fibrinogen and D-dimer levels were 4.99 g/L and 16.25 mg/L, respectively. After anticoagulation using nadroparin calcium subcutaneously (2050 IU; equivalent to 0.2 mL of a 4100 IU/0.4 mL solution) once daily for 7 days, coagulation function was re-examined, revealing an APTT of 31.6 s, and fibrinogen and D-dimer levels of 3.49 g/L and 5.03 mg/L, respectively. Repeat pulmonary angiography indicated multiple pulmonary emboli in both pulmonary arteries. Subsequently, nadroparin calcium at the same dose (2050 IU, subcutaneously) was administered twice daily for 17 days. A subsequent re-examination of coagulation function revealed an APTT of 34.7 s, and fibrinogen and D-dimer levels of 2.45 g/L and 2.06 mg/L, respectively. Changes in coagulation indicators before and after treatment are

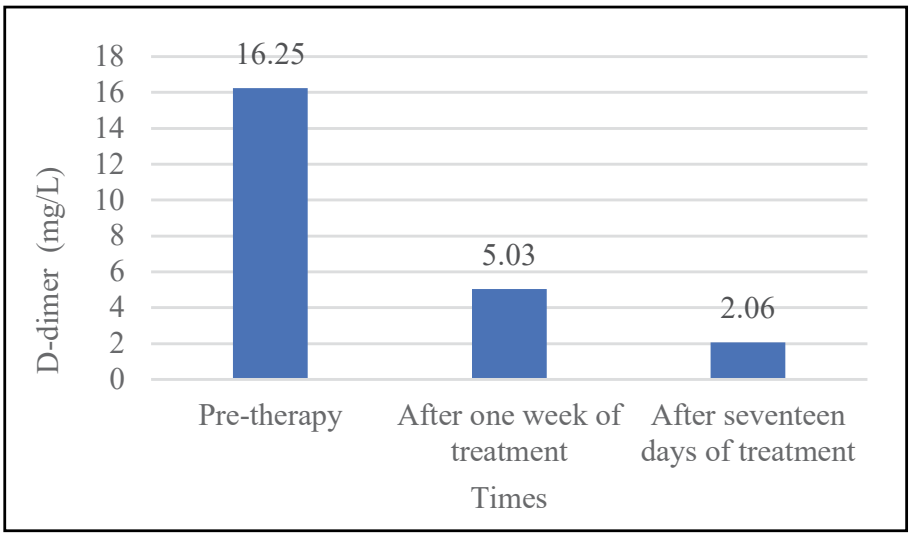

Fig. 4. Serial plasma D-dimer levels before and after anticoagulation therapy. D-dimer was markedly elevated at baseline (16.25 mg/L; normal range 0–0.55 mg/L), reflecting the acute thrombotic burden at the time of PE diagnosis. After 7 days of low-dose nadroparin calcium (2050 IU once daily), D-dimer fell to 5.03 mg/L; after a further 17 days at escalated dose (2050 IU twice daily), it declined to 2.06 mg/L. The stepwise reduction toward near-normal levels is consistent with progressive vascular recanalization, which was documented on follow-up CTA (Figure 5B, 5D).

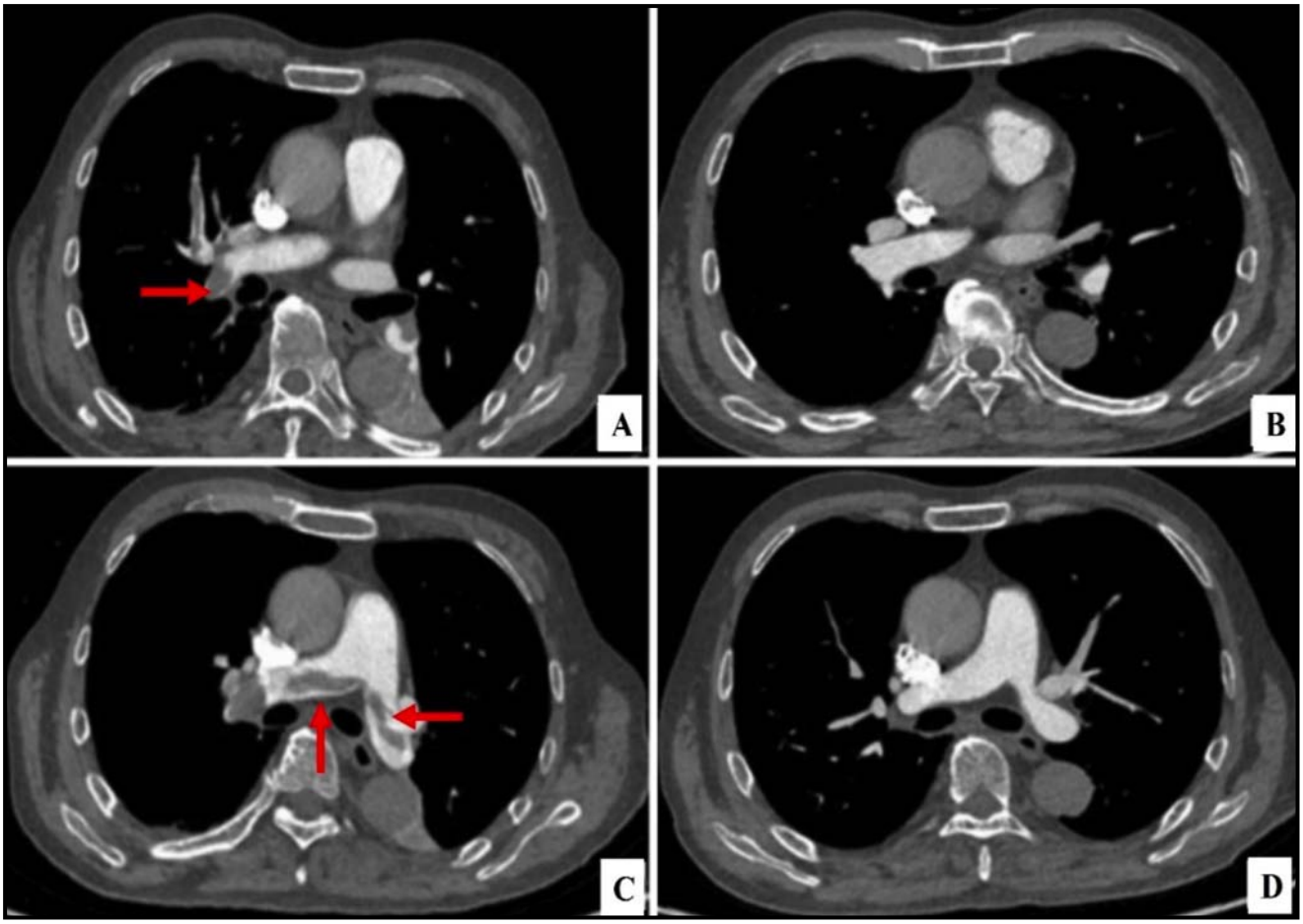

Fig. 5. Pulmonary artery CT angiography (CTA) before and after anticoagulation therapy. 5A, 5C: CTA acquired on day 4 of rehabilitation hospitalization (day 21 post-ICH onset), demonstrating multiple filling defects in both pulmonary arteries consistent with bilateral pulmonary thromboembolism, including a straddling embolus at the main bifurcation. Hemodynamic instability (BP 82/45 mmHg, SpO_2 91%) was present at the time of this study. 5B, 5D: Follow-up CTA acquired 17 days after initiation of low-dose heparin sodium anticoagulation, showing near-complete resolution of bilateral pulmonary emboli with no residual filling defects identified. No new intracranial hemorrhage was detected during the interval between studies.

reported in Figures 2, 3, and 4. Changes in APTT are presented in Figure 2. Because a low-molecular-weight heparin (nadroparin calcium) was utilized, the APTT remained near the baseline level (31.6–34.7 s) rather than reaching the conventional therapeutic target of 1.5–2.5 times the normal reference range, indicating that the anticoagulant drug dosage is appropriate, the anticoagulant state is stable, no over-anticoagulation has occurred, and the bleeding risk is controllable (Kearon *et al.* 2016). Changes in fibrinogen levels are presented in Figure 3. As a coagulation substrate, fibrinogen serves as key laboratory evidence for treatment efficacy. Its progressive decline from 4.99 g/L to 2.45 g/L over 24 days reflects consumption by endogenous fibrinolytic activity and supports that anticoagulation alone was sufficient to permit effective clot dissolution without exogenous thrombolytic agents in this patient (Henke & Wakefield, 2009). Changes in D-dimer level, which is a key molecular marker reflecting the formation and dissolution of fibrin, are presented in Figure 4. In patients with acute PE, baseline levels are associated with disease severity. After effective anticoagulation therapy, a rapid decrease in D-dimer to normal or near-normal levels is considered to be laboratory evidence supporting vascular recanalization and stabilization of fibrinolytic activity, and is closely associated with a favorable clinical prognosis (Konstantinides *et al.* 2020).

Follow-up CTA demonstrated near-complete resolution of bilateral pulmonary emboli, with no residual filling defects identified in either pulmonary artery (Figure 5B and 5D). Rivaroxaban was changed to oral maintenance anticoagulation therapy and no new bleeding or embolism events occurred. The patient was then weaned off the ventilator and discharged. The detailed clinical timeline of key events is summarized in Table 1.

This study was approved by the Ethics Committee of Huadong Hospital Affiliated to Fudan University (No. 20220068). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent for publication of this case report and accompanying images was obtained from the patient and his family.

Tab. 1. Clinical Timeline of Key Events

Calendar Date	Days Post-ICH	Setting	Clinical Event	Key Vitals & Labs / Imaging	Intervention
Oct 20, 2024	Day 0	Emergency Dept.	ICH onset; left limb weakness, dizziness, headache	BP 193/122 mmHg; CT: right parieto-occipital ICH	Haemostasis, neurotrophic & cerebral protection therapy initiated
Nov 6, 2024	Day 17	Rehab Dept. (Admission)	Transfer for post-ICH rehabilitation	BP 112/86; HR 86; MMSE 19; Barthel 30; D-dimer **16.25 mg/L** ↑; lower-limb US: **negative**	Comprehensive rehab programme; close monitoring
Nov 9, 2024	Day 21	Rehab Ward → ICU	Sudden haemodynamic collapse (07:00); atypical PE diagnosed	BP 82/45 mmHg; SpO_2 **91%**; ECG: T-wave narrowing; pulmonary CTA: **bilateral PE + straddling embolus**	O_2, volume expansion; CTA; **transferred to ICU**
Nov 10, 2024	Day 22	ICU Day 2	Repeat lower-limb US; MDT consultation	Bilateral **calf intermuscular vein thrombosis** confirmed	MDT decision: conservative **low-molecular-weight heparin** anticoagulation
Nov 10, 2024	Day 22	ICU Day 2	Anticoagulation baseline & therapy start	APTT 33.1 s; Fibrinogen 4.99 g/L; D-dimer **16.25 mg/L**	**Nadroparin calcium 2050 IU OD** initiated
Nov 17, 2024	Day 29	ICU Day 9	7-day coagulation re-check; repeat CTA	APTT 31.6 s; Fibrinogen 3.49 g/L; D-dimer **5.03 mg/L** ↓; CTA: PE still present	Dose **escalated to 2050 IU BID**
Dec 4, 2024	Day 46	ICU Day 26	17-day coagulation re-check; follow-up CTA	APTT 34.7 s; Fibrinogen 2.45 g/L; D-dimer **2.06 mg/L** ↓; CTA: **bilateral arteries clear**; no new ICH	Switched to oral **rivaroxaban**; ventilator weaned; **discharged**

Abbreviations: BP, blood pressure (mmHg); HR, heart rate (beats/min); RR, respiratory rate (breaths/min); Temp, body temperature (°C); SpO_2, peripheral oxygen saturation; CT, computed tomography; CTA, computed tomography angiography; US, venous ultrasonography; MMSE, Mini-Mental State Examination; ADL, activities of daily living; ICH, intracerebral haemorrhage; PE, pulmonary embolism; ECG, electrocardiography; BNP, brain natriuretic peptide; APTT, activated partial thromboplastin time; OD, once daily; BID, twice daily; MDT, multidisciplinary team.
Symbols: ↑ above reference range; ↓ decreasing from prior value (direction of change). **Bold** values denote clinically critical findings or decision-driving results. Days Post-ICH are counted from the date of intracerebral haemorrhage onset (October 20, 2024 = Day 0). ICU day numbering begins on the day of ICU admission (November 10, 2024 = ICU Day 2, as the patient was transferred on November 9 and assessed the following morning). Calendar dates for ICU Day 9 and Day 26 are derived from the manuscript-stated treatment durations (7 days and 17 days, respectively) described in the case presentation.

DISCUSSION

Etiology, pathophysiology, and diagnosis of cerebral hemorrhage complicated by PE

Etiology

The etiology of PE occurring after cerebral hemorrhage may include the following. First, central nervous system lesions, such as cerebral hemorrhage, can activate the coagulation system, thereby promoting thrombus formation (Gao & Chen, 2016). Second, contralateral limb motor dysfunction — in this case left hemiparesis — leads to weakened lower-limb muscle contraction, resulting in reduced venous flow velocity and stasis in the hemiplegic limb, thereby facilitating thrombus formation (Yuan *et al.* 2023). Patients with intracerebral hemorrhage are prone to aspiration due to excessive bed rest, swallowing dysfunction, and other factors that can lead to respiratory tract infections. Severe cases can develop bloodstream infections. Studies have shown that concurrent respiratory tract and bloodstream infections are associated with venous thromboembolism in patients with intracerebral hemorrhage (Melmed *et al.* 2021). Most patients with intracerebral hemorrhage have multiple comorbidities such as diabetes mellitus and atrial fibrillation. Diabetes mellitus has been confirmed to be associated with various coagulation and fibrinolytic defects (Dong *et al.* 2023). Atrial fibrillation has been confirmed to have a bidirectional interaction with venous thromboembolism (Miao *et al.* 2023). Mural thrombi attached to the atrial wall may dislodge and travel through the bloodstream to the pulmonary artery, forming a pulmonary embolism. Pulmonary embolism increases the afterload on the right ventricle, which, in turn, can induce atrial fibrillation. The occurrence of venous thromboembolism in patients

with intracerebral hemorrhage is associated with the degree of consciousness impairment (Dong *et al.* 2023) and the Glasgow Coma Scale (GCS) score on admission: the lower the GCS score, the greater the risk for venous thromboembolism in patients with intracerebral hemorrhage. The main causes of embolism in such patients include activation of the coagulation system, motor dysfunction, immobility, and impaired consciousness.

Pathophysiology
Virchow's triad (stasis of blood flow, endothelial injury, and a hypercoagulable state) represents the 3 core pathophysiological mechanisms of acute PE (Turpie *et al.* 2002). Severe acute conditions (such as intracerebral hemorrhage) induce systemic inflammation and stress responses that directly lead to a hypercoagulable state. Its pathophysiology is highly complex: sudden obstruction of the pulmonary artery by an embolus triggers varying degrees of pulmonary artery constriction, resulting in reduced or even interrupted pulmonary blood flow. This subsequently causes varying degrees of hemodynamic and gas exchange impairments, primarily manifesting as heart and respiratory failure.

Diagnosis
The clinical manifestations of PE vary depending on factors including the size and number of emboli, location of the embolism, and other comorbidities. Small emboli may not cause clinical symptoms (Delmas *et al.* 2020), whereas larger emboli may cause symptoms, such as tachypnea, dyspnea, palpitations, syncope, panic, and hypoxia. The specific manifestation of PE is the triad (chest pain, hemoptysis, and dyspnea), which is uncommon in clinical practice. The initial clinical presentation in this patient — decreased BP (82/45 mmHg) and oxygen desaturation (SpO_2 91%) — improved only partially with low-flow oxygen therapy and was not accompanied by the classic triad of chest pain, hemoptysis, or dyspnea, illustrating the atypical nature of PE presentation in neurologically impaired patients. Diagnostic methods for deep vein thrombosis (DVT) include D-dimer formation by the conversion of cross-linked fibrin under the action of the fibrinolytic mechanism. When a thrombus occurs, the relevant fibrin is dissolved, leading to an increase in plasma D-dimer concentration (Konstantinides *et al.* 2020). Its sensitivity is as high as 92%–100%; however, its specificity is low and can be influenced by various factors such as surgery, trauma, pregnancy, tumors, infections, and acute myocardial infarction. It is currently the primary indicator of acute PE.

Venous pressure measurement
Elevated venous pressure in the affected limb suggests obstruction of the veins proximal to the measurement site.

Ultrasonography
Mainly used to diagnose proximal DVT, with a positivity rate and specificity of up to 95%.

Radioisotope examination
^{125}I fibrinogen scanning has a positivity rate of 90% for DVT in calf muscles but has poor specificity for the diagnosis of proximal DVT.

Deep vein angiography
Localization and qualitative diagnosis can be performed by observing the filling defects in the deep veins.

Treatment and prevention of acute PE complicating cerebral hemorrhage

Effective anticoagulation
Anticoagulation is the most fundamental treatment for PE and is currently recognized as the method to reduce the associated mortality rate (Konstantinides *et al.* 2020). In a study by Chen *et al.* (Chen & Mei, 2024), 102 patients with cerebral hemorrhage complicated by PE were treated with low-molecular-weight heparin at different doses for anticoagulation. The results demonstrated that low-dose low-molecular-weight heparin anticoagulation therapy effectively improved patient symptoms, coagulation function indicators, and prognosis, and has higher safety. Consistent with these findings, in the present case we selected subcutaneous nadroparin calcium, a low-molecular-weight heparin, instead of unfractionated heparin to provide anticoagulation with a more predictable pharmacological profile in the subacute phase of cerebral hemorrhage. This choice reduced the need for intensive dose titration and frequent APTT monitoring and may have facilitated a more stable rehabilitation course for this patient.

Rational thrombolysis
Thrombolysis is an important and effective treatment for PE. Current guidelines recommend that thrombolysis should be administered to patients with massive PE without contraindications. However, thrombolysis may increase the risk for bleeding in patients with cerebral hemorrhage combined with PE and hemodynamic instability. For example, bleeding caused by secondary factors, such as the rupture of arteriovenous malformations or cerebral amyloid angiopathy, has a high risk for rebleeding, making thrombolysis or anticoagulation contraindicated (Konstantinides *et al.* 2014). Some investigators have argued that the current contraindications for thrombolysis are mainly derived from data on acute coronary syndrome. Cases of massive PE with high mortality should be considered relative rather than absolute contraindications (Meneveau, 2010). For example, one case report described a 75-year-old male with thalamic hemorrhage complicated by a massive PE and cardiac arrest. According to current guidelines, thrombolysis is contraindicated; however, after weighing the risks and benefits, thrombolysis

was successfully performed, and the patient recovered safely without further intracranial bleeding (Akanmode *et al.* 2023). This study also provides a reference for the successful rescue of such patients.

Timely prevention

The focus of early treatment for intracerebral hemorrhage is hemostasis and the prevention of hematoma expansion, whereas the focus of treatment for acute PE is anticoagulation and the prevention of thrombus recurrence. The clinical conditions are complex and variable. In patients with intracerebral hemorrhage complicated by acute PE, preventing intracranial hemorrhage and reducing thrombus formation present a "double-edged sword". Treatment measures for acute PE in patients with intracerebral hemorrhage are empirical, and guideline recommendations remain lacking (Chu *et al.* 2021). Therefore, active prevention should be implemented in patients at a risk for thromboembolism. According to the 9th Edition of the Clinical Practice Guidelines for Antithrombotic Therapy and Thromboprophylaxis, published by the American College of Chest Physicians, for patients with low thrombosis risk, basic prevention measures can be used, such as health education, early mobilization, and avoidance of dehydration. For patients with higher thrombosis risk, anticoagulant drugs for prevention should be used, such as sodium heparin, rivaroxaban, or warfarin, and it is not recommended to continue their use after unrestricted activity or discharge. For patients with yet higher thrombosis and bleeding risks, mechanical prevention can be initially adopted, such as compression stockings, lower limb pneumatic compression devices, and transcutaneous electrical nerve stimulation, until the bleeding risk is reduced to the minimum (Achraf *et al.* 2025).

Anatomical factors in PTE

Anatomical basis of thrombosis formation

First, there are a large number of venous valve sinuses in the deep veins of the lower extremities, especially in the gastrocnemius venous plexus. The blood flow in the sinus behind the valve tip is slow and prone to vortex formation, thus leading to primary thrombosis. Consistent with this mechanism, ICU ultrasound in the present patient confirmed thrombosis specifically in the intermuscular veins of both calves — the precise anatomical location predicted by this pathophysiology — rather than in the more proximal vessels where initial rehabilitation-admission ultrasound had been negative. This progression from a clinically silent calf thrombus to bilateral straddling PE illustrates how the rehabilitation environment (immobility between active therapy sessions, diminished muscle pump activity from hemiplegia) may accelerate venous stasis in precisely these susceptible segments. Second, left common iliac vein compression syndrome is the most important local anatomical factor. The left common iliac vein passes in front of the fifth vertebral body and behind the right common iliac vein at this location, where it is prone to mechanical compression (May-Thurner anatomical structure), which can lead to intimal hyperplasia, adhesion, and lumen stenosis of the vein, significantly increasing the risk for DVT in the left lower limb (May & Thurner, 1957). It has been reported that approximately 20%–25% of left lower extremity vein thromboses are associated with this anatomical variation (Kibbe *et al.* 2004).

Vascular lumen volume and blood flow velocity

The venous system has a large lumen, low pressure, and low flow velocity. In particular, when lying down or immobilized, muscle pump function disappears, exacerbating blood stasis.

Anatomical sites of vascular endothelial injury

Venous bifurcations, valve tips, and venous segments directly involved in surgery or trauma (e.g., the popliteal vein region after total knee arthroplasty) are prone to endothelial injury, which can trigger the coagulation process due to changes in blood flow shear stress or direct damage (López & Chen, 2009).

Fixed pathway of thrombus migration

After detachment, thrombi strictly follow the venous return direction of the systemic circulation: peripheral deep veins → iliac veins → inferior vena cava → right atrium → right ventricle → pulmonary artery. The uniqueness of this pathway determines that lower extremity DVT is the primary source of PTE.

The 'sieve' anatomy of the pulmonary artery

The main trunk of the pulmonary artery bifurcates into the left and right pulmonary arteries, which further branch into the lobar, segmental, and subsegmental arteries. The embolus ultimately becomes lodged in a vessel whose diameter matches its diameter. The area of the pulmonary infarction comprises the region supplied by the embolized branch of the pulmonary artery. Typical pulmonary infarcts are wedge-shaped, with their base located in the pleura and their apex pointing toward the hilum of the lung.

Risk Stratification and Multidisciplinary Management

Given the "double-edged sword" nature of treating hemodynamically unstable PE in the setting of recent ICH, no single specialty holds the complete risk picture. In our case, the MDT approach was essential: respiratory medicine assessed PE severity and favored anticoagulation; cardiology considered catheter-directed thrombolysis but ultimately recommended low-molecular-weight heparin; and neurology/neurosurgery advised strict vigilance for rebleeding given the ICH contraindications. This collaborative format ensured that competing risks were weighed collectively before an individualized plan was agreed upon. Based on the successful management of this single case, we

Tab. 2. Pre-Anticoagulation Risk-Stratification Checklist For Post-ICH Patients with Suspected or Confirmed PE in a Rehabilitation Setting

Step / Domain	Clinical Criteria & Actions (Based on the specific case profile)
STEP 1: Confirm PE Diagnosis	• Pulmonary CTA performed and bilateral/unilateral filling defects confirmed • Haemodynamic instability documented (BP, HR, SpO_2 recorded) • Cardiac enzymes and BNP checked to exclude primary cardiac cause • ECG obtained
STEP 2: Timing Gate	• Document **exact days elapsed since ICH onse**t • Note: in this case, conservative anticoagulation was deemed feasible at **≥ Day 17 post-ICH** • Earlier timing requires additional MDT justification and neurosurgical sign-off
STEP 3: Hematoma Stability Assessment	• Repeat cranial CT obtained at time of PE diagnosis • Hematoma size measured: document length × width (mm) • Confirm **no midline shift** • Confirm **no new haemorrhagic expansion** vs. prior CT • Note: in this case, stable hematoma of **24 × 17 mm in absorption phase** was the threshold accepted as safe to anticoagulate
STEP 4: Baseline Coagulation Profile	• APTT measured: _ s (baseline in this case: **33.1 s**) • Fibrinogen measured: _ g/L (baseline: **4.99 g/L**) • D-dimer measured: _ mg/L (baseline: **16.25 mg/L**; normal range 0–0.55 mg/L) • Confirm no pre-existing coagulopathy or anticoagulant use
STEP 5: Contraindication Screening	• Systemic thrombolysis: **CONTRAINDICATED** if active or recent ICH • Novel oral anticoagulants: cardiology noted **no significant safety advantage** in this context • Anticoagulation: proceed only if haemostasis confirmed and hematoma stable (Steps 2–3 passed) • Document presence/absence of: arteriovenous malformation, cerebral amyloid angiopathy (high rebleed risk → contraindication [Konstantinides *et al.* 2014])
STEP 6: MDT Consultation	• Respiratory medicine, Cardiology, Neurology, and Neurosurgery consulted • Risks and benefits discussed with patient/family and consent documented
STEP 7: Initiate Conservative Anticoagulation	• Phase 1: Nadroparin calcium 2050 IU once daily for 7 days • Phase 2: Nadroparin calcium 2050 IU twice daily for 17 days • Target: APTT maintained near baseline (low-molecular-weight heparin profile) • APTT checked at Day 7 of anticoagulation • APTT checked at Day 24 of anticoagulation (end of BID phase) • If APTT > 2.5× normal → bleeding risk elevated; reassess dose
STEP 8: Escalation Decision at Day 7	• Repeat coagulation labs (APTT, fibrinogen, D-dimer) • Repeat pulmonary CTA • If PE persists AND APTT remains stable AND no new ICH → **escalate to BID dosing** • If new ICH detected → **halt anticoagulation; urgent neurosurgical review**
STEP 9: Treatment Response Targets	• D-dimer (mg/L): Baseline (16.25) → Day 7 (5.03) → Day 24 (2.06) → Goal: Trend toward ≤ 0.55 • Fibrinogen (g/L): Baseline (4.99) → Day 7 (3.49) → Day 24 (2.45) → Goal: Progressive decline (active endogenous fibrinolysis) • APTT (s): Baseline (33.1) → Day 7 (31.6) → Day 24 (34.7) → Goal: Stable near baseline (confirming no over-anticoagulation) • CTA findings: Bilateral PE → Persistent PE → Bilateral clearance → Goal: No residual filling defects
STEP 10: Discharge Decision	• CTA confirms bilateral pulmonary artery clearance • No new intracranial haemorrhage on follow-up CT • APTT within target throughout course • Switch to oral anticoagulant (rivaroxaban used in this case) • No new bleeding or embolic events prior to discharge

Important caveat: This checklist reflects a single patient's management. Every threshold listed is specific to this patient's risk profile. It must not be adopted without analogous individual risk stratification and MDT review. No international guideline currently endorses a standard protocol for this clinical scenario.

Abbreviations: PE, pulmonary embolism; CTA, computed tomography angiography; BP, blood pressure; HR, heart rate; SpO_2, peripheral oxygen saturation; BNP, brain natriuretic peptide; ECG, electrocardiography; ICH, intracerebral hemorrhage; MDT, multidisciplinary team; CT, computed tomography; APTT, activated partial thromboplastin time; BID, twice daily.

propose a pre anticoagulation risk stratification checklist (Table 2) as a hypothesis generating reference rather than a prescriptive protocol for clinicians facing similar dilemmas.

CONCLUSION

Several limitations of this report merit acknowledgment. First, as a single-patient case report, the findings cannot be generalized; the management strategy described was individualized to this patient's specific risk profile (day 17 post-hemorrhage, stable 24×17 mm hematoma, APTT maintained near baseline) and should not be adopted without analogous risk stratification. The management strategy described was tailored to a patient presenting at day 17 post-haemorrhage with a radiologically stable, moderate-sized hematoma (24 × 17 mm in absorption phase), hypertension stage 3, and no prior anticoagulant exposure — a constellation that permitted a conservative low-dose low-molecular-weight heparin approach that may not be safe or sufficient in patients with larger or actively expanding hematomas, earlier post-ictus timing, or pre-existing coagulopathy. Clinicians should therefore view the thresholds reported here (timing, hematoma size, APTT targets, dose escalation schedule) as hypothesis generating reference points that require validation in larger cohorts, not as transferable decision rules for the heterogeneous post ICH population seen in rehabilitation settings. Second, the initial negative lower-limb ultrasound at rehabilitation admission demonstrates the inherent insensitivity of this modality for intermuscular calf thrombosis — a limitation that contributed to the delayed diagnosis of the embolic source. Third, while D-dimer normalized and CTA confirmed bilateral clearance, no long-term follow-up data beyond the point of discharge are available to assess for chronic thromboembolic complications.

In summary, patients with intracerebral hemorrhage are at a risk for developing PE, and there are currently no clear guidelines for their treatment. Active prevention is, therefore, crucial. When such conditions occur in a rehabilitation ward, rapid identification and completion of relevant examinations should be performed and a multidisciplinary collaborative model should be initiated to develop an individualized optimal treatment plan for the patient. The present article introduces the process from onset to successful diagnosis and treatment of a patient with intracerebral hemorrhage complicated by acute PE, as well as the related anatomical factors, with the aim of providing a reference for the effective prevention and treatment of this patient population.

Take-away lessons

- **The rehabilitation ward is a distinct, high-risk setting for occult PE.** Intermittent vital-sign monitoring, baseline neurological deficits masking classic symptoms, and hemiplegia-driven venous stasis create conditions in which PE can develop and go unrecognised until haemodynamic collapse occurs.
- **Atypical PE presentation must be actively considered in post-ICH rehabilitation patients.** Isolated hypotension and oxygen desaturation — without chest pain, haemoptysis, or dyspnea — can be the sole manifestation; clinicians should not require the classic triad before initiating diagnostic workup.
- **An elevated D-dimer at rehabilitation admission (here 16.25 mg/L) warrants heightened vigilance even when lower-limb venous ultrasound is negative.** Intermuscular calf thrombosis is below the sensitivity threshold of standard proximal-vein ultrasound and may be the silent embolic source.
- **Low-dose low-molecular-weight heparin (nadroparin calcium) anticoagulation may achieve complete bilateral pulmonary artery clearance in carefully selected post-ICH patients, without precipitating recurrent intracranial haemorrhage.** Maintaining APTT near baseline provides a practical safety boundary confirming that no over-anticoagulation has occurred.
- **Anticoagulation versus rebleeding is a "double-edged sword" with no guideline consensus; individualised risk stratification is therefore mandatory.** Key parameters to document before initiating anticoagulation include: days elapsed since ICH onset, hematoma size and stability on CT, and APTT baseline.
- **Multidisciplinary team consultation is essential and should be convened promptly.** Divergent departmental opinions (respiratory medicine favoured anticoagulation; neurology and neurosurgery recommended neutral/conservative treatment; cardiology raised catheter-directed thrombolysis) highlight that no single specialty holds the full risk picture in this population.
- **Serial coagulation markers (APTT, fibrinogen, D-dimer) combined with follow-up CTA provide objective, stepwise evidence of treatment response** and should be used to guide dose escalation decisions rather than relying on clinical improvement alone.
- **Early prevention is more achievable than late rescue.** Mechanical prophylaxis (compression stockings, pneumatic compression devices) should be initiated at the earliest opportunity in all post-ICH rehabilitation patients, before pharmacological anticoagulation becomes safe.

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