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CASE REPORT

Therapeutic Challenge in a Case of Extensive Pulmonary Embolism with Concomitant Intracranial Haemorrhage: When Less is Really More

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Abstract: Pulmonary embolism is a conundrum that clinicians often need to grapple with when managing patients with recent intracranial hemorrhage (ICH). A 50-year-old woman was admitted to the medical ward for left basal ganglia hemorrhage caused by a hypertensive emergency with resultant dense right hemiparesis. The ICH was managed conservatively by neurosurgical team. On day 9 of admission, the patient developed acute respiratory distress that required high-flow nasal cannula support. Her D-dimer level was increased with bedside ultrasonography revealing right heart strain patterns that warranted empirical pulmonary embolism (PE) treatment. Balancing the risk of ICH expansion and imminent respiratory collapse, she was empirically treated with intermediate dose low molecular weight heparin (LMWH) while awaiting CT pulmonary angiogram (CTPA) examination. CTPA confirmed the diagnosis of extensive PE with right lower lobe pulmonary infarction. Subsequent serial brain CT showed steady resolution of the ICH, accompanied by improvement in respiratory function. She transitioned from LMWH to apixaban two days before discharge. Overall, the favorable outcome of the modified intermediate LMWH regimen followed by maintenance apixaban in this case adds evidence to the balanced approach in the anticoagulation strategy for concurrent PE and ICH.

Keywords: anticoagulation, apixaban, intracranial haemorrhage, low molecular weight heparin, pulmonary embolism

Introduction

Pulmonary embolism (PE) is a potentially life-threatening event with an increasing incidence over the past decades.[1] The co-occurrence of PE and intracranial hemorrhage (ICH) represents a fatal duo that could augment the fatality rate of each condition. A literature review showed that the co-occurrence of ICH and acute PE increased the inpatient fatality rate to 36.6% as opposed to cohorts with PE alone, which reported an inpatient fatality rate of 8.0%.[2] Further, review of autopsy report by Stein et al on patients who died from PE, 70% were unsuspected of PE prior to their demise.[3] Hence, it is logical to assume that the actual prevalence of venous thromboembolism (VTE) and fatality rate among ICH subjects could be even higher; as the PE death might be misattributed to the direct neurological sequalae of the ICH or other aetiologies. Considering this, ICH combined with PE should be viewed as a potentially fatal combination and warrants an expedient diagnosis, as these dual pathologies would invariably augment the risk of fatality if left unattended.

Treatment of PE in the setting of ICH remains a significant therapeutic conundrum, as standard PE treatment modality revolving around systemic anticoagulant or thrombolytic therapy risks the exacerbation of existing ICH. The American Stroke Association (ASA) guidelines recommend delaying therapeutic anticoagulation for 1–2 weeks following the onset of ICH; however, the life-threatening nature of PE often precludes such delays, as postponement of treatment carries an imminent risk of irreversible obstructive shock.[4] In this context, endovascular interventional techniques have positioned catheter-directed thrombolysis (CDT) and percutaneous thrombectomy (PT) as the next viable and safer options in the management of intermediate- and high-risk patients with contraindications to systemic anticoagulation or thrombolytic therapy.[5] Yet, such advanced therapy are not widely accessible and transferring out such cases without interim treatment would subject the patient to further hemodynamic deterioration. In this vignette, we illustrate a novel and middle-ground

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approach using a modified anticoagulation strategy to manage intermediate-risk PE on day 9 of ICH, with favorable long-term clinical outcomes. The ensuing section of the discussion expounds on the possible pathomechanism of PE in ICH as well as its mitigation measures.

Case Presentation

A 50-year-old Indonesian woman with underlying hypertension was brought to our Emergency Department (ED) with reduced responsiveness following an alleged fall in the bathroom. Further history indicated that she had defaulted on antihypertensive treatment and follow-up. Upon arrival at our ED, she was afebrile with a Glasgow Coma Scale score of E4V2M6 and symmetrical pupils of 3 mm bilaterally. Her vital signs were as follows: blood pressure (BP) 196/108 mmHg; pulse rate, 71 beats/min; peripheral oxygen saturation, 98%; and respiratory rate, 20 breaths/min. Neurological examination showed that she had right hemifacial weakness and dense right hemiparesis with power graded 0/5, while power over the left upper limb was graded 4/5, and the left lower limb was graded 3/5 on the Medical Research Council (MRC) scale. Brain computed tomography (CT) revealed an acute left basal ganglia intraparenchymal hemorrhage with mass effects (Figure 1). She was diagnosed with left basal ganglia hemorrhage due to a hypertensive emergency. In the ED, the patient was reviewed by a neurosurgical team, and ICH was managed conservatively. To control her BP, she was started on intravenous infusion of labetalol targeting systolic BP below 140 mmHg and diastolic BP below 90 mmHg respectively. Subsequently, the patient was admitted to the medical ward for further stabilization.

In the medical ward, a repeat brain CT was performed the day after the initial scan, which showed an unchanged left basal ganglia hemorrhage with similar adjacent cerebral edema, mass effect, and midline shift (Figure 1). Owing to dense

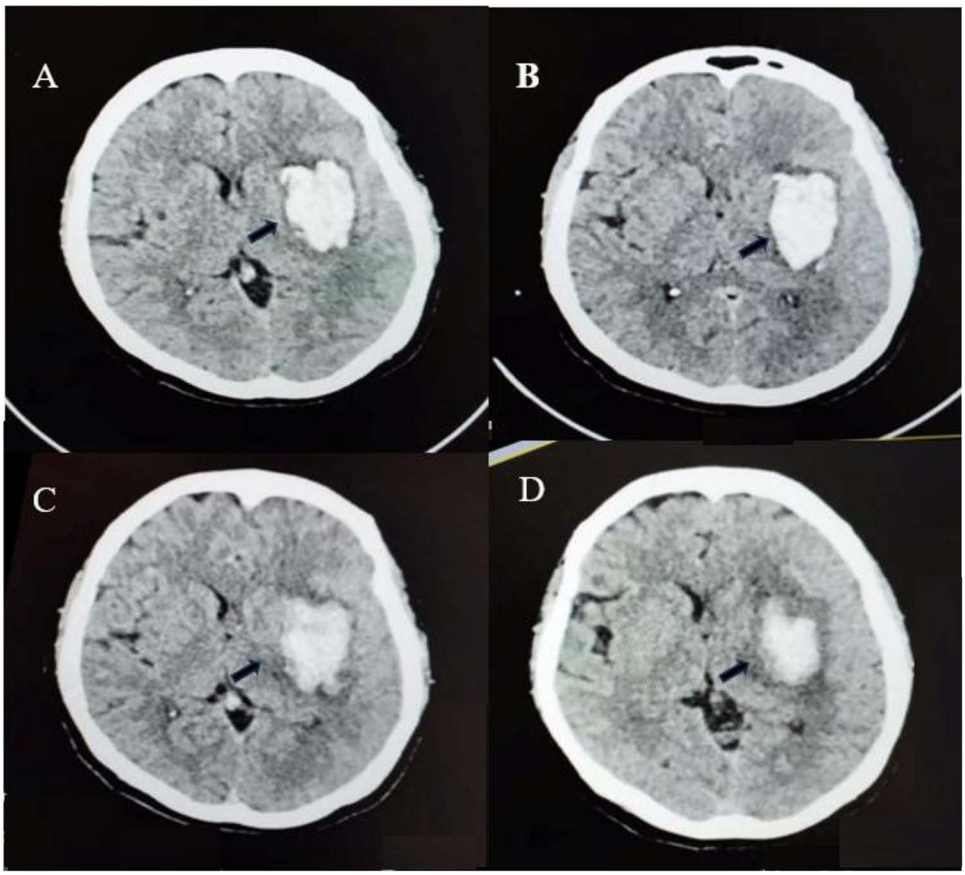

Figure 1 CT brain scans obtained at different time interval. (**A**) CT brain on day 1 showed acute left basal ganglia intraparenchymal hemorrhage (black arrow) with an estimated volume of 27.6cm^3 causing cerebral edema, mass effect, midline shift and suspicion of early obstructive hydrocephalus. There was no intraventricular haemorrhage extension. (**B**) CT brain on day 2 showed an unchanged left basal ganglia haemorrhage (black arrow) with similar adjacent cerebral edema, mass effect, and midline shift. (**C**) CT brain on day 8 before the initiation of LMWH showed improved left basal ganglia intraparenchymal hemorrhage with reduced cerebral edema (black arrow) (**D**) CT brain on day 12 showed smaller left basal ganglia bleed with improving mass effect and residual subarachnoid bleed (black arrow).

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hemiparesis, she remained bedridden during the initial days of hospitalization and required nasogastric tube feeding. She remained relatively stable until the 8th day of admission when she developed sudden hypoxemia with arterial blood gas showing type 1 respiratory failure. Clinical assessment showed that she was tachypneic, with a respiratory rate of 28 breaths per minute, requiring a face mask to deliver 5 liters of oxygen per minute. Other vital signs showed that the patient was afebrile, with a BP of 150/70 mmHg and a pulse rate of 105 bpm. The lungs were clear on auscultation, and there were no clinical signs suggestive of lower limb deep vein thrombosis (DVT). Chest radiography revealed consolidation in the right lower zone. A full septic workup was initiated, and the patient was covered for nosocomial pneumonia with intravenous (IV) piperacillin/tazobactam 4.5 g QID. Due to the abrupt onset of respiratory distress with elevated D-dimer measured 9.81µg/mL (NR<0.5 µg/mL), an urgent CT brain and CT pulmonary angiogram (CTPA) was requested to exclude acute PE and to evaluate the resolution of the ICH. The CT brain urgent done showed improved left basal ganglia intraparenchymal haemorrhage (Figure 1). However, CTPA was deferred by virtue of new-onset right-lung consolidation, suggestive of infectious causes of respiratory deterioration.

Within 6 hours, her respiratory distress worsened, and oxygen supplementation was increased to a high-flow nasal cannula (HFNC) at 50 L/min with a FiO^2 of 50%. A point-of-care ultrasound was performed, revealing evidence of right heart strain, as evidenced by a dilated right ventricle (RV) and right lower zone atelectasis. The inferior vena cava diameter was 1.43 cm and appeared well filled. A presumptive diagnosis of acute PE with an intermediate pulmonary embolism severity index (PESI) was established. An urgent family discussion was conducted to explain the benefits and risks of anticoagulation therapy to the patient. Additionally, a neurosurgical consultation was obtained, which indicated no contraindication for anticoagulation, although there was a risk of worsening ICH during anticoagulation treatment. Given the risk of ICH expansion, we opted for an intermediate dose of subcutaneous (sc) enoxaparin 40 mg BD (0.6 mg/kg; body weight 68 kg) while awaiting CTPA as she was deemed unstable for transfer to CT suite at that juncture.

The patient demonstrated a remarkable response to intermediate anticoagulation therapy, as evidenced by improved oxygenation, and no new neurological deficits were observed throughout the treatment. Notably, the patient was successfully weaned from HFNC to a Venturi mask 60% (15 L/min) the next day. Subsequently, urgent CTPA was performed, which showed extensive pulmonary artery thromboembolism with a right lower lobe pulmonary infarct (Figure 2). Echocardiography performed on day 15 of admission did not detect evidence of right heart strain, with a pulmonary artery systolic pressure of 27 mm Hg. Serum tumor markers and anti-nuclear antibodies were negative. She successfully weaned off supplemental oxygen to room air later, and a repeat CT brain scan showed a smaller left basal ganglia hemorrhage with an improved mass effect. Two days later, she was switched to apixaban 5 mg BD after 5 days of subcutaneous enoxaparin 40 mg BD. In addition to medical therapy, the patient underwent active neurorehabilitation and was eventually discharged after wheelchair ambulation. During a clinic review four months after discharge, her right hemiparesis significantly improved, with muscle strength graded as 3–4/5 on the MRC scale. The patient was able to walk without assistance. She was advised to continue taking 5 mg of tab apixaban twice daily for a total duration of six months.

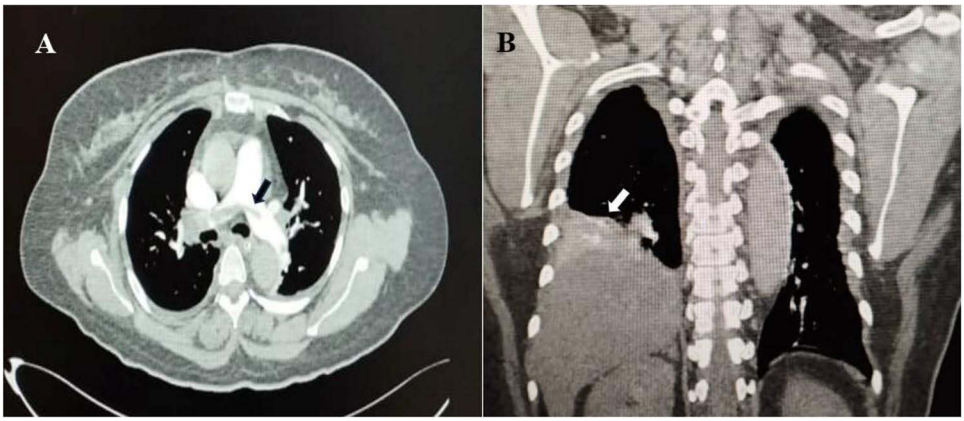

Figure 2 CT pulmonary angiogram. (**A**) Cross-sectional view showed evidence of filling defect at the bifurcation of the main pulmonary artery in keeping with saddle emboli (black arrow) (**B**) Longitudinal view showed right lower lobe pulmonary infarct (white arrow).

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Discussion

ICH with PE is a time-sensitive diagnosis. According to Stein et al, the mortality rate of acute massive PE is >50% in the first two hours of symptom onset.[3] A review of the literature showed that the onset of right ventricular failure, which characterizes submassive or massive PE as the principal driver of short-term mortality.[6] Treating a PE, especially in the presence of submassive or massive PE occurring within 14 days of ICH, remains a clinical challenge and lacks randomized controlled trial evidence. In such a conundrum, both untreated PE and anticoagulation-induced hematoma expansion would equally compound the existing disease state. The ASA guidelines recommend delaying anticoagulation treatment for 1 to 2 weeks after the onset of ICH, but the critical nature of PE may not afford patients the ability to wait weeks with an imminent risk of irreversible obstructive shock.[4] Therefore, a rational approach for intermediate-to high-risk PE with absolute contraindication to systemic anticoagulation therapy would be surgical embolectomy, PT, or CDT.[5,7,8]

With recent advances in endovascular interventional techniques, CDT and PT have emerged as less invasive options for the management of intermediate- and high-risk patients with contraindications to systemic anticoagulation or thrombolytic therapy.[5] However, the application of limited doses of thrombolytics in catheter-based approaches does not entirely eliminate the risk of bleeding complications, as the risk of major bleeding complications has been reported even with local limited thrombolytic infusion.[9–11] Extracorporeal membrane oxygenation (ECMO) therapy has also gained increased recognition as a bridging therapy for massive PE; it provides cardiorespiratory support allowing time for definitive advanced therapy to reduce thrombus burden.[12] Although the the-before-mentioned represents a major advancement and expanded armamentarium in PE treatment, such services that include cardiothoracic services are not omnipresent, limited to only a few tertiary or even quaternary hospitals. The critical decision within such an urgent timeframe is contingent on expert opinions when making a balanced decision, including modified therapeutic approach. A summary of published case reports illustrating the range of management strategies for this challenging clinical scenario is presented in Table 1.

Notably, our center lacks ECMO, interventional radiological, or cardiothoracic services at our disposal, and transferring out such cases without interim stabilization could result in progressive haemodynamic deterioration. Hence, the anticoagulation strategy was reconsidered and weighed against the risks and benefits of each viable option. On balance, we revised anticoagulation therapy as a relative contraindication, considering that serial imaging demonstrated a stable hematoma without evidence of re-bleeding and a clear source of the original bleeding with a low likelihood of rebleeding on day 9. To further mitigate the risk of hematoma expansion, a middle-ground approach was deployed by titrating LMWH to an intermediate dose. The choice of LMWH over unfractionated heparin (UFH) infusion was supported by a systematic review by Costantino et al, which reported a lower risk of major bleeding with LMWH compared with UFH infusion in VTE treatment.[18] In our case, the intermediate LMWH dose further mitigated the bleeding risk. Additionally, fixed-dose LMWH offers better pharmacokinetic predictability, lacked of need for regular laboratory monitoring and predicable onset of action in contrast to UFH infusion.[19] Overall, we believe these advantages outweigh the UFH's shorter half-life and the availability of reversible antidote, particularly when bleeding risk is substantially reduced with intermediate-dose LMWH. Evidently, intermediate anticoagulation had not only successfully treated the submassive PE, but a continuous resolution of the cerebral hematoma was also observed during the course of treatment.

It is also noteworthy that we decided to omit the lead-in dose for apixaban, defined as 10 mg BD, which should be served for seven days preceding the maintenance dose defined as 5 mg BD. This is because there is a lack of evidence regarding the safety of lead-in dose among patients with recent ICH, which was excluded from the AMPLIFY trial.[20] Also, in-patient initiation of such a modified regimen allows continuous treatment response monitoring. Kong et al similarly reported a massive PE successfully treated with a titrated dose of alteplase infusion 25 mg followed by unfractionated heparin infusions without exacerbating the existing ICH.[15] Nonetheless, it is important for the readers to appreciate that our report as well as others remain anecdotal and cannot suggest a routine approach with a similar strategy, as each case warrants detailed analysis balancing against treatment effectiveness, access to alternative therapy, and risk of re-bleeding. Overall, the successful and uncomplicated outcome of our case has proven the mantra of "less is more" when modified systemic anticoagulation therapy is used appropriately.

Table 1 Summary of Published Case Reports Describing Management Strategies and Outcomes in Patients with Coexisting Pulmonary Embolism and Intracranial Haemorrhage

Case	Sex	Age (Years)	Aetiology of ICH	Day of Onset of PE from ICH	Highest Oxygen Support	PE Phenotype	PE Treatment (Day of ICH Onset)	Clinical DVT	Outcome
1. Index case	Female	50	Hypertensive ICH	Day 8	HFNC	Submassive, intermediate-risk PE	Modified-dose anticoagulation was administered using sc Enoxaparin 40mg BD-0.6mg/kg BD (Day 9)	No*	Survive
2. Ciurylo W et al, 2022[13]	Female	80	Rivaroxaban associated ICH post-ischaemic stroke	Day 10	LFNC	Submassive, high risk PE	Percutaneous suction thrombectomy followed by placement of IVC filter (Day 11)	No*	Survive
3. Lee WC et al, 2018[14]	Male	57	Hypertensive ICH	Day 18	MV	Massive PE	UFH infusion for 3 days followed by rivaroxaban (Day 18)	No**	Survive
4. Kong CY et al, 2021[15]	Female	67	Hypertensive ICH	Day 13	MV	Massive PE	Modified-dose thrombolysis was administered using 25 mg of alteplase (Day 13)	No#	Survive
5. Mittal et al, 2020[16]	Female	70	Apixaban associated ICH	Day 8	MV	Massive PE	iNO followed by suction mechanical thrombectomy (N/A)	No*	Survive
6. Oneglia et al, 2008[17]	Male	46	Hypertensive ICH	Day 14	N/A	Massive PE	UFH infusion followed by warfarin (Day 14)	No#	Survive
7. Fukuda et al, 2006[7]	Female	57	ICH post-MVA	Day 16	MV	Massive PE	Rescue surgical embolectomy (N/A)	No*	Survive
8. Fukuda et al, 2006[7]	Female	63	ICH	Day 7	MV	Massive PE	Rescue surgical embolectomy (N/A)	No*	Survive
9. Fukuda et al, 2006[7]	Male	67	Hypertensive ICH	Day 8	MV	Massive PE	Rescue surgical embolectomy (N/A)	No*	Survive

Notes: *Ultrasound doppler vein test was not done. **Ultrasound doppler vein test and venography showed possible deep vein thrombosis (DVT) of the left leg with patent venous flow. #Deep vein thrombosis was ruled out by ultrasound doppler vein test.

Abbreviations: DVT, deep vein thrombosis; HFNC, high flow nasal canula; ICH, intracranial haemorrhage; iNO, inhaled nitric oxide; IVC, inferior vena cava; LFNC, low flow nasal canula; MV, mechanical ventilation, MVA, motor vehicle accident; N/A, not available; PE, pulmonary embolism; sc, subcutaneous; UFH, unfractionated heparin.

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Before drawing a conclusion regarding the present case, it is worthwhile to ruminate on the possible causative mechanism leading to acute PE in our case, as well as the latest VTE thromboprophylaxis guideline recommendations. Based on ASA guideline on spontaneous ICH, it states that low dose UFH or LMWH prophylaxis at 24 to 48 hours from ICH onset may be reasonable among non-ambulatory patients. In addition, intermittent pneumatic compression is recommended on the day of ICH diagnosis, while the use of graduated compression stockings of knee high or thigh-high length alone is no longer considered beneficial in VTE prophylaxis.[4] In practice, adoption of such a recommended strategy was not observed among published cases as well as in our case.[13,15,17] The disparity between guideline recommendation and real-world practice should be examined, especially on the risk-benefits of early chemical thromboprophylaxis when the specter of re-bleeding is invariably present among clinicians. Hence, knowledge, attitude, and practice surveys would be useful in analyzing the determinants of VTE prophylaxis among clinicians treating ICH.

The absence of clinically detected DVT among published extensive PE cases, including this case, challenges the traditional view of migratory embolus as the presumptive etiology of PE (Table 1).[13–17] Herein, we propose a few plausible explanations for VTE among ICH patients, in addition to the classical view of clot migration theory. First, reduced chest motility and respiratory excursions among ICH patients with depressed consciousness and prolonged bed stays are believed to contribute to a prothrombogenic state due to pulmonary circulation stasis. This mechanism is analogous to lower limb DVT among immobilized patients, especially when considering pulmonary circulation occurring in low-pressure high-capacitance vessels similar to the lower limb, and pulmonary arteries at rest have either absent or low vascular tone.[21] Additionally, the brain is rich in tissue factor, a major initiator of the coagulation cascade, and neuronal injury during ICH leads to the release of procoagulant phospholipids and increased tissue factor activity into the cerebrospinal fluid, contributing to a systemic prothrombotic state that may predispose to thrombotic events such as pulmonary embolism.[22] Furthermore, stroke-induced sympathetic overactivity and inflammation can initiate a cascade of events that damage the endothelium, thereby predisposing the pulmonary vasculature to in-situ thrombosis.[23] Also, the initial clotting event has a downward spiral effects caused by a mal-adaptive self-propelling cycles of sympathetic bias, inflammation and coagulation fueling vasoconstriction that may exacerbate pulmonary vascular stasis and clot propagation.[23] Henceforth, it is conceivable that pulmonary thrombosis in ICH is far more nuanced and represent an interplay of multiple factors in addition to the traditional view of a dislodged migratory clot theory. Future studies are needed to explore the teleological basis of VTE disorders, which could lead to additional preventive strategies, such as early pulmonary rehabilitation and sympathetic blockade during acute stroke.

Conclusion

PE in ICH is a vexing clinical scenario, in which the risk of bleeding and thrombotic sequelae must be carefully considered during therapeutic decisions. It would be even more difficult to manage intermediate- and high-risk PE within a resource-limited center, which prohibits long-distance transfer to another institution with alternative advanced interventional therapy. This case demonstrates the use of a modified dose of LMWH as a potential therapeutic option with favorable clinical outcomes for the treatment of submassive PE with recent ICH. Further studies are needed to compare therapeutic options and outcomes in patients with concomitant PE and ICH.

Ethics Statement

This case report has obtained approval from National Medical Research Registry (NMRR), Ministry of Health Malaysia: NMRR ID-25-03628-UYD. The Medical Research Ethics Committee (MREC) waived the requirement for ethical approval, as this work constituted a case report.

Consent for Publication

Informed consent for publication was obtained from the patient's sister, as the patient lacked decision-making capacity due to an acute haemorrhagic stroke.

Acknowledgments

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Disclosure

The authors report no conflicts of interest in this work.

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