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From Intracerebral Hemorrhage to Pulmonary Embolism: A Management Dilemma

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Abstract **Case Report**

The occurrence of a pulmonary embolism after a cerebral hemorrhage presents a major challenge for clinicians. On one hand, pulmonary embolism is a potentially life-threatening complication that requires treatment with anticoagulants and/or thrombolytic agents, but on the other hand, these treatments carry a heightened risk of recurrent hemorrhage, which is especially concerning in this context. To date, there are no specific guidelines for managing such complex situations. Through this clinical case, we demonstrate that a combination of heparin therapy followed by treatment with direct oral anticoagulants appears to be a safe and effective strategy for managing intermediate-risk pulmonary embolism after a recent cerebral hemorrhage, pending the development of new management approaches in the future.

Keywords: Intracerebral Hemorrhage, Pulmonary Embolism, Direct Oral Anticoagulants, Hypertension.

INTRODUCTION

The occurrence of a pulmonary embolism is a common complication after spontaneous intracerebral hemorrhage (ICH), with a cumulative incidence ranging from 0.5% to 5% [1]. Its management presents a clinical dilemma when it occurs early because the treatment of pulmonary embolism requires the use of anticoagulants or thrombolytics to limit the risk of complications and death. However, this treatment exposes the patient to the risk of hematoma expansion, which can lead to a poor prognosis [2]. Most similar studies have examined the initiation or resumption of anticoagulant therapy in patients with atrial fibrillation and found a highly variable time frame to minimize hemorrhagic complications, ranging from 2 to 8 weeks depending on the studies [3, 4]. We report the case of a 45-year-old man with uncontrolled hypertension who presented with bilateral pulmonary embolism 15 days after a spontaneous intracerebral hemorrhage and was treated at the Omar Bongo Ondimba Military Teaching Hospital in GABON.

CASE REPORT

This is a 45-year-old male patient with a history of hypertension, poorly controlled despite dual therapy, who was admitted to the neurosurgery department for left hemiplegia related to a spontaneous right lenticulo-insular cerebral hemorrhage (Figure 1).

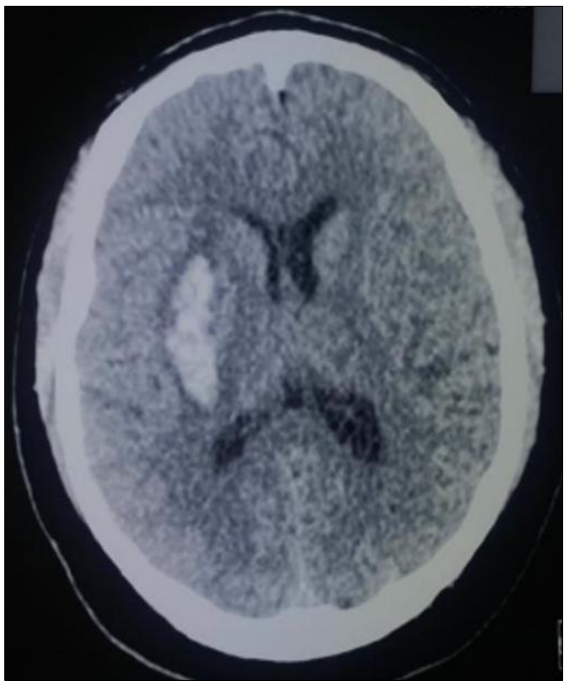

Fig. 1: brain CT: Right lenticulo-insular cerebral hemorrhage

Fifteen days after his hospitalization, he developed acute respiratory distress with tachycardia. The clinical examination revealed a blood pressure of 150/90 mmHg, a heart rate of 122 bpm, and an oxygen saturation of 89% on room air. The patient was transferred to an intensive care unit where an electrocardiogram showed sinus tachycardia at 120 bpm with left ventricular hypertrophy (LVH) and negative T waves in the anteroseptal region. A venous Doppler ultrasound of the lower limbs revealed left femoral deep vein thrombosis (figure 2), and a chest CT angiography confirmed the presence of bilateral pulmonary embolism (Figure 3).

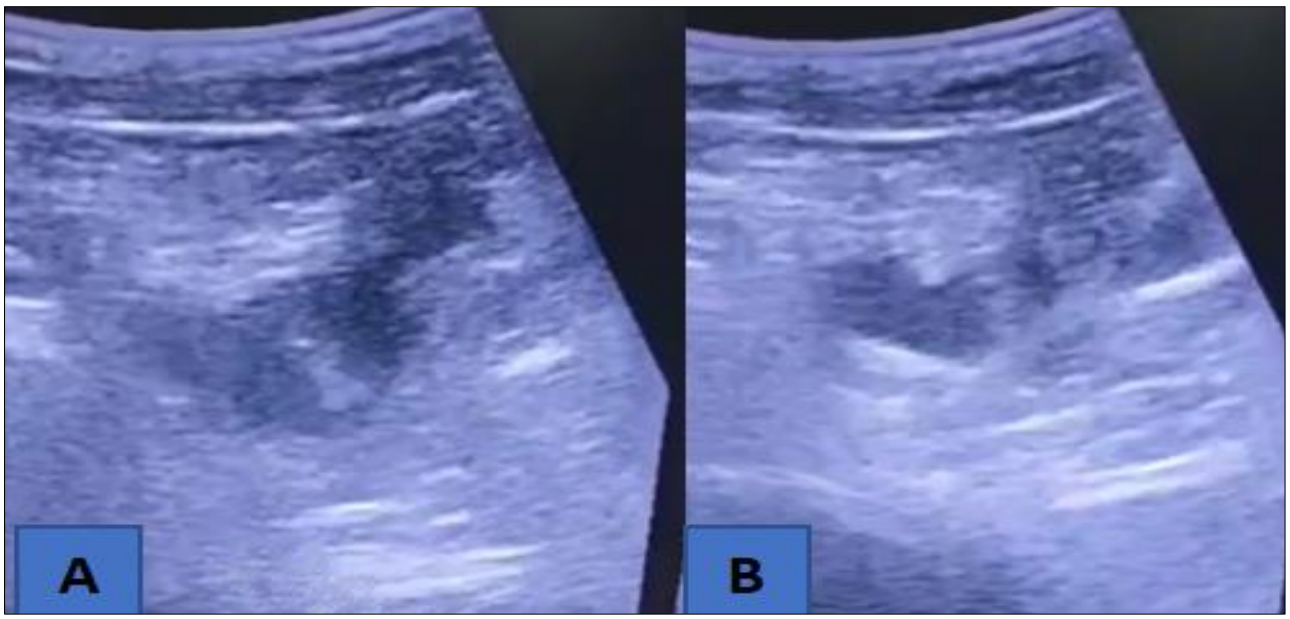

Fig. 2: Venous Doppler ultrasound: deep femoral venous thrombosis (A) non-compressible(B)

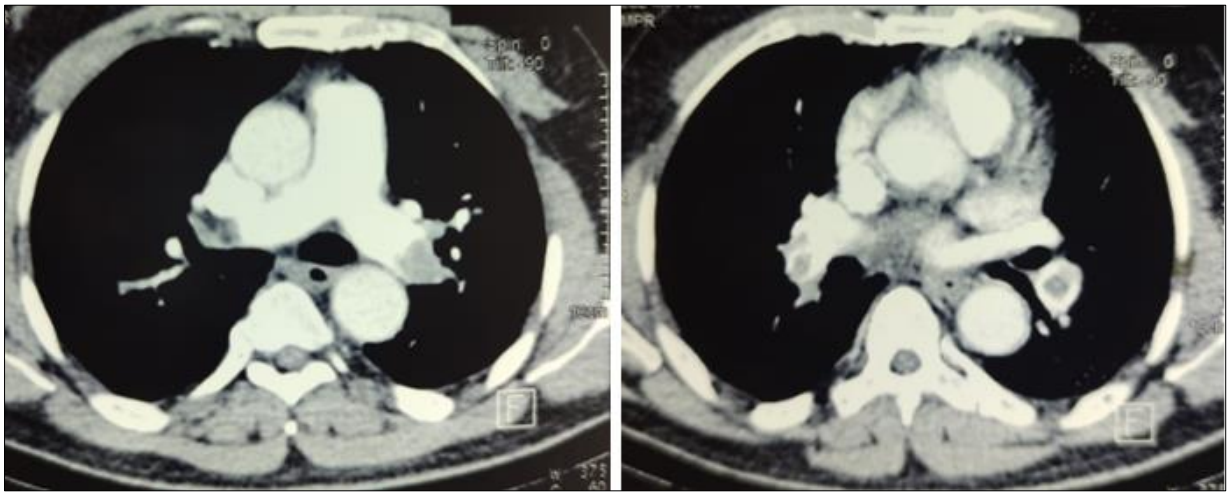

Fig. 3: Chest CT angiography: bilateral pulmonary embolism

Transthoracic echocardiography showed a moderately dilated right ventricle with preserved systolic function and a pulmonary artery systolic pressure (PASP) of 50 mmHg. Troponin and pro-BNP levels were positive, and the patient's simplified Pulmonary Embolism Severity Index (sPESI) score was calculated to be 2, indicating an intermediate-low risk of pulmonary embolism. The bleeding risk, according to the RIETE (Registro Informatizado de Enfermedad TromboEmbólica) score, was estimated at 3.5, corresponding to a moderate risk.

Given this clinical presentation, it was decided to start the patient on enoxaparin 8000 IU every 12 hours, combined with 3L/min oxygen therapy and close monitoring. After 7 days of treatment, the patient stabilized respiratory-wise, with good oxygen saturation on room air and no neurological worsening. The anticoagulation was then transitioned to rivaroxaban 20 mg once daily, and the patient remained hospitalized. He also received physiotherapy sessions and his antihypertensive therapy was adjusted from dual to triple therapy.

After 10 days, the patient showed significant recovery from his hemiplegia, was able to walk with the assistance of a walking frame, and his blood pressure was well controlled. Before discharge, a follow-up brain CT scan was performed, which showed that the hematoma was liquefying (figure 4).

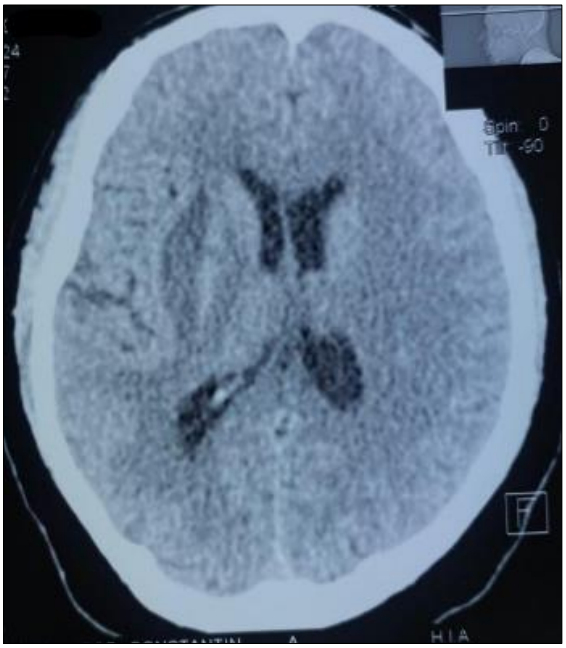

Fig. 4: Brain CT: liquefaction of the hematoma

DISCUSSION

Venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), are serious complications after ICH. The overall rate of symptomatic VTE after ICH ranges from 2% to 5% [5]. A previous report of patients with ICH demonstrated a prevalence of 2% for PE and 1% for DVT [6]. PE is a life-threatening condition requiring treatment with anticoagulants or even thrombolytic agents in high-risk cases. However, anticoagulants and thrombolytic agents are contraindicated after a recent hemorrhagic episode. The treatment of a life-threating PE in the setting of a recent ICH presents a difficult problem. In the literature, there is no clear consensus on the initiation of anticoagulant therapy within one month after a hemorrhagic stroke. Most studies have focused on anticoagulation after hemorrhagic stroke in patients with atrial fibrillation. For instance, in a retrospective study conducted by Pennlert *et al*., in Sweden [7], involving 2,679 survivors of hemorrhagic stroke with atrial fibrillation, it was found that initiating anticoagulant therapy 7 to 8 weeks after the event minimized the risk of hemorrhagic complications while providing clear benefits in preventing ischemic events. A recent meta-analysis by Huang *et al*., [8], conducted on patients with atrial fibrillation, suggested a timeframe between 2 weeks and 1 month for starting anticoagulation. However, there was a slight increase in hemorrhagic complications, particularly in patients treated with vitamin K antagonists. Wei-Chie Lee [9], reports a case similar to ours in which a patient presented with high-risk bilateral pulmonary embolism 18 days after a hemorrhagic stroke, successfully treated with unfractionated heparin and direct oral anticoagulants, leading to a favorable outcome. Oneglia [10], reports a similar case in a hypertensive patient who was treated with unfractionated heparin followed by oral warfarin, also resulting in a favorable outcome.

Our therapeutic approach followed the same logic, focusing on the use of low-molecular-weight heparin in the presence of an intermediate-risk pulmonary embolism, and direct oral anticoagulants (DOACs) due to their ease of use and, most importantly, their lower risk of bleeding.

As venous thromboembolism (VTE) is independently associated with poor outcomes in patients with intracerebral hemorrhage (ICH), prophylaxis is crucial. One early study demonstrated that, without prophylaxis, 75% of ICH patients with hemiplegia developed deep vein thrombosis (DVT), and pulmonary embolism (PE)–related death occurred in approximately 5% of these patients [11].

Intermittent pneumatic compression devices have been widely used in the hyperacute phase of intracerebral hemorrhage (ICH) for venous thromboembolism (VTE) prophylaxis. International guidelines, including those from the American Heart Association/Stroke and the European Stroke Organization, recommend their use [12, 13]. However, studies assessing the efficacy of pharmacological prophylaxis after intracerebral hemorrhage (ICH) have yielded conflicting results. A network meta-analysis showed no relationship between prophylaxis and the occurrence of venous thromboembolism (VTE) (OR, 0.93 [95% CI, 0.19–4.37]) [14], while an earlier meta-analysis found that anticoagulant chemoprophylaxis initiated between 1 and 6 days after admission resulted in a significant reduction in the risk of pulmonary embolism (1.7% versus 2.9%, relative risk, 0.37 [95% CI, 0.17–0.80]) [15]. Several guidelines, including those from the American Heart Association/Stroke [12], recommend the use of low-dose chemoprophylaxis after hematoma stability has been demonstrated. However, its implementation in real-world practice remains surprisingly low.

CONCLUSION

The management of pulmonary embolism after intracerebral hemorrhage represents a true therapeutic challenge due to the increased risk of thromboembolic recurrence and hemorrhagic complications. Although there is no clear consensus on the optimal timing for initiating anticoagulation, a personalized approach is essential to balance the risks and benefits. A combination of heparin therapy followed by treatment with direct oral anticoagulants appears to be a safe and effective strategy for managing intermediate-risk pulmonary embolism

after a recent cerebral hemorrhage, pending the development of new management approaches in the future.

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