

Acute Pulmonary Embolism Following Hemorrhagic Stroke

Madhu Gupta,¹ Maheshwar Prasad,¹ Achutanand Lal,¹ Jeetendra Mishra,¹ Imran Ali,¹ Parash Bhatta,¹ Salina Khadka¹

¹Department of Cardiovascular, National Medical College-Teaching Hospital, Birgunj, Parsa, Nepal.

Received: 15th July, 2023

Accepted: 19th August, 2023

Published: 31st December, 2023

ABSTRACT

Venous thromboembolism results from extended immobilization following intracerebral hemorrhage. Massive pulmonary embolism in association with right heart failure is a life-threatening condition and requires treatment with anticoagulants or thrombolytic agents. There are no guidelines for treatment of pulmonary embolism with recent hemorrhagic stroke and these medicines are contraindicated, as they may induce farther hemorrhage.

Keywords: pulmonary embolism; intracranial hemorrhage; newer oral anti-coagulant.

INTRODUCTION

Massive PE is a life-threatening condition and requires emergent thrombolysis with thrombolytic agents followed by anticoagulant. Thrombolytic and anticoagulants are contraindicated after a recent hemorrhagic episode. A treatment difficulty exists in cases with life-threatening pulmonary embolism (PE) and venous thromboembolism (VTE) following recent hemorrhagic stroke. Clinicians must decide which anticoagulants to use in patients who have had a recent cerebral hemorrhage. We report a case of Massive pulmonary embolism with right heart failure and shock following recent intracranial hemorrhage without surgical intervention. A protocol of unfractionated heparin infusion and non-Vitamin K antagonist oral anticoagulants (NOAC) was used with good outcome, and did not affect in secondary hemorrhagic events, or recurrent thromboembolism.

Case Report

A 56 years old man, known for hypertension, type -2 diabetes mellitus presented with sudden onset of shortness of breath and chest pain for several hours. On examination at our emergency his systolic blood pressure was 70 mmHg, heart rate was 120 beats/min/regular with spo2 92% with O2 @ 5l/min on face mask. There was B/L crepitations on chest auscultation. Electrocardiography showed incomplete RBBB with tachycardia and D-dimer was 18.6 mg/l (fibrinogen equivalent units). Transthoracic echocardiography showed a dilated right ventricle (RV) with poor RV function (tricuspid annular plane systolic excursion)-

(TAPSE) <10MM HG), and RV to left ventricle (LV) ratio was greater than 1.2. Acute kidney injury with serum creatinine 2.6mg/dl was noted. Chest computed tomography (CT) pulmonary angiogram showed bilateral pulmonary trunk embolism as seen in (Figure 1).

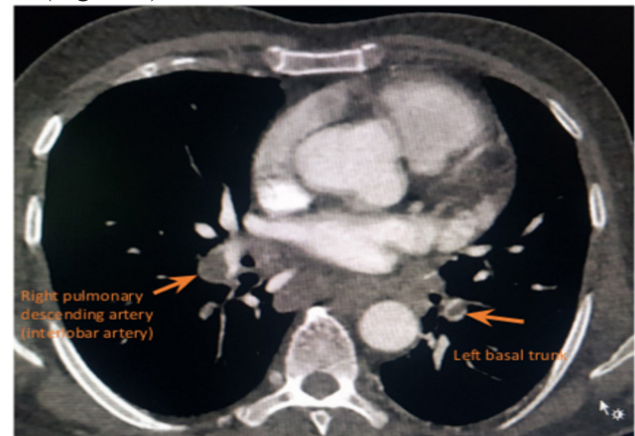


Figure 1. CT Pulmonary angiogram: shows non-enhancing intraluminal central as well as eccentric

There is increased in RV to LV ratio (1.2) suggestive of right ventricular dysfunction (Figure 2).

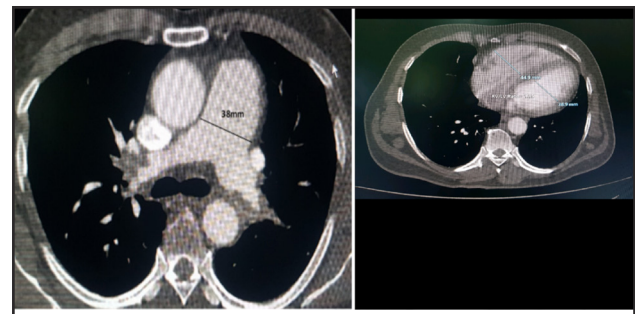


Figure 2. CT Angiogram with Increased RV to LV ratio (1.2) noted suggestive of Acute Pulmonary Embolism.

Correspondence: Dr. Madhu Gupta, Department of Cardiology, National Medical College, Teaching Hospital, Bahuwari, Birgunj, Parsa, Nepal. Email: madhugupta.md@gmail.com, Phone: +977-9851174042.

Color Doppler study of the left leg showed heterogeneous (<50%) echogenic areas within the lumen of the common femoral vein, a superficial vein extending up to the popliteal vein of the left leg which is completely non-compressible and shows no color flow nor any venous spectral flow likely Deep Vein Thrombosis (DVT) type II. The patient had a medical history of a right sided hemiparesis, 6 weeks prior to this episode as shown in (Figure 3).

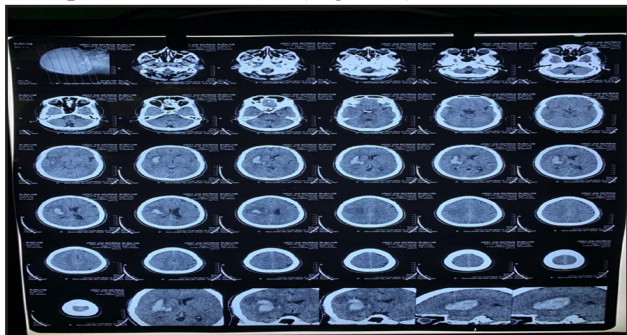


Figure 3. Intraparenchymal hemorrhage involving right centrum, lentiform nucleus and external capsule with midline shift.

The diagnosis of acute pulmonary embolism was established and patient was shifted to the ICU. Adequate hydration along with intravenous dopamine and noradrenaline was started for circulatory support as well to manage for the acute kidney injury and PE related right ventricular failure. Due to recent ICH, systemic thrombolysis was not suitable for this patient, thus unfractionated heparin (UFH) was considered and activated partial thromboplastin time (APTT) was maintained 50 to 70 seconds. Patient's hemodynamic improved after 24 hours of treatment, and no new neurological signs were detected. The patient's renal function also returned to normal. We started high doses of NOAC (Rivaroxaban) 15 mg twice a day for 3 weeks as per pulmonary embolism guideline. We did not consider warfarin because of the high risk of ICH compared to NOAC. An inferior vena cava (IVC) filter for DVT was not implanted because of lack of facilities; and the patient was on anticoagulant and patent venous flow.

DISCUSSION

Current guidelines recommend thrombolytic therapy for cases of massive pulmonary embolism (PE) without any contraindications. However, thrombolysis is contraindicated in the presence of a recent hemorrhagic stroke or other conditions.¹ ICH is the most severe form of stroke, and survivors suffer high rates of functional disability, resulting in an increased likelihood of thromboembolic complications such as DVT, PE, and myocardial infarction.² A previous report of patients with ICH demonstrated a prevalence of 2% for PE and 1% for DVT.³

In the large registry recording patients with AF, the optimal timing of restarting treatment was 7 to 8 weeks after ICH. Oral anticoagulants were better than antiplatelet agents, due to the lower incidence of recurrent ischemic events, as well as higher survival benefit. Another meta-analysis describing the restarting of anticoagulants following ICH, was associated with a lower risk of thromboembolic complications, and a similar risk of ICH recurrence.⁴ However, no large randomized studies have been performed for anticoagulants use in the patient with life-threatening VTE following ICH. Despite the indication of use of thrombolytics in case of massive pulmonary embolism in cardiogenic shock, our team decided to manage with UFH (2Xcontrol) infusion followed by newer oral anticoagulant as per the pulmonary embolism guideline. PE is an uncommon but serious medical complication after a hemorrhagic stroke. It is associated with higher risk of death, worse disability, higher in-hospital complications, and longer LOS. The risk of PE is higher in older patients with more severe strokes, history of cancer or DVT/PE, or DVT during admission. Early recognition of predisposing conditions may help implement strategies for its early detection and the appropriate preventative measures aimed at improving the quality of stroke care.

CONCLUSIONS

The combination of UFH infusion and newer oral anticoagulant drugs can be used to manage life-

threatening PE following recent ICH. The use of NOAC is a safer strategy if the patient has a previous history of major ICH. Further investigations and

controlled trials for uncommon cases of ICH and acute PE are still needed.

REFERENCES

1. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galiè N, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. *Eur Heart J*. 2014 Nov 14;35(43):3033–69.
2. Goldstein JN, Fazen LE, Wendell L, Chang Y, Rost NS, Snider R, et al. Risk of Thromboembolism Following Acute Intracerebral Hemorrhage. *Neurocrit Care*. 2009;10(1):28–34.
3. Lee LH. DOACs – advances and limitations in real world. *Thromb J*. 2016 Oct 4;14(1):17.
4. Murthy SB, Gupta A, Merkler AE, Navi BB, Mandava P, Iadecola C, et al. Restarting Anticoagulant Therapy after Intracranial Hemorrhage A Systematic Review and Meta-Analysis. *Stroke*. 2017 Jun;48(6):1594–600.

Citation: Gupta M, Prasad M, Lal A, Mishra J, Ali I, Bhatta P, Khadka S. Clinical Profile and Etiology of Patients with Pericardial Effusion. *JNHLS*. 2023; 2(2):114-16.