



Acute Massive Pulmonary Embolism During Craniotomy: A Case Report

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Abstract

Background: Venous thrombosis is a common complication among critically ill patients with intracerebral hemorrhage, which may lead to pulmonary embolism.

Case Presentation: In this report, we present a case of a man who was diagnosed with left basal ganglia region hemorrhage, hypertension, and venous thromboembolism. Considering the risks, including rebleeding and the expansion of the hematoma, he did not receive any anticoagulation after onset. The thrombus located in the pulmonary artery was found by transesophageal echocardiography after he suffered cardiac arrest during the craniotomy. Additionally, a thrombus attached to the central venous catheter was observed by ultrasound. Unfortunately, he died of PE without receiving any effective treatment.

Conclusions: This case emphasizes the importance of prophylactic and therapeutic strategies for thromboembolic events among critically ill populations. It also underscores the critical role of perioperative ultrasound.

Keywords

Craniotomy, Pulmonary Embolism, Transesophageal Echocardiography, Catheter Related Thrombosis, Calf Muscle Venous Thrombosis

Introduction

Acute massive pulmonary embolism (PE) is a life-threatening condition, with a significant number of deaths occurring within 1 hour. Therefore, rapid diagnosis and treatment are crucial. Guidelines recommend the use of transesophageal echocardiography (TEE) in cases where unexplained life-threatening circulatory instability persists despite corrective therapy during non-cardiac surgery [1]. In this report, we present the case of an elderly man who was diagnosed with massive pulmonary embolism after experiencing cardiac arrest during a craniotomy,

as detected by TEE. The emboli causing the pulmonary embolism may have originated from calf muscle venous thrombosis (CMVT) and/or catheter-related thrombosis (CRT). Despite the high risks of venous thromboembolism among critical populations, the optimal timing and approach to anticoagulation and antithrombotic therapy for patients with contraindications or risk factors remain unclear. Unfortunately, the patient died from PE due to the absence of effective antithrombotic therapy in the context of continuous cardiopulmonary resuscitation.

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Case Presentation

A 59-year-old man experienced a sudden loss of consciousness and fell down. He was taken to a local hospital where a brain computed tomography (CT) scan confirmed the diagnosis of intracranial hemorrhage. He was subsequently placed on mechanical ventilation via a tracheal cannula and received mannitol infusion through a central venous catheter (CVC). The patient had a history of untreated primary hypertension and a thirty-year history of smoking and drinking. After three days, he remained in a continuous coma and was transferred to our hospital's intensive care unit (ICU).

Upon admission to the ICU, the patient was unconscious, with both pupils measuring 2mm in diameter and showing a dull light reflection. His Glasgow Coma Scale score was 5. His blood pressure (BP) was 190/121mmHg, heart rate (HR) was 78 beats per minute (bpm), and pulse oxygen saturation (SPO₂) was 100%. A double-lumen CVC had been inserted into his right internal jugular vein (RIJV), with one lumen being nonfunctional. A CT scan confirmed the presence of a hematoma in the left basal ganglia region (Fig-1).



Fig-1:
Brain CT Scan Showing a Hematoma on the Left Basal Ganglia Region

Laboratory tests revealed a hemoglobin level of 144g/L, a white blood cell count of 15.64×10^9 /L, a platelet count of 327×10^9 /L, a prothrombin time of 11.1 seconds, an activated partial prothrombin time of 27.7 seconds, a fibrin degradation product level of 5.2, and a D-dimer level of 3.25. Arterial blood gas analysis (ABG) showed a pH of 7.5, a PCO₂ of 31.7mmHg, a PO₂

of 137mmHg, an HCO₃⁻ level of 24.9, an SPO₂ of 100%, and a lactic acid level of 2.7mmol/L.

Transthoracic echocardiography (TTE) and cerebral angiography results were normal. However, ultrasound of the blood vessels in the extremities revealed bilateral calf muscle venous thrombosis (CMVT). Then the patient was diagnosed with left basal ganglia region hemorrhage, hypertension, and venous thromboembolism (VTE).

In addition to mannitol, the patient received continuous infusion of urapidil hydrochloride to treat hypertension. Due to the risk of hematoma expansion, the intensivist made the decision to avoid anticoagulants. While the patient's vital signs remained stable within the first 24 hours of admission, there was no significant improvement in his level of consciousness.

However, several hours later, the patient's hemodynamics became unstable. His systolic blood pressure dropped to 90mmHg, and his heart rate increased to 110bpm. As a result, the intensivist discontinued the urapidil hydrochloride infusion and initiated norepinephrine infusion through the central venous catheter (CVC) to maintain hemodynamic stability. The repeated arterial blood gas analysis (ABG) results are shown in **Table-1**.

Due to the understanding that severe intracranial pressure (ICP) can potentially result in hemodynamic collapse, the neurosurgeon determined that a craniotomy was necessary. The patient was transferred to the emergency operating room, where the following vital signs were observed: blood pressure (BP) of 85/50mmHg, heart rate (HR) of 102 bpm, pulse oxygen saturation (SPO₂) of 100%, end-tidal carbon dioxide (ETCO₂) level of 18mmHg, and a norepinephrine infusion at 0.2ug/kg/min.

After the arterial line was inserted, a bedside arterial blood gas (ABG) analysis was performed (**Table-1**) prior to the induction of anesthesia. Anesthesia was induced using cisatracurium, sufentanil, and propofol, followed by the use of sevoflurane and remifentanil for maintenance. During the procedure, when the surgeon

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opened the endocranium, they discovered severe encephaledema and weak pulsations. They proceeded to evacuate the blood clot, but no fresh clot was found.

Two hours later, despite receiving 3000ml of volume therapy and a 2000ml succinyl gelatin injection, the patient's hypotension did not improve. Urinary output was only 100ml. The norepinephrine infusion was increased to 0.45ug/kg/min. At the end of the operation, the patient's blood pressure decreased to 68/42mmHg, heart rate decreased to 30bpm, with SPO₂ at 97% and ETCO₂ at 23mmHg. The ABG results are shown in **Table-1**. Following the administration of a 10ug intravenous injection of epinephrine, the blood pressure increased to 100/60mmHg, and the heart rate increased to 100bpm.

Unfortunately, 20 minutes later, the patient experienced a cardiac arrest, and chest compressions were started. The anesthesiologist performed a transesophageal echocardiography (TEE) scan to investigate the cause of the cardiac arrest. The TEE imaging revealed an embolus located in the main

pulmonary artery, as well as in the right and left pulmonary arteries (**Fig-2**). The definitive cause of the cardiac arrest was determined to be acute massive pulmonary embolism (PE). Furthermore, the anesthesiologist identified an embolus attached to the central venous catheter (CVC) by scanning the neck (**Fig-3**), suggesting that the embolus resulted in PE from catheter-related thrombosis (CRT) and/or calf muscle venous thrombosis (CMVT).

Despite continuous chest compressions and repeated infusions of epinephrine over the course of one hour, the patient did not achieve return of spontaneous circulation (ROSC).

Discussion

The direct cause of death was PE for this patient, and the embolus may have originated from the lower extremity venous vessel and/or central venous catheter (CRT). The three risk factors for venous thromboembolism include hypercoagulability, blood stasis, and vascular endothelial injury. The incidence of PE in intensive care units is 2%, and the average

Table-1: The Arterial Blood Gas Analysis Before and During Operation

Time (FiO ₂)	In ICU -0.4	Before induction(1.0)	Operation start(0.5)	End of operation(0.7)	CPR -1
ETCO ₂ (mmHg)	/	18	22	23	21
PH	7.44	7.46	7.21	7.24	6.98
PO ₂ (mmHg)	72	352	81	191	49
PCO ₂ (mmHg)	33	34	57	56	105
HCO ₃ ⁻ (mmol/L)	23	25	23	24	25
BE (mmol/L)	2	2	-5	-3	-7
SaO ₂ (%)	100	100	93	99	60

Abbreviations: FiO₂, fraction of inspired oxygen; CPR, cardiopulmonary resuscitation



Fig-2:

In the mid-esophageal pulmonary artery long-axis view of TEE. A, the thrombus was in the pulmonary artery (arrow). B, the thrombus was in the right pulmonary artery (arrow). C, the thrombus was in the opening of the left pulmonary artery (arrow). TEE: transesophageal echocardiography, PA: pulmonary artery

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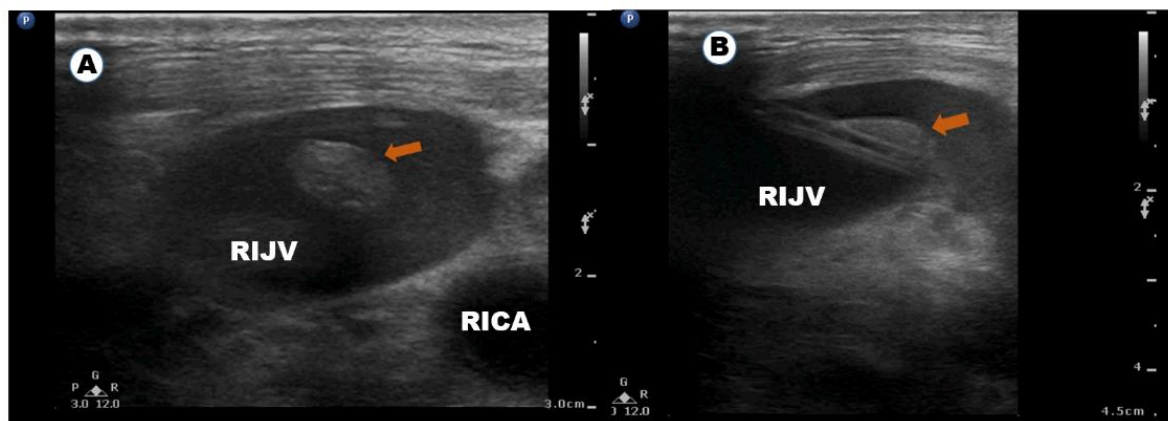


Fig-3:

The ultrasonic scanning of the right internal jugular vein. A, short-axis scanning, the thrombus was in the RIJV (arrow). B, long-axis scanning, the thrombus attached to the catheter (arrow). RIJV: right internal jugular vein; RICA: right internal carotid artery

duration of occurrence is 8 days after admission. Predictive factors for PE include acute medical illness, length of hospital stay, presence of meningeal hemorrhage, spine fracture, hypoxemia with a $\text{PaO}_2/\text{FiO}_2$ ratio <300 , absence of pharmacological prevention of venous thromboembolism, tumor, activated partial thromboplastin time (APTT), and platelet count [2,3]. This patient had several risk factors without any chemoprophylaxis. The formed embolus traveled through the bloodstream and eventually blocked the pulmonary artery. The clinical manifestation of PE depends on the extent of obstruction in the pulmonary artery, where more than 50% obstruction may lead to shock and circulatory collapse [4].

The classic clinical presentations in anesthetized patients with mechanical ventilation include decreased SPO_2 , precipitous decrease in ETCO_2 , hypotension, tachycardia, and a right heart strain pattern on electrocardiogram [5]. This patient developed hypotension 24 hours after admission to the ICU and required vasoconstrictor infusion to manage blood pressure changes, which might indicate sub-massive PE rather than increased intracranial pressure (ICP). Severe hypotension was difficult to treat with volume resuscitation and vasoconstrictors. Transesophageal echocardiography (TEE) scan revealed an embolus in the pulmonary artery after cardiac arrest, and **Figure 2** confirmed the presence of massive PE. According to previous studies, the exact location of PE was only detected in 26% of patients with severe PE.

Ultrasound is not the preferred method for diagnosing PE compared to CT angiography and ventilation-perfusion scan, but point-of-care ultrasound is a valuable diagnostic modality for anesthesiologists in the operating room, as it avoids the need to transfer the patient and interrupt the surgical procedure [5,6]. Right ventricle dilation is the most common finding on TEE after PE, but at least 30% obstruction of the pulmonary vasculature is required to produce right ventricle dilation. This must be differentiated from right ventricular infarction, chronic PE, and pulmonary heart disease with pulmonary hypertension [6,7]. If TEE cannot identify the embolus in the pulmonary artery for highly suspected patients, other common echocardiographic findings associated with PE can be assessed, such as RV hypokinesis, RV/LV diastolic diameter ratio >0.7 , McConnell's sign, septal shift, tricuspid regurgitation, etc. [6,8].

Both the size of the embolus and the patient's underlying cardiopulmonary function determine the outcome after PE. The presence of shock is associated with higher mortality, and most deaths occur within an hour, highlighting the importance of prompt diagnosis and resuscitation during this critical time [7]. In cases of massive PE, thrombolysis leads to rapid improvement in hemodynamics and reduces mortality compared to anticoagulation alone. However, for patients with contraindications to anticoagulation and thrombolysis, surgical embolectomy and catheter-based therapy are viable options [9]. A retrospective study showed that catheter-based therapy reduced the

number of recurrent PE cases compared to the medical group, but the mortality rate in the surgical group was lower than that in the interventional therapy group [10]. However, data on safety and efficacy are limited, and randomized trials and long-term observations are lacking. Therefore, the choice of treatment depends on the patient's clinical status, bleeding risk, local expertise, and available devices [5,11,12]. In cases where ROSC cannot be achieved after initial chest compression, ECMO (extracorporeal membrane oxygenation) plays a crucial role as a bridging therapy before thrombolysis [13,14].

The incidence of central venous catheter-related thrombosis (CRT) is 2%-4%, and approximately 15% of individuals with CRT-related upper extremity deep vein thrombosis (DVT) develop PE [15]. CRT can not only cause vessel and catheter blockages but also lead to infections and PE. The incidence of CRT varies depending on patient characteristics, catheter-related factors, and the catheter insertion process. Ultrasound guidance is useful in reducing the number of attempts during catheter insertion [16], and the optimal timing for catheter removal remains uncertain. Current guidelines recommend removing the catheter only if it is no longer functional or necessary. Previous studies have shown that appropriate anticoagulation therapy can reduce the risk of CVC obstruction, recurrent DVT, or DVT extension [16]. Therefore, regardless of whether the catheter is removed or not, anticoagulant therapy should be initiated once CRT occurs.

Prior to the finding of CRT, bilateral calf muscle vein thrombosis (CMVT) was identified during the patient's ICU stay. Due to concerns about hematoma expansion, the physician opted against anticoagulation and only implemented mechanical prophylaxis. The rate of PE associated with calf vein thrombosis ranges from 0% to 5.8%, and no studies have reported deaths resulting from PE [17]. The European Society for Vascular Surgery's 2021 clinical practice guidelines on the management of venous thrombosis recommend making decisions regarding anticoagulation for patients with calf DVT based on symptoms and risk factors for progression and bleeding [18]. The reported incidence of PE in isolated CMVT cases varies

widely, and the optimal type and duration of anticoagulation have not been established in the existing literature. However, a recent systematic review and meta-analysis showed that in patients with intracerebral hemorrhage, pharmacologic thromboprophylaxis significantly reduced the risk of pulmonary embolism without an increase in rebleeding, hematoma enlargement, major disability, or death [19].

The pulmonary thrombus in this case could have originated from either CMVT or CRT, but it is more likely to have originated from CRT. Due to the patient's severe brain injury, the multidisciplinary team decided against thrombolytic therapy. Additionally, the patient did not achieve return of spontaneous circulation (ROSC) after one hour of chest compression, and there were limitations preventing transfer to the catheterization room or availability of emergency extracorporeal membrane oxygenation (ECMO) at the institution. This case highlights the importance of screening patients with high-risk factors, particularly those with obstructed central venous catheters. The risks and benefits of anticoagulation after thrombus formation need to be carefully evaluated. Massive PE can cause sudden hemodynamic collapse in patients, and transesophageal echocardiography (TEE) plays a critical role in perioperative management by aiding in diagnosis.

Author Contributions

XH and RW conceived the study, XH collected the data and performed the imaging, XH and YC wrote the paper, and RW and TY supervised the study and revised the paper. Additionally, all authors read and approved the final manuscript.

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Availability of Data and Materials

The data used to support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This ethics approval was waived.

Consent for Publication

Written informed consent for publication for the data was waived.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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