CASE REPORT

Resuscitated patient after simultaneous intravascular thrombolytic therapy for massive pulmonary embolism and embolization of an injured hepatic artery : a case report

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Abstract : We report a case of successful management of a massive pulmonary embolism (PE) with simultaneous hepatic arterial injury using anticoagulation and interventional radiology (IVR). The patient, with pre-existing muscle atrophy, decreased lower extremity mobility, and spinal stenosis, developed bilateral PE during rehabilitation. Following cardiopulmonary arrest, chest compressions for resuscitation resulted in hepatic hemorrhage. The patient was treated with intravenous heparin, transcatheter arterial embolization, thrombectomy, and blood transfusion, leading to full recovery without neurological complications. This case highlights the importance of assessing PE risk in hospitalized patients and highlights the efficacy of IVR in complex cases. J. Med. Invest. 72 : 177-181, February, 2025

Keywords **:** *Pulmonary embolism, spinal stenosis, interventional radiology, hepatic injury, resuscitation*

INTRODUCTION

Massive pulmonary embolism (PE) with concurrent right heart failure and systolic blood pressure <90 mmHg caries a high mortality rate, with >50% of deaths occurring within 90 days of onset and >90% occurring within the first 10 days (1). Current guidelines recommend anticoagulation, thrombolysis, surgical thrombectomy, and percutaneous catheter-based interventions as standard treatments for PE (2, 3). However, the first-line treatment for massive PE, which is particularly lethal, remains under debate (4). Systemic anticoagulation and thrombolytic therapy, while critical, increase the risk of bleeding, potentially leading to serious adverse events. Here, we report a case of simultaneous massive PE and hepatic arterial injury successfully treated with a combination of anticoagulation and interventional radiology (IVR) procedures.

CASE REPORT

The patient was a 92-year-old woman (body mass index, 30.7 kg/m^2) with a history of lumbar spinal stenosis (surgery 17 years prior), neurogenic bladder (managed with a balloon catheter for three years), osteoporosis, hypertension, and diabetes mellitus. She was initially admitted for treatment of a urinary tract infection and lower limb weakness. One week after admission, she complained of sudden chest pain during rehabilitation, and her systolic blood pressure and transcutaneous oxygen saturation dropped to less than 60 mmHg and 70%, respectively. Arterial blood gas analysis revealed an arterial partial pressure of oxygen (PaO_2) of 35.7 mmHg (Table 1). Bedside cardiac echocardiography showed right heart enlargement and left ventricle flattening and narrowing, and PE with right-sided heart failure was suspected. During an emergency computed tomography (CT) scan for a definite diagnosis, the patient lost consciousness (Glasgow Coma Scale : E1V1M1) and experienced cardiopulmonary arrest with asystole. Chest compressions were initiated, and one milligram of adrenaline was administered. The heartbeat of the patient resumed after three minutes, and her level of consciousness improved to E2V4M4. Contrast-enhanced CT scan revealed bilateral PE, rib fractures, and liver injury (Fig. 1). No thrombus was identified in the femoral vein, but the vessel diameters were irregular on both sides.

The hematoma was localized around the liver, and given the severity of the pulmonary embolus, thrombolysis was prioritized. Heparin (80 units/kg) was administered as a single infusion, according to the Guidelines for the Diagnosis, Treatment, and Prevention of Pulmonary Thromboembolism and Deep Vein Thrombosis (JCS 2017) (5). Continuous infusion of heparin was initiated at 18 $units \cdot kg^{-1} \cdot h^{-1}$, adjusted to achieve a 1.5–2.5-fold increase in activated partial thromboplastin time (APTT). The intra-abdominal hematoma expanded, revealing extensive bleeding from the medial regional branch (A4) of the left hepatic artery. Therefore, transcatheter arterial embolization with GelFoam was performed, targeting the bleeding hepatic artery (Fig. 2). Simultaneously, local administration of 120 000 units of urokinase, and thrombectomy with a pigtail catheter was performed in the bilateral pulmonary arteries (Fig. 3). The X-ray fluoroscopy showed that the contrast agent flowed smoothly which meant the successful thrombus removal. Following the IVR procedure, the oxygenation of the patient, which had been 80% SpO_2 on 15 L/minute of oxygen via a reservoir bag, improved to 99% on 12 L/minute of oxygen. Four units of red blood cell concentrate were transfused to address hemorrhage-related hemoglobin loss. Due to fluid volume-dependent hypotension, the patient was treated with a high-volume infusion (14 bottles of 500 mL saline per day), 100 mg hydrocortisone, and continuous

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Table 1. Laboratory data at the onset of pulmonary embolism

Assessment	Results	Reference range
Arterial blood gas		
pH	7.32	7.35-7.45
$PaCO_2$	41.5 mmHg	35-46
PaO_2	35.7 mmHg	70-100
HCO_3-	20.9 mmol/L	21-26
O_2 saturation	64.8 %	92.0-98.5
Lactate	3.47 mmol/L	0-1.8
Complete blood cell count		
White blood cell count	84.6 $x10^2$/μl	33.0-86.0
Red blood cell count	388 $x10^4$/μl	386-492
Hemoglobin	11.3 g/dl	11.6-14.8
Hematocrit	36.6 %	35.1-44.4
Platelet count	32.4 $x10^4$μl	15.8-34.8
Coagulation fibrinolysis examination		
Prothrombin time (PT)	9.2 sec	9.8-12.1
PT activity	136.5 %	70.0-130.0
PT-international normalized ratio	0.85	0.84-1.40
Activated partial thromboplastin time	30.2 sec	25-38
D-dimer	28.8 μg/ml	0-0.5
Blood chemistry		
Total bilirubin	0.42 mg/dl	0.4-1.5
Albumin	2.7 g/dl	4.1-5.1
Total protein	6.1 g/dl	6.6-8.1
Alkaline phosphatase	63 U/L	38-113
Asparate aminotransferase	16 U/L	13-30
Alanine aminotransferase	10 U/L	7-23
Lactate dehydrogenase	148 U/L	124-222
Cholinesterase	175 IU/L	201-421
Gamma-glutamyl transpeptidase	15 U/L	9-32
Urea nitrogen	12.4 mg/dl	8.0-20.0
Creatinine	0.55 mg/dl	0.46-0.79
Uric acid	3.8 mg/dl	2.6-5.5
Creatine kinase	28 U/L	41-153
Na (serum)	140.7 mmol/L	138-145
K (serum)	4.3 mmol/L	3.6-4.8
Cl (serum)	108.3 mmol/L	101-108
C-reactive protein	3.41 mg/dl	0.00-0.14
Blood glucose	192 mg/dl	73-109
Estimated glomerular filtration rate	75.32	-
Estimated creatinine clearance	72.12 ml/min	-
Troponin T	(-)	(-)

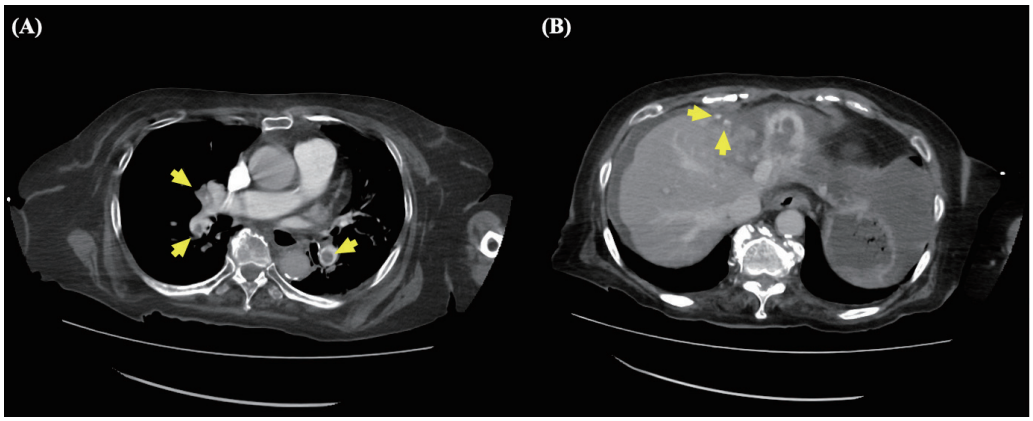

Figure 1. Contrast CT images showing massive PE (A) and hematoma around the liver (B). Arrowheads in (A) indicate blood clots. Arrowheads in (B) indicate bleeding spots in the liver.

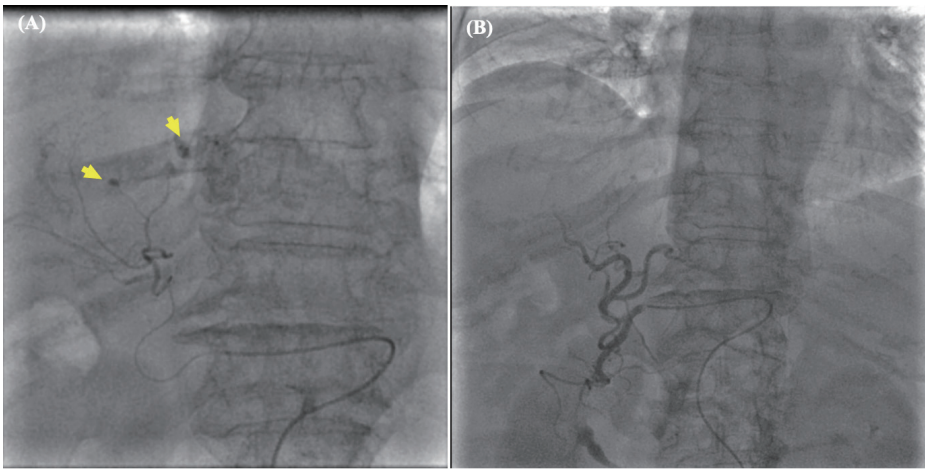

Figure 2. Digital subtraction angiography before (A) and after (B) transcatheter arterial embolization (TAE) using GelFoam on the injured hepatic artery.
Arrowheads in (A) indicate bleeding spots in hepatic artery A4. Panel (B) shows the interruption of contrast flow at the base of A4.

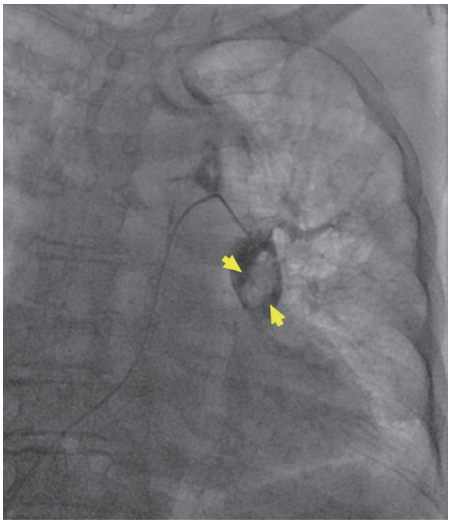

Figure 3. Digital subtraction angiography before thrombectomy of the PE with a pigtail catheter. Arrow heads indicate the massive PE.

dopamine to manage the circulation of the patient. By the end of the day, her consciousness improved to E4V4M6. The following day, SpO_2 was 95% on 3 L/minute of oxygen. As her APTT exceeded 600 s, heparin was discontinued the next day, and coagulability was controlled with edoxaban. As the urinary output of the patient was maintained at 1–2 L/day with furosemide, and blood pressure was stable, dopamine tapering was completed the day after IVR. Blood transfusions and intravenous fluids were discontinued after two weeks of administration. There were no signs of post-resuscitation encephalopathy, such as cognitive decline or paralysis. She continued rehabilitation for approximately two months and was discharged from the hospital and admitted to a nursing facility.

DISCUSSION

In this case, the patient developed PE during rehabilitation, one week after admission. The PE was probably caused by long-term muscle atrophy in the lower extremities due to lumbar spinal stenosis, which contributed to the formation of deep vein thrombosis (DVT) from prolonged bed rest. The risks of perioperative DVT and PE complications during spinal surgery have been widely reported (6-8). A high incidence of thromboembolism has been reported in patients with paralysis of the lower extremities due to acute spinal cord injury (9-11). However, the risk of DVT and PE in patients more than 10 years post-spinal surgery, or in those with gradually progressive muscle atrophy without complete paralysis, remains inadequately studied. The patient had overlapping risk factors for thrombosis, including obesity, advanced age, and prolonged bed rest. Despite these risks, preventive measures, such as anticoagulation or elastic stockings, were not implemented because the patient had no prior history of thrombosis, and no surgery was planned. A recent retrospective cohort study using the Japanese receipt database indicated a higher incidence of PE in hospitalized patients, suggesting that hospitalization itself may be a risk factor. However, prophylactic treatment for DVT is less frequently administered in patients not scheduled for surgery during their hospital stay (12). This case emphasizes the need for thorough assessment of DVT and PE risk and the implementation of preventive measures, regardless of whether surgery is planned.

A thrombus lodged at the base of the bilateral pulmonary arteries can lead to cardiac arrest, requiring prompt thrombectomy. Although bleeding from the ruptured liver was observed after CPR, systemic anticoagulation was initiated, with thrombolytic therapy prioritized as the first treatment step. Upon contrast administration for endovascular treatment, the hematoma enlarged markedly, prompting hepatic arterial embolization before treating PE. Controlled bleeding allowed a more aggressive treatment regimen involving simultaneous local thrombolysis and PE fragmentation. Previous reports described the need for extracorporeal cardiopulmonary resuscitation and extracorporeal membrane oxygenation in patients with PE and cardiogenic shock (13, 14). However, in the present case, prompt IVR stabilized the respiratory and circulatory status of the patient without requiring such multidisciplinary treatments, even after cardiac arrest. A 2020 retrospective cohort study of PE-related cardiac arrest showed higher mortality in patients with older age, female sex, diabetes mellitus, end-stage renal disease, and hypothermia, which are considered prognostic determinants (15). Although this patient met all three of these criteria and experienced hypothermia due to massive rehydration, prompt improvement in oxygenation might have led to resuscitation and prevented the onset of post-resuscitation encephalopathy. One reason for a good prognosis was normal renal function of the patient. Despite receiving 7 L of saline in one day, the patient recovered without any exacerbation of cardiac or renal failure. The most important factor was the decision to perform catheter-based thrombectomy in addition to pharmacologic thrombolysis. At 92 years old, the family of the patient declined aggressive life-prolonging treatment, including invasive respiratory support with intubation upon admission. Therefore, in the absence of hepatic hemorrhage, invasive IVR may not have been selected, as pharmacologic thrombolysis alone might have failed to restore cardiopulmonary function. A Scientific Statement from the American Heart Association also recommends the addition of interventional therapy to systemic anticoagulation against massive PE, unless contraindicated (16). This case demonstrated the effectiveness of IVR in managing massive PE. Furthermore, combining IVR thrombolytic therapy with transcatheter hemostasis proved beneficial for a patient at high risk of bleeding.

CONFLICT OF INTEREST

There authors declare no conflict of interest.

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PATIENT CONSENT

Informed consent was obtained for patient information to be published in this article.

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