Multiple cardiac arrests induced by pulmonary embolism in a traumatically injured patient
A case report and review of the literature

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Abstract
Rationale: Pulmonary embolism-induced cardiac arrest should not be given up arbitrarily, knowing that the etiology of pulmonary embolism is reversible in most cases.

Patient concerns: We present a case of continuous resuscitation lasting approximately 4 hours, during which 21 episodes of cardiac arrest occurred in a 46-year-old man who sustained high-level paraplegia after a road traffic accident.

Diagnoses: Multiple cardiac arrests induced by pulmonary embolism.

Interventions: The patient received cardiopulmonary resuscitation and thrombolytic therapy.

Outcomes: The patient was discharged in 2 weeks when his condition turned for the better.

Lessons: Cardiopulmonary resuscitation of patients with pulmonary embolism-induced cardiac arrest should not be given up arbitrarily, knowing that the etiology of pulmonary embolism is reversible in most cases. Effective external cardiac compression can not only save the patient's life but also attenuate neurological sequelae. Thrombolytic therapy is the key to the final success of resuscitation.

Abbreviations: BP = blood pressure, ECG = electrocardiography, HR = heart rate, RR = respiratory rate.

Keywords: pulmonary embolism, recurrent cardiac arrest, traumatically injury

1. Introduction

Pulmonary embolism is a causative factor of potentially reversible shock and cardiac arrest, which may cause acute elevation of pulmonary arterial pressure and right ventricular pressure, resulting in quick circulation failure. Treatment of acute pulmonary embolism depends on the severity of the clinical symptoms and signs. The severe form of pulmonary embolism is characterized by cardiac arrest or severe hemodynamic instability. Less than 5% cases of pulmonary embolism progressed to cardiac arrest, with a fatality rate of 65% to 90%.[1–3] Cardiac arrest can occur within several hours. Approximately 5% to 13% cases of cardiac arrest with unknown reasons are related to pulmonary embolism.[4,5] It is difficult to make a diagnosis of pulmonary embolism in patients with cardiac arrest because the clinical symptoms and signs of severe pulmonary arrest are not specific. Conventional risk factors of thrombosis, prodromal symptoms of dyspnea, and respiratory distress may all suggest cardiac arrest from pulmonary embolism.[4,6]

2. Case report

Mr Xi, a 46-year-old farmer from Qingzhou (Shandong, China), was admitted on the evening of January 16, 2017 after a road traffic accident, causing loss of sensation in both lower extremities at the time of admission. The patient had been healthy previously without a remarkable family history. Physical examination on admission showed temperature 37°C, heart rate (HR) 80/min, respiratory rate (RR) 17/min, and blood pressure (BP) 114/70 mm Hg. He was conscious but unable to move his lower extremities. There was no sensation below the xiphoid process and in both lower extremities. The muscle tension of the lower extremities was decreased and muscle strength was 0; Achilles tendon reflex of both knees, patellar and ankle clonus, and bilateral Babinski sign were negative (−). Bulbocavernosus reflex and perianal reflex were positive (+). Head CT on January 17, 2017 showed subarachnoid hemorrhage. Chest MRI suggested T8 fracture, T7/8 intervertebral disk herniation, and T7/8 and T8 spinal cord edema, which were considered the result of thoracic spinal contusion. A diagnosis of spinal vertebral fracture and dislocation with paraplegia and traumatic subarachnoid hemorrhage was made. On January 19, 2017, posterior decompression of thoracic spine + bone graft fusion + internal fixation was performed.

On the morning of February 7, 2017, the patient suddenly fainted and lost consciousness for about 1 minute, accompanied with shortness of breath and profuse sweating, when SpO$_2$ was 88%, HR 99 bpm, RR 34/min, and BP 85/46 mm Hg. No dry and
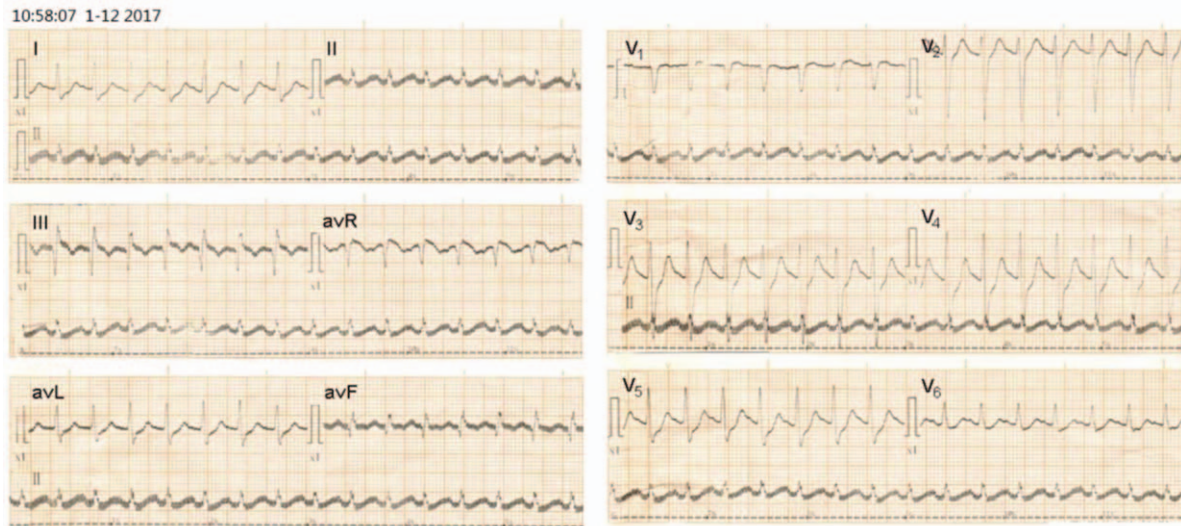
Figure 1. Electrocardiograms at the time of initial admission to the ICU.

wet rale was audible. Emergency blood glucose was 6.4 mmol/L. After intravenous (IV) dripping of dopamine (10 μL/min/kg) and 500 mL 0.9% sodium chloride, BP rose to 102/62 mm Hg and SpO$_2$ to 93%. Other clinical parameters were as follows: D-dimer 6001 ng/mL (reference 0–500), WBC 18.14×10^9/L, RBC 4.33×10^{12}/L, Hb 121 g/L, PLT 267×10^9/L, CK-MB 8u/L, PT 10.4 s (reference 8.8–13.8), APTT 31.70 s (reference 26.0–42.0), INR 0.94 (0.8–1.2), and FIB 1.53 g/L. The patient was then transferred to the ICU as an emergency case of "shock to be investigated and pulmonary embolism to be ruled out." At the time of ICU admission, the patient was confused, with BP 65/43 mm Hg and SpO$_2$ 78%, and therefore oral endotracheal intubation respirator-assisted breathing was initiated promptly, together with dopamine with noradrenaline for maintaining BP, and 500 mL hydroxyethyl starch for volume expansion. At the same time, IV access was set up. After 30 minutes, the respirator parameters were as follows (under the PCV mode): P 15 cmH$_2$O, PEEP 0 cmH$_2$O, FIO$_2$ 100%, SpO$_2$ 80%, and BP 80/60 mm Hg. Emergency subclavian IV access was set up to monitor CVP dynamically, showing CVP >32 cmH$_2$O. Recheckup examination showed D-dimer 4996 ng/mL, PH 7.50, PCO$_2$ 32 mm Hg, PO$_2$ 63 mm Hg (pure oxygen inhalation via the respirator), blood lactic acid 2.0 mmol/L, and AB/HCO3$^-$ 5 mmol/L. Bedside Color Doppler ultrasound showed enlargement of the right atrium and

pulmonary arterial pressure 38 cmH$_2$O with no significant deep vein thrombosis (DVT) in both lower extremities. Bedsides, chest X-ray showed increased lung markings and a small amount of pleural effusion. ECG showed QIII TIII change and oblique depression of V3-6 ST segments (Fig. 1).

At 14:10 of February 7, 2017, the patient developed cardiac arrest all of a sudden, for which external cardiac compression was applied and 1 mg adrenalin was injected IV every 3 to 5 minutes. After 15-minute resuscitation, the patient's spontaneous circulation was returned. After a multidisciplinary discussion about the case within the hospital, thrombolytic therapy was decided on by giving the patient 50 U urokinase IV at 15:20, followed by common heparin at a dose of 600 U/h. In <10 minutes after the thrombolytic therapy, the patient developed cardiac arrest again, for which external cardiac compression was applied and 1 mg adrenalin was injected IV. After about a resuscitation cycle (2 minutes), the spontaneous circulation was returned. Subsequently, cardiac arrest occurred at an approximately 10-minute interval, for which the above resuscitation was repeated. At approximately 16:00, 100 U urokinase was administered IV again. At approximately 18:30, the patient's spontaneous circulation was returned again. A total of 21 episodes of cardiac arrest occurred during this period, and no electric defibrillation or cardioversion was applied. Details of ECG monitoring during cardiac arrest are shown in Figures 2–4.
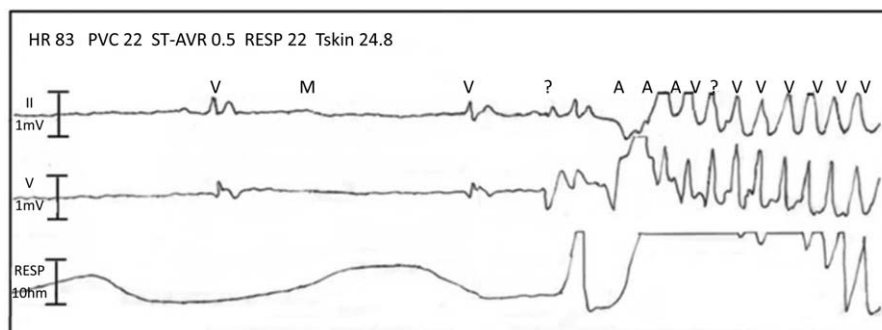

Figure 2. Anterior segment: pulseless electrical activity; posterior segment: at the time of cardiac compression, similar to ventricular tachycardia.
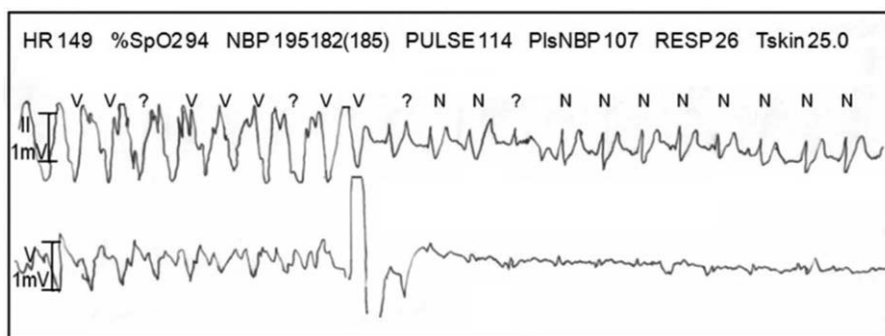
Figure 3. Anterior segment: at the time of cardiac compression; posterior segment: after restoration of spontaneous circulation.

On February 8, 2017, morning ward rounds revealed macroscopic hematuria and decreased Hb, for which concentrated RBCs were transfused to correct anemia, in addition to anti-infection and nutritional supportive therapies. The patient developed massive bleeding of the digestive tract and hemorrhagic shock on the very night. After discontinuation of the heparin therapy, plasma and concentrated RBC transfusion, and administration of proton pump inhibitors (PPIs), the condition was improved. The patient was discharged in 2 weeks after his condition turned for the better.

3. Discussion

This was a paraplegic patient who had been confined to bed for 22 days. Posttraumatic coagulation dysfunction is an important risk factor for pulmonary embolism. When circulation failure occurs in such patients, pulmonary embolism should be considered. As such patients are poorly responsive to fluid resuscitation, large amounts of pressor agents are often required to maintain BP. Even though Doppler ultrasound may not indicate the presence of emboli, pulmonary arterial hypertension still suggests the presence of embolism. According to the British Thoracic Society guidelines, the presence of wedge-shaped shadows induced by bleeding or infarction in the lungs on echocardiographs is a reliable reference for the clinical diagnosis of massive pulmonary embolism.[7] However, it was not performed in this patient due to the technical limitations of our hospital. QIII TIII change on electrocardiography (ECG) is also an indicator of embolism. Changes in ECG can exclude cardiogenic shock from acute coronary syndrome. As there was

no significant fluid and blood loss, hypovolemic shock was excluded. In addition, distributive shock is likely to occur in paraplegic patients. But as patients with hypovolemic shock are usually well responsive to fluid therapy and vasoconstrictive drugs, it could also be excluded in this patient. The most probability was obstructive shock. As Doppler ultrasound did not find pericardial effusion and chest X-ray did not detect pneumothorax in our case, we suspected it as obstructive shock from pulmonary embolism. In such a case, repatency treatment of the pulmonary vessel is critical. But as the patient in our case suffered from traumatic cerebral hemorrhage not long ago, thrombolytic therapy was contraindicated. How to select the treatment became a dilemma. Let us have a review of the literature concerning this issue. According to 2010 American Heart Association for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care,[8] it is appropriate to use thrombolytic agents in patients with cardiac arrest that is suspected or confirmed as being caused by pulmonary embolism (class II, LOE B). The use of cellosolve can help improve the success of resuscitation in patients with pulmonary embolism. Although thrombolytic therapy runs a high risk of hemorrhage, it can improve the success rate of resuscitation of cardiac arrest from pulmonary embolism, and help long-term recovery of the neurological function. The 2015 American Heart Association for Cardiopulmonary Resuscitation guidelines recommend the prompt use of thrombolytic therapy in patients with cardiac arrest that is confirmed as being caused by pulmonary embolism.[9] Surgical or percutaneous mechanical removal of the thrombi, pulmonary reopening (class IIa LOE C-LD), and thrombolytic therapy are also beneficial to patients who have
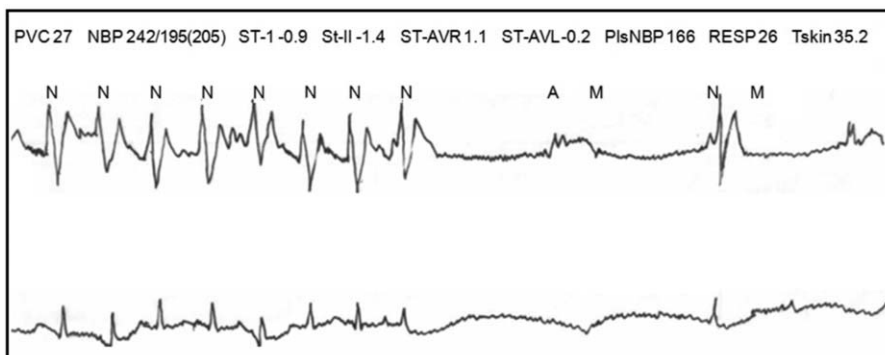

Figure 4. Anterior segment: electrical activity after restoration of spontaneous circulation; posterior segment: pulseless electrical activity.

received external cardiac compression (class IIa LOE C-LD). Thrombolytic therapy can be applied promptly and more positively in patients whose cardiac arrest is highly suspected as being caused by pulmonary embolism (class IIa LOE C-LD).

Pulseless electrical activity (PEA) can be observed in 36% to 53% patients with pulmonary embolism-induced cardiac arrest, whereas major electrical shock rhythms are rarely seen.[4,10,11] In the present case, Philips Electrocardiograph Monitor detected quick occurrence of PEA (Figs. 2–4). As circulation was returned in a short time after application of external cardiac compression, it is prudent to recommend positive defibrillation. Defibbrilation can be considered only when rhythms are confirmed as shockable. To patients with severe pulmonary embolism, thrombolytic therapy alone is not enough; opening the blood vessel through thrombolysis or surgery is the treatment of choice.[1,12] There are individual studies reporting the successful treatment of patients with severe pulmonary embolism-induced cardiac arrest by surgical or percutaneous mechanical removal of the thrombi.[13–15] However, most hospitals are not equipped with such conditions. In our patient, there was no direct evidence of pulmonary embolism. Although pulmonary embolism was highly suspected, it was difficult to perform CTA and MRI on the patient because of the unstable circulation. Bedsides, ultrasonography was the only accessible diagnostic method for the patient. Seeing that the patient was contraindicated for thrombolytic therapy and the hospital lacked necessary technical conditions of surgery at the time, we invited doctors from the departments of respiration and cardiology to have a discussion on the treatment strategy. After obtaining informed consent and support from the family, we finally decided on thrombolytic therapy for the patient.

The experience with our successful treatment of the present case suggests that although thrombolytic therapy runs a high risk, it can be used to save the life of some patients with pulmonary embolism-induced cardiac arrest, although this decision-making process may delay the initiation of thrombolytic therapy. Knowing that pulmonary embolism is a reversible etiology, cardiopulmonary resuscitation should not be given up arbitrarily. Effective cardiac compression can not only save the patient's life but also reduce the neurological sequelae. Another important factor that may affect survival of such patients is thrombolytic therapy-associated complications. In the present case, resuscitation was continued for approximately 4 hours, during which 21 episodes of cardiac arrest occurred. On the heel of the thrombolytic therapy came massive bleeding of the digestive tract and hemorrhagic shock. Fortunately, all these complications were overcome successfully with a satisfactory clinical outcome.

References

[1] Kucher N, Rossi E, De Rosa M, et al. Massive pulmonary embolism. Circulation 2006;113:577–82.

[2] Kasper W, Konstantinides S, Geibel A, et al. Management strategie and determinants of outcome in acute major pulmonary embolism: result of a multicenter registry. J Am Coll Cardiol 1997;30:1165–71.

[3] Janata K. Managing pulmonary embolism. BMJ 2003;326:1341–2.

[4] Kürkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as cause of cardiac arrest: presentation and outcome. Arch Intern Med 2000;160:1529–35.

[5] Hess EP, Campbell RL, White RD. Epidemiology, trends, and outcome of out-of-hospital cardiac arrest of non-cardiac origin. Resuscitation 2007;72:200–6.

[6] VandenHoek TL, Morrison LJ, Shuster M, et al. Part 12: cardiac arrest in special situations: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122(18 Suppl. 3):S829–61.

[7] British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group. British Thoracic Society guidelines for the management of suspected acute pulmonary embolism. Thorax 2003;58:470–84.

[8] Lavonas EJ, Drennan IR, Gabrielli A, et al. Part 10: Special circumstances of resuscitation: 2015 American heart association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2015;132(18 Suppl. 2):S501–18.

[9] Courtney DM, Kline JA. Identification of prearrest clinical factors associated with outpatient fatal pulmonary embolism. Acad Emerg Med 2001;8:1136–42.

[10] Courtney DM, Kline JA. Prospective use of a clinical decision rule to identify pulmonary embolism as likely cause of outpatient cardiac arrest. Resuscitation 2005;65:57–64.

[11] Comess KA, DeRook FA, Russell ML, et al. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. Am J Med 2000;109:351–6.

[12] Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. Chest 2002;121:877–905.

[13] Konstantinov IE, Saxena P, Koniuszko MD, et al. Acute massive pulmonary embolism with cardiopulmonary resuscitation management and results. Tex Heart Inst J 2007;34:41–5.

[14] Doerge HC, Schoendube FA, Loeser H, et al. Pulmonary embolectomy: review of a 15-year experience and role in the age of thrombolytic therapy. Eur J Cardiothorac Surg 1996;10:952–7.

[15] Fava M, Loyola S, Bertoni H, et al. Massive pulmonary embolism: percutaneous mechanical thrombectomy during cardiopulmonary resuscitation. J Vasc cInterv Radiol 2005;16:119–23.