


Right ventricular blowout rupture complicating cardiopulmonary resuscitation in a patient with acute pulmonary embolism

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DESCRIPTION

A 79-year-old man collapsed during gait rehabilitation in the hospital on the ninth day after burr-hole evacuation for chronic subdural haematoma. The medical emergency team was called and arrived 2 min later. The patient regained consciousness, but reported of dyspnoea and showed facial pallor with a feeble radial pulse. His blood pressure and oxygen saturation were unmeasurable. The ECG showed irregular tachyarrhythmia with complete right bundle branch block, and point-of-care transthoracic echocardiogram revealed right ventricular dilatation with a paradoxical septal motion at end systole. Based on these clinical findings, acute pulmonary embolism was suspected. Just 28 min after the collapse, the patient deteriorated to pulseless electrical activity, and manual cardiopulmonary resuscitation (CPR) was initiated.

He was transferred to the catheterisation laboratory, continuing chest compression with a mechanical device (LUCAS2; Physio-Control Operations, Scanfil Åtvidaberg, Åtvidaberg, Sweden). After 30 min of CPR, the establishment of venoarterial extracorporeal membrane oxygenation (ECMO) led to termination of chest compression. Subsequent pulmonary angiography demonstrated massive thrombi bilaterally in the main pulmonary arteries. Although aspiration mechanical thrombectomy successfully retrieved most of the thrombi, the ECMO flow was unstable at 1.0–1.7 L/min with his haemoglobin level lowered from 13.0 mg/dL to 4.7 mg/dL. Massive left intrathoracic fluid accumulation was identified by repetitive bedside ultrasonography. Massive blood transfusion for acute

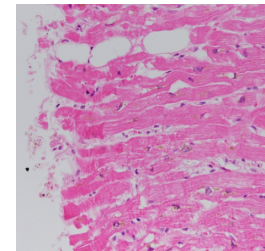


Figure 2 Histologic examination at the rupture site of the right ventricle showing no myocardial ischaemic changes.

haemorrhage was performed, but the patient died 4 hours after cardiac arrest.

At autopsy, multiple rib fractures (the right third and the left second and third), a left haemothorax with an intercostal artery injury and right ventricular rupture with haemopericardium and pulmonary artery trunk distention, were observed. No pericardial injuries or perforations were found. The right ventricle showed normal thickness, but a transmural longitudinal laceration approximately 7 cm in length was located along the interventricular septum, with the laceration turning outward, indicating a blowout-type rupture ([figure 1](#)). Histologic examination showed no vulnerability, such as ischaemic change, in the myocardium at the rupture site of the right ventricle ([figure 2](#)).

Cardiac rupture, which occurs in 0.6% of CPR-treated patients, is fatal and mostly associated with thoracic wall injuries, including rib or sternal

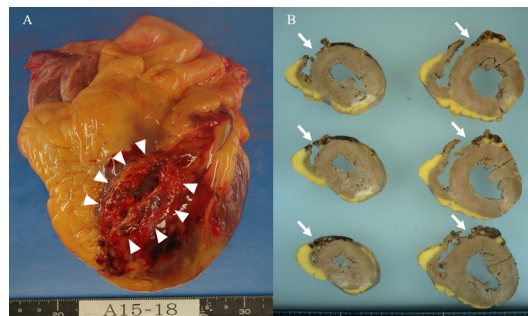


Figure 1 Macroscopic finding of the heart. (A) External view (arrowheads) and (B) axial slices (arrows) showing longitudinal laceration approximately 7 cm in length along the interventricular septum, with the laceration turning outward.

Learning points

- ▶ Cardiac rupture is a rare, fatal complication of cardiopulmonary resuscitation (CPR). Most cases are associated with thoracic wall injuries, including rib or sternal fractures, or myocardial ischaemia.
- ▶ CPR-associated right ventricular rupture independent of these factors can occur in the setting of acute massive pulmonary embolism. The possible mechanism in this case was the excessive increase in right ventricular pressure during chest compression concurrent with the obstruction of right ventricular outflow.
- ▶ The right ventricular wall adjacent to interventricular septum is vulnerable to the increase in intracardiac pressure.

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fractures, or myocardial ischaemia that usually occurs in the left ventricle.¹ The present case reported a rare CPR-associated right ventricular rupture independent of these factors. The possible mechanism of the right ventricular rupture in this case was the increase in intracardiac pressure due to chest compression in the setting of acute massive pulmonary embolism.^{2,3} Direct compression of the right heart during chest compression, concurrent with the obstruction of right ventricular outflow due to giant thrombi, may produce excessive intracardiac pressure. This excessive pressure may, in turn, result in the blowout rupture at the most vulnerable lesion of the right ventricular wall, the area adjacent to the interventricular septum.^{2,4}

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