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CASE REPORT

Successful Recovery After Prolonged Cardiopulmonary Resuscitation and Rescue Thrombolytics in a Patient with Cardiac Arrest Secondary to Presumed Massive Pulmonary Embolism

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Abstract: We present the case of a 60-year-old woman who suddenly suffered a witnessed cardiac arrest and did not achieve return of spontaneous circulation despite being given 150-minute ultra-long cardiopulmonary resuscitation (CPR). During CPR, pulmonary embolism was suspected and was eventually diagnosed based on refractory pulseless electrical activity, elevated serum D-dimmer, and a markedly enlarged right ventricle chamber. After rescue thrombolytic alteplase therapy, the patient was successfully resuscitated and had a good neurological recovery.

Keywords: cardiac arrest, pulmonary embolism, thrombolysis

Introduction

Whenever an atraumatic cardiac arrest (CA) happens to a previously healthy adult, it is of paramount importance to analyze the underlying etiology as well as the implementation of standard cardiopulmonary resuscitation (CPR). Unlike coronary artery disease, pulmonary embolism (PE) is an uncommon and easily neglected cause of CA, accounting for only 4–10% of all cases.[1] However, the early awareness and timely diagnosis of PE in patients with CA, particularly in those without any previous history of heart disease, may significantly improve outcomes in such patients.[2,3] Here, we report on the successful treatment of a 60-year-old woman with sudden CA caused by PE.

Case Report

A 60-year-old woman presented to our department after a witnessed 6-minute CA. She was otherwise healthy and had no history of heart disease, asthma, or deep vein thrombosis. Before her presentation, she experienced chest discomfort and shortness of breath lasted for about an hour, while brushing her teeth in the morning. In the ambulance on the way to hospital, her condition deteriorated and suffered a sudden CA. Standard CPR was carried out by the pre-hospital emergency doctors. Upon arrival at our emergency resuscitation unit, she was immediately intubated and given a single dose of epinephrine (1 mg, i.v.) and continuous manual chest compressions.

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An arterial blood gas test showed pH 6.87, PaO_2 39 mm Hg, $PaCO_2$ 49 mm Hg, HCO_3^- 9.4 mmol/L, BE −24.3 mmol/L, AG 29 mmol/L, K^+ 5.2mmol/L, and Lac 14.1mmol/L, and the peripheral oxygen saturation (SpO2) was 0%. The electrocardiogram showed no acute ischemic changes and myocardial enzymes were normal; hence, myocardial infarction appeared less likely. Pulseless electrical activity (PEA) was observed after 11 minutes of CPR, and electrical defibrillation was then implemented, but it was ineffective. Recurrent wide QRS complex PEA, at a rate of 30–120 beats per minute, dominated the rest of the 150-minute ultralong CPR, at the end of which, an intermittent return of spontaneous circulation was achieved in the patient. During CPR, bedside echocardiography was performed, and the results demonstrated a markedly dilated right ventricle deviating to the left side, which was significantly depressing the left ventricular systolic function. In addition, the patient's serum D-dimmer showed a significant elevation (3391 μg/L, reference 0–500 ug/L). Based on the above findings, the patient was strongly suspected of having a massive PE and a PE-related CA. We decided to give her rescue thrombolysis treatment using a 5-mg bolus dose of alteplase followed by another 65 mg, which was given continuously. About 20 minutes after the alteplase infusion and the continuous support of a dopamine and intra-aortic balloon pump (IABP), sinus rhythm was restored, and spontaneous circulation was relatively stable, with blood pressure increased to 76/54 mmHg. The patient was then transferred to the emergency intensive care unit for advanced life support.

The IABP was withdrawn two days later for the stable spontaneous circulation while heparin infusion was constantly administered. Bedside echocardiography was performed again, and the results showed right ventricular volume overload, an improvement of the mild tricuspid regurgitation, and right ventricular systolic pressure lowered to 40 mmHg. The patient's consciousness was recovered three days after the resuscitation and was extubated on day 9. After that, she underwent a computed tomography pulmonary angiography, which confirmed the presence of extensive bilateral pulmonary emboli (Figure 1). Coronary angiogram examination on day 24 showed no signs of coronary atherosclerotic heart disease or myocardial infarction (Figure 2). A magnetic resonance imaging scan on day 25 showed ischemic infarction in the globus pallidus caudate nucleus lobe. Fortunately, the patient only manifested a slight decline in memory and complex physical activity but had no major neurological sequelae. The patient finally survived and was discharged from the hospital 45 days after admission.

Discussion

Pulmonary embolism (PE) is an important cause of cardiac arrest representing at least 4% to 10% of these cases and associated with exceedingly unfavorable prognosis.[1] The main reason is that it is usually difficult to make an immediate and confirmed diagnosis of PE in CA patients during resuscitation partly due to fairly non-specific in presentation.[4,5] Several lessons have been learned from the successful treatment of this case. First, timely and well-performed CPR is of critical importance, as it could earn valuable time for doctors to perform and comprehensively analyze the work-up results. In our case, laboratory tests and bedside diagnostic instrument played an essential role in making an early diagnosis of PE. Portable bedside

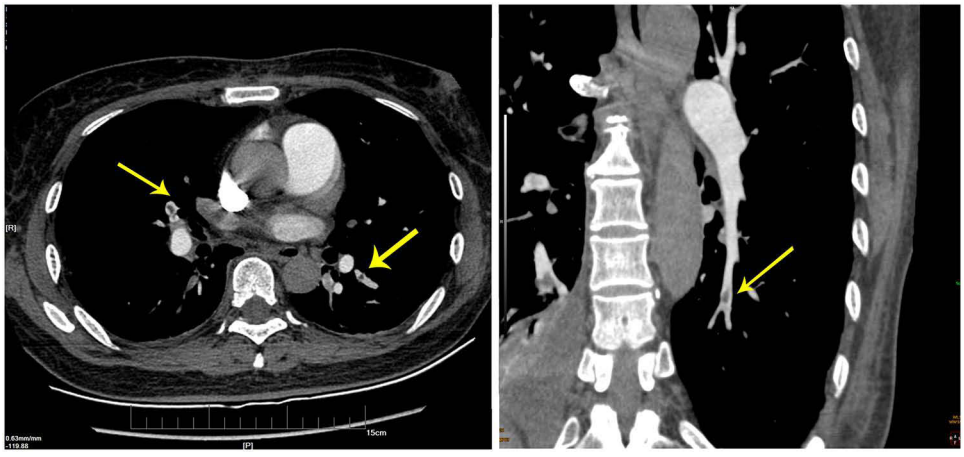

Figure 1 Pulmonary artery computerized tomography scan showing filling defects in the bilateral basement segment of the inferior branches of the artery (see arrow).

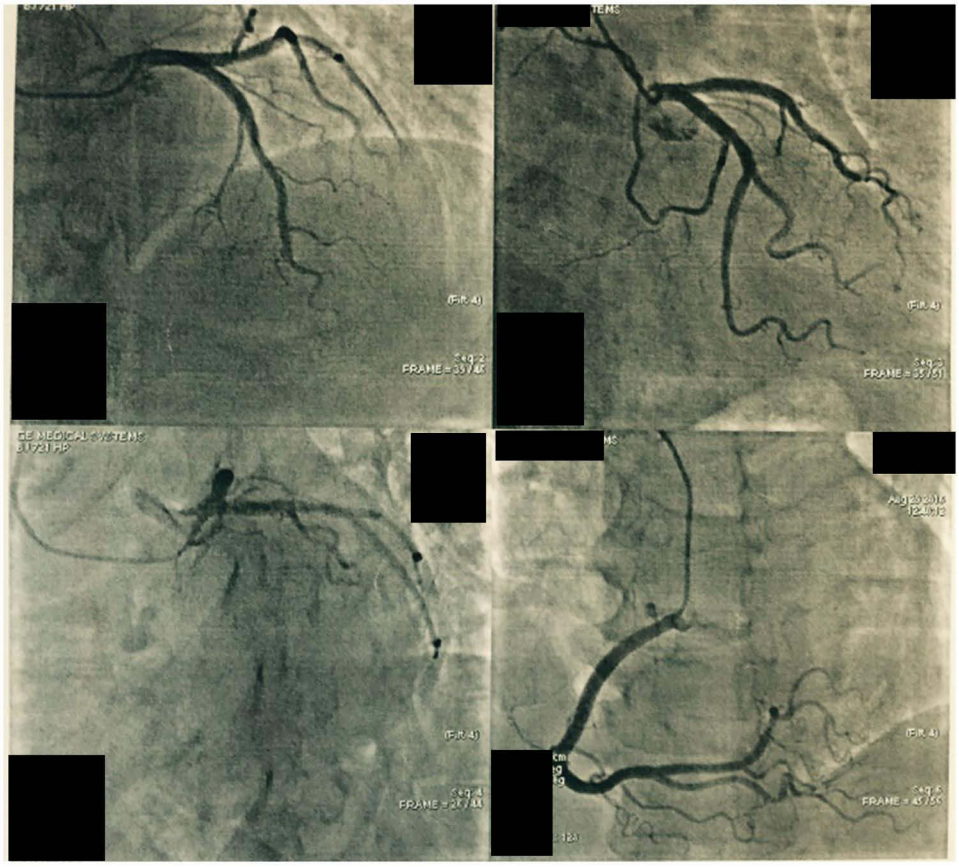

Figure 2 A coronary angiogram demonstrating no signs of coronary atherosclerotic heart disease.

echocardiography is a reliable tool as it recognizes the characteristics of PE, even during CPR.[6,7] Some studies from case report and case series showed that prolonged CPR, especially in combination with the utility of extracorporeal membrane oxygenation has meaningful survival rates and the survivors are able to regain favorable neurological outcome and return to normal life.[8–12] However, successful recovery after extremely long CPR with manual chest compressions, as in our case, has rarely been reported.[8,13] Our case suggested that prolonged resuscitation is not futile although it poses a real challenge for doctors with uncertain outcomes. We believe that the duration of CPR should ultimately be determined by the doctors in scene and depend on the specific condition of the patient. Prudent inspection of evidence that is strongly associated with PE helps to establish an early presumptive diagnosis in the absence of clear alternative etiologies. For example, in fulminant PE, up to 90% of CA occurs within one to two hours after the onset of symptoms.[1] Scrutinizing any electrocardiogram may also be helpful, as previous studies demonstrated that a right bundle branch block was present in 67% of PE cases and was strongly associated with CA caused by massive PE.[1,5] The dilatation of the right ventricle on ultrasound during cardiopulmonary resuscitation is partly associated with pulmonary embolism. Aagaard et al showed that the right ventricular was more dilated when CA was caused by pulmonary embolism compared with other etiologies such as primary arrhythmia.[14]

Thrombolysis is a potentially beneficial treatment option for patients in cardiac arrest due to presumed PE. Javaudin et al showed that thrombolysis during cardiopulmonary resuscitation may contribute to survival.[15] From the experience of our case, rescue thrombolytic therapy should be actively encouraged when a diagnosis, or even a presumed diagnosis, of PE has been made. CPR should be prolonged to make enough time for the thrombolytic therapy to take effect.[16] Despite potentially improved outcomes with thrombolytic therapy, this treatment is bearing risks. Patients received thrombolytics may undergo major bleeding events, such as devastating intracranial hemorrhage. To decrease the risk of thrombolytics for therapy of

CA due to PE, the clinician must correctly distinguish patients with presumed PE and must also adopt an appropriate thrombolytic agent and dosing regimen. Furthermore, proper high level supportive circulation care, such as IABP in our case, must be considered whenever it is needed.

In summary, the early recognition of etiology is of paramount importance for the successful resuscitation of patients with atraumatic CA and for CA caused by massive PE, and it appeared that a good recovery can be achieved with the use of rescue thrombolysis, even after an ultra-long CPR.

Informed Consent Statement

The written informed consent was obtained for the publication of the case details and relevant images from patients.

Disclosure

The authors report no conflicts of interest in this work.

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