

CASE REPORT

The use of transthoracic echocardiography to guide thrombolytic therapy during cardiac arrest due to massive pulmonary embolism

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The use and potential benefits of the focused trauma ultrasound examination in the accident and emergency setting has been increasingly recognised in recent years.¹ We report a case re-emphasising the benefits of immediate access to skilled ultrasound examination in the critically ill non-trauma patient.

CASE REPORT

A 25 year old woman presented to our accident and emergency department with a three hour history of retrosternal pleuritic chest pain and dyspnoea. She was previously completely well, was a non-smoker and her only medication was a

levonorgestrel-based second generation oral contraceptive. Initial clinical examination revealed moderate obesity, tachypnoea (oxygen saturation of 94% on air) and a tachycardia of 110 bpm. There were no other abnormal clinical signs and no evidence of lower limb venous thrombosis. 12-lead ECG showed an "S₁Q₃T₃" pattern.

After initial assessment she was accompanied to the toilet, where she collapsed with no palpable cardiac output. She was immediately transferred to the resuscitation room. Appropriate cardiopulmonary resuscitation was started and electromechanical dissociation was noted. A presumptive diagnosis of massive pulmonary embolism was made. She was given 3x1 mg

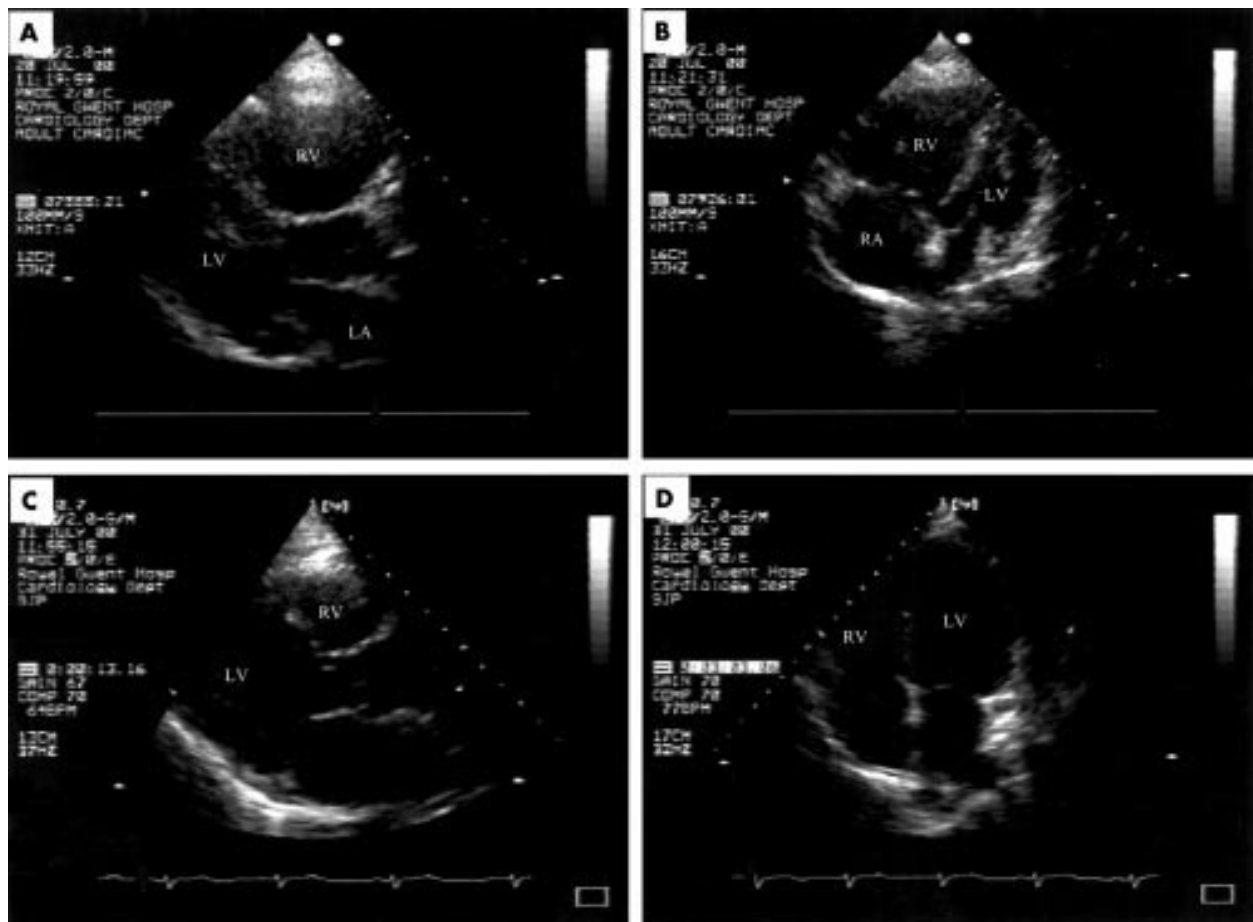


Figure 1 (A) Parasternal long axis and (B) apical 4-chamber transthoracic echocardiographic views obtained during cardiopulmonary resuscitation, showing gross right ventricular (RV) dilatation and an underfilled left ventricle (LV); (C) parasternal long axis and (D) apical 4-chamber echocardiographic views obtained nine days after thrombolysis (before discharge) showing normal cardiac chamber size. LA, left atrium; RA, right atrium.

adrenaline (epinephrine), 10 000 units of unfractionated heparin and 500 ml of colloid, with no return of cardiac output. External cardiac massage was interrupted (briefly) to allow transthoracic echocardiography at this stage. This revealed a massively dilated right ventricle and an empty, vigorously contracting left ventricle (fig 1A, B). Two 10 mg boluses of recombinant tissue type plasminogen activator (rtPA) were administered via the femoral vein, followed by an infusion of 90 mg over one hour. A further 1500 ml of colloid was given centrally over the following 10 minutes. At this point (after a total of 45 minutes of external cardiac compressions) cardiac output returned. The patient was transferred to the intensive care unit where she stabilised with a systolic blood pressure of 110 mm Hg and a sinus tachycardia of 140 bpm. ECG at this stage showed right bundle branch block. A chest radiograph was unremarkable.

Anticoagulation with intravenous unfractionated heparin followed her thrombolytic therapy and repeat echocardiography at 12 hours after arrest showed adequate left ventricular filling with a moderately dilated right ventricle and paradoxical septal motion indicating residual right ventricular pressure overload. She remained haemodynamically stable and was extubated after 24 hours. Renal and cognitive function remained normal. Venous Doppler studies confirmed the presence of an occlusive femoral vein thrombus. Abdominal ultrasound was normal. Warfarin was commenced on day 2, but intravenous heparin was continued for six days. Echocardiography on day 9 showed normal cardiac chamber size and function (fig 1C, D) and she was discharged well on the same day.

DISCUSSION

Pulmonary embolism is one of the few causes of catastrophic haemodynamic collapse and cardiac arrest in previously fit, well and often young patients. Electromechanical dissociation is the most common rhythm, although asystole and ventricular fibrillation have been described.² While surgical embolectomy and transcatheter techniques may have a role in selected patients, these are not usually available within a realistic timeframe. Thrombolysis, however, is almost universally available and is thought to improve survival in this situation. A review of three case series involving 34 patients given thrombolytic agents during cardiopulmonary resuscitation for massive pulmonary embolism suggested an initial survival rate of 55%–100%.³ A separate retrospective study of 60 patients admitted to an emergency department with cardiac arrest after pulmonary embolism showed a significantly higher rate of initial return of spontaneous circulation in patients given thrombolysis compared with those who were not thrombolysed (81% v 43%), although only 2 of the 21 patients given rtPA survived to hospital discharge.

Given the potential benefit of thrombolytic therapy, every effort should be made to diagnose massive pulmonary embolism quickly and accurately. A clear history is often difficult to obtain in this setting and attending physicians rely heavily on non-specific investigations that give inconclusive results. In this particular case, given the nature of the presentation, a presumptive diagnosis of massive pulmonary embolism had already been made before echocardiography and it could be argued that thrombolysis should have been administered anyway. However, a firm diagnosis is often easier to make retrospectively than in the setting of a cardiac arrest and there is an obvious (and appropriate) reluctance to use thrombolysis in the face of diagnostic uncertainty, because of its potential catastrophic effects, for example, if given to a patient with an aortic dissection. Transthoracic echocardiography is more diagnostically powerful and is widely available in district general hospitals, although its 24 hour availability is still unusual. A transthoracic echocardiogram is usually abnormal in massive pulmonary embolism, showing right ventricular enlargement, a consequent increase in right ventricular to left ventricular diastolic diameter and paradoxical septal motion, both in systole and diastole.⁴ It also allows the simultaneous exclusion of important left heart or aortic abnormality.

This case report illustrates that transthoracic echocardiography has the potential to substantiate the clinical diagnosis of massive pulmonary embolism during cardiopulmonary resuscitation to the extent that thrombolytic therapy can be rapidly, safely and effectively administered.

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