Westermark sign on chest X-ray in a patient following cardiac arrest due to massive pulmonary embolism

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DESCRIPTION

A woman in her early 60s was brought to the emergency room by the emergency medical services after presenting with a sudden cardiac arrest (CA). Her medical history included psychiatric disorders, tobacco dependence and diabetes, and she had undergone orthopaedic foot surgery 3 months earlier.

On their arrival at the scene, the emergency services estimated that there had been no cardiac output (no flow) for approximately 7 min with a pulseless electric activity being the first observed rhythm. Cardiopulmonary resuscitation (CPR) was initiated and 2 mg of epinephrine was administered resulting in a first return of spontaneous circulation and an estimated low flow period of 6 min. Initially ventilated with a supraglottic device, the patient was subsequently intubated. No reliable oxygen saturation measurement was obtained throughout the prehospital period although $EtC0_2$ target of 25–35 mm Hg was successfully maintained. Due to her being severely haemodynamically compromised, the patient was administered continuous intravenous epinephrine to maintain a targeted systolic blood pressure >90 mm Hg. The ECG showed sinus tachycardia with a normal QTc interval, right bundle branch block and an absence of any sign of acute coronary syndrome.

On arrival in the emergency department (ED), the patient's blood pressure was 82/42 mm Hg, heart rate 92/min and respiratory rate between 10 and 15/min (under mechanical ventilation). Severe desaturation (70% under fractional inspired oxygen 100%, digital saturation monitor) was observed and was evidently a major concern for the ED medical team. Pneumothorax, displacement or obstruction of the tracheal tube, and a lack of oxygen or ventilatory supply were successively and reasonably excluded with endotracheal aspiration, focused ultrasound and a ventilator technical check.

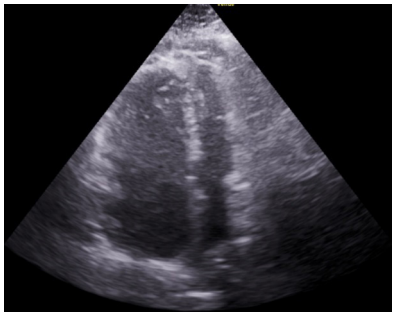

Figure 1 Apical four-chamber view on transthoracic echocardiography.

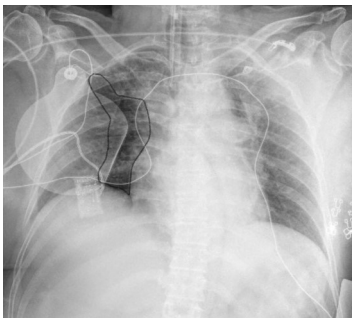

Figure 2 Anteroposterior chest X-ray, black delimitation showing the oligaemia part of the right lung.

A venous blood gas analysis showed a pH of 6.9 with a lactate value of 9.2 mmol/L. Focused echocardiography ruled out pericardial effusion and showed preserved cardiac motion with severe dilation of the right ventricle (right ventricle/left ventricle ratio of 3) and interventricular septal wall abnormalities suggesting right ventricular high pressure (figure 1). Other indirect echographic signs of pulmonary embolism (PE), such as inferior vena cava dilation, could not be obtained due to technical limitations.

PE resulting in consecutive haemodynamic compromise, profound hypoxaemia and cardiac reversible arrest was consequently suspected. Intravenous thrombolysis was thus initiated, as the patient's clinical condition was considered too unstable to perform a CT scan to confirm the hypothesis of PE.

A chest X-ray was also carried out, which confirmed the correct positioning of the tracheal tube and the absence of a pneumothorax. Interestingly, it additionally showed Westermark sign,

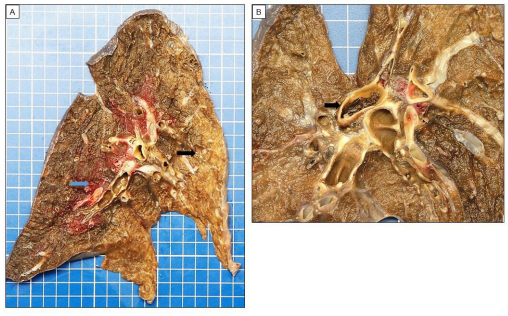

Figure 3 (A) Blue arrow shows central haemorrhagic area; black arrow shows a pale periphery of the right lung parenchyma related to a non-perfused lung. (B) Thrombus in the right pulmonary artery.

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corresponding to an area of pulmonary hyperlucency in the vascular territory amputated by the embolism (figure 2).

Unfortunately, in spite of thrombolysis, the patient suffered multiple further CAs, and CPR was eventually interrupted due to the accumulation of poor prognostic factors including cardiac standstill on focused echocardiography. A postmortem examination confirmed the presence of diffuse right-sided PE (figure 3A,B) as well as a dilation of the right ventricle without chronic component.

Westermark sign is a rare radiological sign first described by Westermark in 1938.[1] It is characterised by the appearance of oligaemia in the pulmonary vascular territory amputated by the embolism. Worsley *et al*[2] report a sensitivity of 14% and a specificity of 92%, based on the PIOPED cohort study.[3]

The diagnosis of massive PE is based on the clinical history and bundles of indirect arguments, in particular acute heart right-sided echographic signs. Chest CT scan remains the gold standard; its realisation is however often limited by the unstable clinical condition. Chest X-ray is still part of routine evaluation of CA but globally offers low diagnostic performance and clinical impact: it actually essentially aims at ruling out CPR complications and checking endotracheal tube position. However, the high reported specificity (92%) of Westermark sign suggests that it might be an aid to the clinical decision-making in CA, in particular in the absence of other hypotheses or in resource-limited settings (unavailability of ultrasound, for example).

To our knowledge, this is the first documented observation of Westermark sign on a chest radiography during CA caused by a PE.

Learning points

- In cardiac arrest, chest X-ray has low diagnostic performance.
- Westermark sign could be a diagnostic aid due to its reported high specificity, in particular in the absence of other hypotheses or in resource-limited settings.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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