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An unusual cause of ventricular tachycardia

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A 64-year old male presented for chest pain. His medical history included arterial hypertension, diabetes and a familial history of sudden cardiac death. Four weeks before he received a surgical treatment for a leiomyoma of the right malleolus, followed by 15 days of adequate thromboprohilaxis with low molecular weight heparin and early mobilization. In emergency department he suddenly experienced a pulseless ventricular tachycardia (VT) and was immediately defibrillated with 250 J (Fig. 1, panel A). After the resuscitation, blood pressure was 88/62 mm Hg, heart rate (HR) 124 beats per minute and oxygen saturation 88% on room air. His right leg appeared tender and swollen with the right calf 2 cm wider than the left. The electrocardiogram showed sinus tachycardia and negative T waves from V1 to V3. Bedside transthoracic echocardiography (TTE) revealed a mild enlarged and hypokinetic right ventricle. High-sensitivity troponin I was 85 ng/l (reference value < 45 ng/ml) while NT-pro-BNP was 864 pg/ml (reference value < 300 pg/ml). Compression ultrasonography (CUS) was negative for deep vein thrombosis (DVT).

What is the diagnosis?

Urgent computed tomography angiography revealed the presence of a large saddle pulmonary embolus (Fig. 1, Panel B, red arrow) with concomitant filling defects among the right and left pulmonary arteries (Fig. 1, Panel B, white arrows). The patients received i.v. thrombolysis with a rapid improvement of his clinical condition. Both a coronary angiography and a cardiac magnetic resonance, resulted negative, were

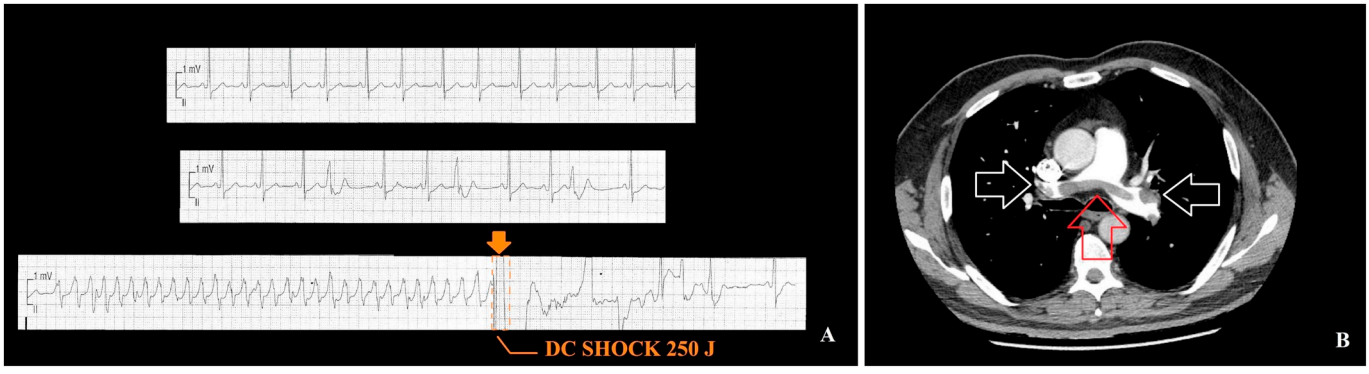

Fig. 1. (A) Evolution of ECG during the observation into the emergency department. DC shock is evidenced by orange arrow. (B) Computed tomography angiography (CTA) demonstrating saddle pulmonary embolism (red arrow) with concomitant major filling defects on the left and right pulmonary arteries (white arrows). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

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performed to exclude that the VT could be due to an ischemic genesis. Moreover, the work-up for congenital coagulation defects was negative as well the abdomen ultrasound performed to exclude an unknown cancer. The occurrence of ventricular arrhythmias as presenting symptom of acute PE is a very rare event. Indeed, the few reported cases of ventricular arrhythmic event occurred during PE were mainly due to a concomitant acute myocardial infarction and/or previous known/unknown coronary artery disease [1]. From a pathophysiological point of view, we assume that this strange arrhythmic presentation could be due to the arrhythmogenic properties of the tricuspid annulus which are potentially triggered by the transit of larger emboli. Indeed, similar arrhythmogenic presentations have already been reported during the ventricular embolization of vena cava filter [2]. Physicians should be suspicious of the diagnosis of acute PE in patients developing VT and with symptoms and/or medical history suggestive of venous thromboembolism since larger thrombi could trigger ventricular arrhythmic events.

Conflict of interest

None of the authors have conflicts of interest to declare.

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