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Make Your Diagnosis

Diagnosing Emergency Causes of Syncope, RUSH Based Approach

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Case Presentation

A 22-year-old woman with a 1-month medical history of left femoral shaft fracture s/p open reduction and internal fixation was sent to our hospital because of syncope. On arrival, she was mildly confused but could answer some questions. She was tachypneic, tachycardic, and afebrile. Blood pressure was 134/55 mmHg, and oxygen saturation was 84%. Physical examination revealed that she was in respiratory distress, and her skin was pale, cold, and cyanotic. No other remarkable findings were noted. Laboratory findings revealed mixed respiratory-metabolic acidosis and hypoxia. In addition, bedside ultrasound showed right ventricular dilation. Ten minutes after the patient arrived at the emergency department (ED), asystole occurred suddenly. Advance cardiac life support interventions were performed, and 50 mg of tissue plasminogen activator (tPA) bolus was given under the impression of massive pulmonary embolism (PE). Return of spontaneous circulation was noted after 10 minutes. Chest computed tomography (CT) performed after the cerebro-cardio-pulmonary resuscitation (CCPR) showed PE with pulmonary infarcts (Fig. 1). The patient was discharged 12 days later without further complications.

Discussion

When young patients are sent to the ED because of sudden syncope, the pathophysiology of the cardiovascular system and the brain should be considered in the differential diagnostic procedure. Table 1 lists possible differential diagnoses.[1]

This case exhibited several typical characteristics of massive PE. However, the immediate confirmation of this diagnosis was difficult because of the contextual factors at the ED. These contextual factors included an extremely short time for diagnosis, inability to retrieve complete case history, unsatisfactory angle or resolution of ultrasonography, and physician's concern regarding the side effects of thrombolytic agent injection, particularly when the diagnosis cannot be verified using CT images. Table 1 indicates two differential diagnoses that can result in fatal effects if thrombolytic agents are used: cerebral hemorrhage and aortic dissection. Without verification through CT, the distinction between these two conditions and PE can only depend on the limited case history, physical examination, and ultrasonography. Cerebral hemorrhage and aortic dissection both feature sudden attacks without prodromes, whereas PE occurs progressively with prodromes.[1] Therefore, to obtain key information, focused clinical inquiry and physical examination should be conducted during the short period when the patient recovers consciousness (Table 1).

Studies have reported a considerably varying survival rate for patients with cardiopulmonary arrest caused by massive PE who receive intravenous injection of tPA (10–80%).[2,3] In this case, the successful recovery was attributed to early diagnosis. Fig. 2 details the rapid diagnosis pathway. If the syncope is caused by critical arrhythmia, then the patient may exhibit mild symptoms after recovery; the physician should identify whether any subtle characteristics are

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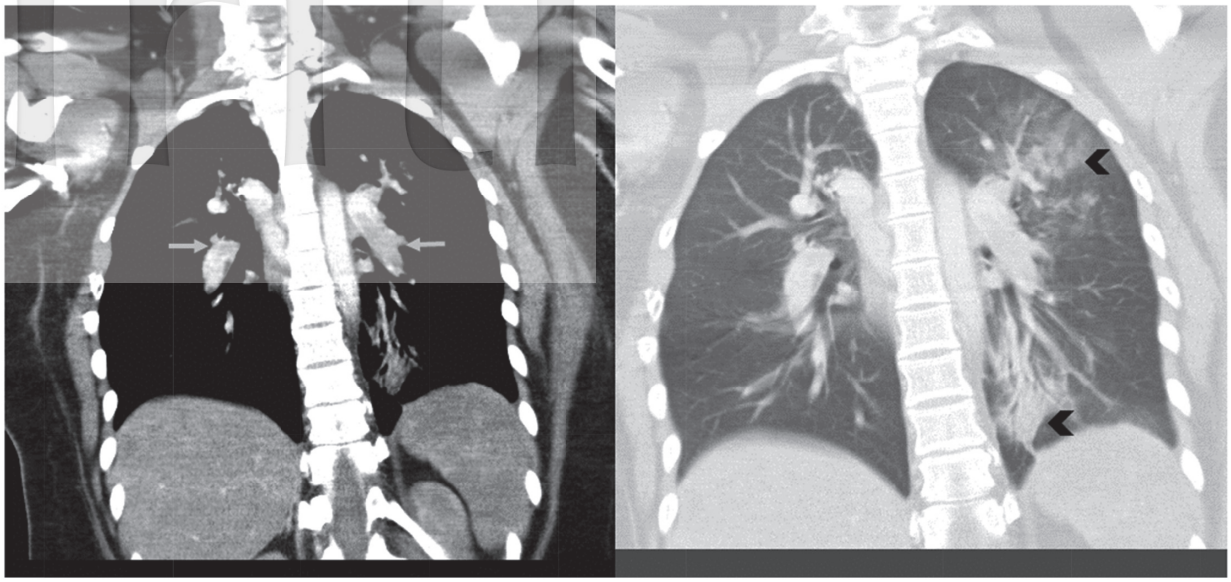

Fig. 1. Bilateral trunk embolism (arrow) and pulmonary infraction (arrowhead).

Table 1. Critical causes of diseases manifesting as syncope that require differential diagnoses within a short period

	Clinical clues	Features in ultrasonography results
Aortic dissection	Severe tearing pain in the chest	Pericardial effusion
	Unilateral neurological symptoms	Cardiac tamponade
	Pulse deficit in extremities	Aortic root dilation
	Marfan syndrome	Aorta flap
		Carotid artery flap
Massive	Chest pain	Right ventricular dilation
Pulmonary embolism	Decreased blood oxygen level	Left ventricular collapse
	Risk factors for pulmonary embolism	Jugular vein dilation
	Unilateral edema in lower extremities	Venous thrombosis in lower extremities
Fulminant myocarditis	Chest pain	Hypokinesis
	Dyspnoea	Pericardial effusion
	Manifestations of congestive heart failure	Lung rockets
Abdominal aortic aneyrysm	Severe abdominal pain	Increasing aorta diameter
	Shock	Aorta intraluminal clot
Internal Bleeding	Severe pain near the bleeding source	Asciates
		Pleural effusion
		Hematoma
IVH/SAH	Dizziness	None
	Severe headache	
	Lethargy	
Intraparenchymal hemorrhage	Abnormality in neurological examination	None
CO intoxication	Clusters of patients from a single locale	None
	Cold weather	
	Residual dizziness or headache	

IVH:intraventricular hemorrhage; SAH: subarachnoid hemorrhage.

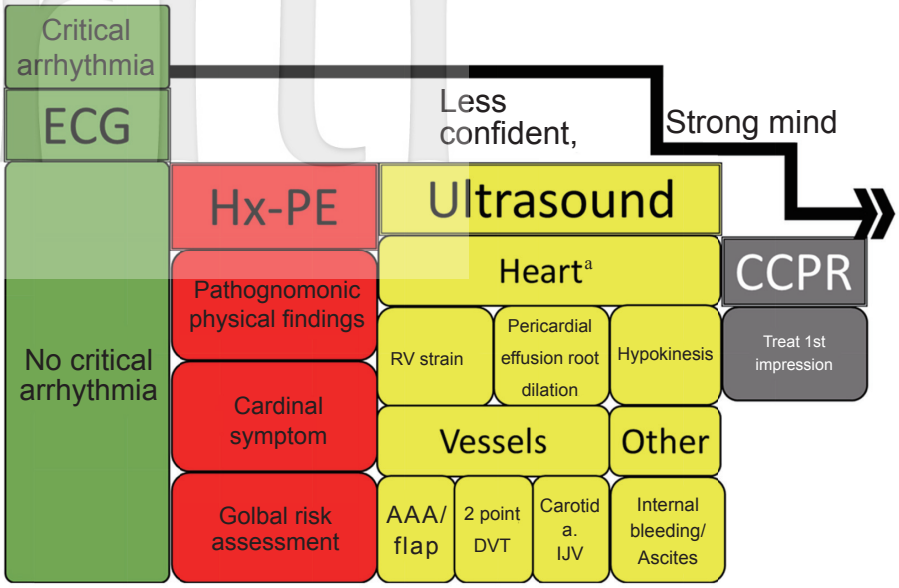

Fig. 2. Rapid diagnosis pathway for the emergency causes of syncope.
AAA: abdominal aortic aneurysm; CCPR: cerebro-cardio-pulmonary resuscitation; DVT: deep venous thrombosis; ECG: electrocardiography; Hx-PE: history-physical examination; IJV: internal jugular vein; RV: right ventricle.
[a]In the hands of experienced sonographers, hypertrophic cardiomyopathy and myxoma, may be diagnosed.

present in the electrocardiogram. For patients without critical arrhythmia, first steps involves retrieving detailed history. Physical examination should focus on neurological symptoms, abdominal mass, pulse deficit or unilateral edema. If no conclusion can be reached, cardiac ultrasonography can be adopted as the next step to obtain persuasive evidence. If no abnormalities are detected, the physician should scan peripheral blood vessels and abdomen to identify the presence of abdominal aortic aneurysm, aortic flaps or internal bleeding.[4,5] If the patient's condition has progressed to requiring CCPR, under the consideration that death is inevitable if the cause of arrest is not treated, the physician should immediately treat patients under the 1st impression without waiting definite diagnosis.

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