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Transient atrial fibrillation with syncope as presentation in pulmonary embolism.

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Abstract:

67 year old male with history of hypertension was brought to the emergency department (ED) after brief episode of loss of consciousness. Electrocardiogram in ED showed atrial fibrillation (AF) with rapid ventricular response. Physical examination was significant for discrepancy in calf size. Laboratory values were found to be in normal range except for mildly elevated troponin. Doppler studies of lower extremities showed right popliteal vein thrombosis. Computerized Tomography scan of chest revealed bilateral massive pulmonary embolism. 2 D echocardiogram showed right ventricular enlargement. Patient was started on anticoagulation with intravenous unfractionated heparin. He had spontaneous conversion of his AF to normal sinus rhythm within 24 hours without any recurrence. He was discharged home on coumadin without any complications. We hereby present an unusual and interesting presentation of pulmonary embolism masquerading as syncope associated with newly diagnosed AF and right ventricular strain.

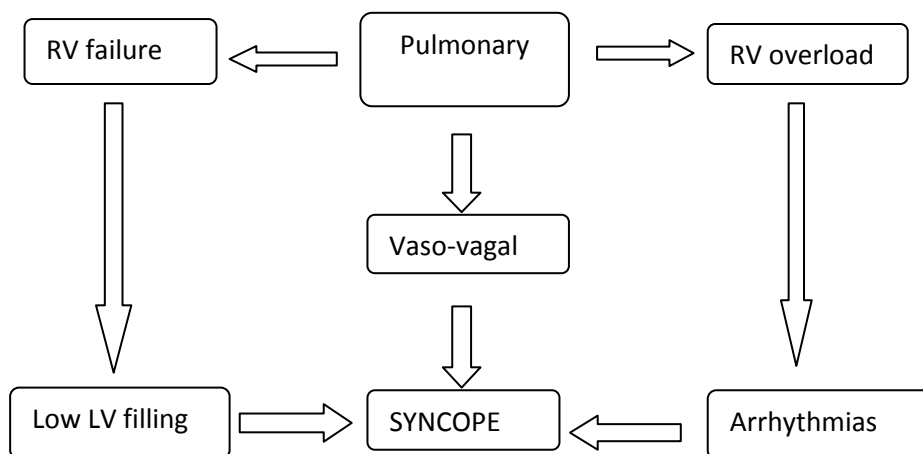
Introduction:

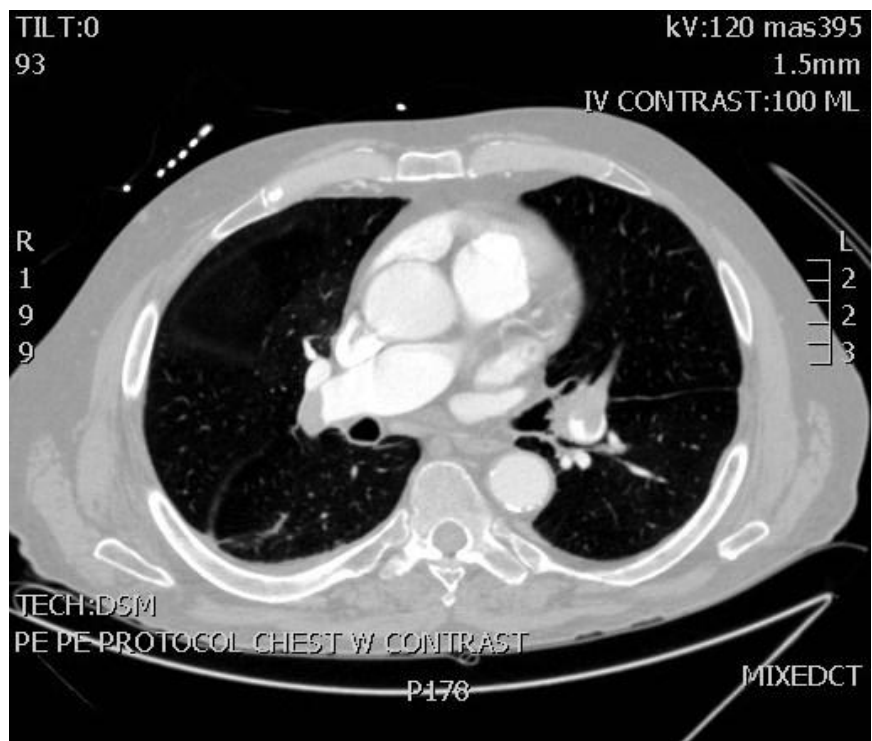
Syncope is easy to detect, but difficult to ascertain a specific cause in most of the cases. It is an uncommon presenting symptom of pulmonary embolism (PE) and is a symptom consigned to oblivion in the diagnosis of this life threatening condition because of the difficult correlation to make¹. Pulmonary embolism when masquerading as syncope tends to have high fatality rate¹. While syncope is an unusual presentation of pulmonary embolism, transient atrial fibrillation occurring in this setting is rare. We describe a case of elderly gentleman presenting with hypotension, syncope and atrial fibrillation found to have bilateral pulmonary embolism who was successfully treated with anticoagulation. Pathophysiology, poor prognostic factors and treatment options are discussed.

CASE:

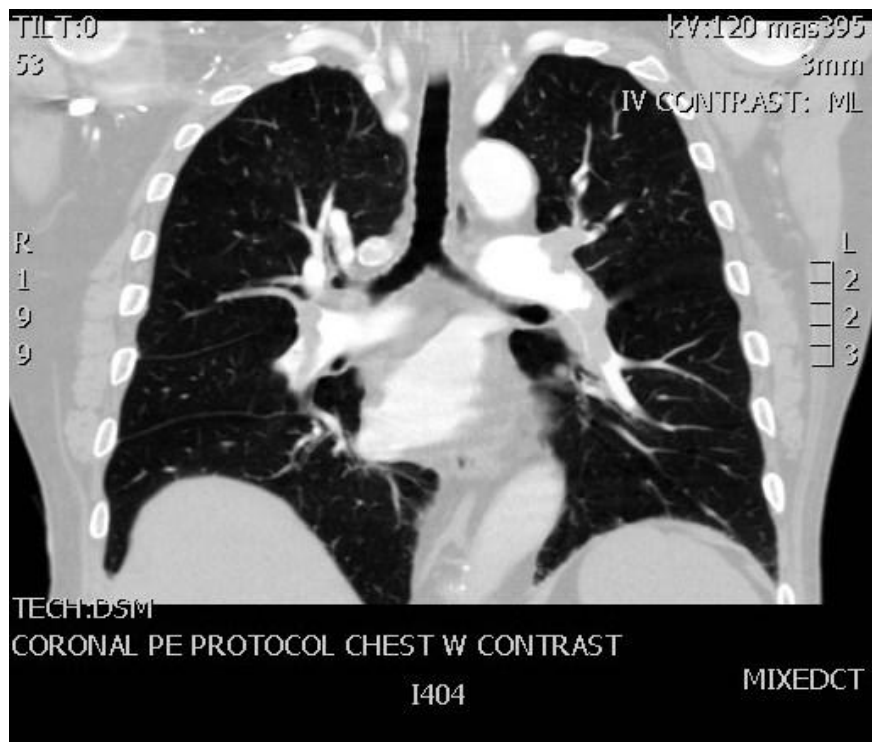
67 year old African American male with history of hypertension was brought to the emergency department (ED) after brief episode of loss of consciousness. Patient was in his usual state of health without any symptoms prior to the episode. He had gone for his regular walk and suddenly found himself on the ground; when he woke up, he was surrounded people. No convulsions or incontinence was witnessed. He had episode of hypotension (blood pressure 80/50), which resolved without any intervention in ambulance enroute to hospital. Review of system are negative for any prodromal symptoms, weight loss, fever, cough, chest pain, shortness of breath, palpitations or any similar episode in the past. Family history was negative for sudden cardiac death, malignancy and clotting disorders. Social history was negative for smoking, alcohol or illicit drug abuse. Physical examination was significant for discrepancy in calf size, right side measuring 1.5cm more than left. Electrocardiogram in ED showed newly diagnosed atrial fibrillation (AF) with ventricular rate of 130 beats per minute, which converted to normal sinus rhythm spontaneously within 24 hours after admission. Telemetry did not show any significant events. Lab work revealed normal values of complete blood count, electrolytes, renal functions and arterial blood gas parameters including A-a gradient. Hypercoaguable work up was negative. Troponin T was elevated to 0.01ng/ml (normal-less than 0.003). Doppler venous duplex study confirmed right popliteal vein thrombosis and subsequently patient underwent Computerized Tomography (CT) scan which revealed multiple bilateral main, upper and lower segmental and sub-segmental acute pulmonary emboli (Image 1 and 2). 2 D Echocardiogram (ECHO) showed right ventricular enlargement and elevated pulmonary artery pressure of 58 mm Hg (Video 1, Image 3). Patient was started on unfractionated heparin, transitioned to Coumadin and was discharged home in stable condition after few days of hospitalization. After three months, he is continued on Coumadin without any recurrence of symptoms.

Figure 1





(Image 1)



(Image 2)

DISCUSSION:

A documented cause of syncope can be found in only 60-80 percent of all syncopal events despite thorough workup. Diagnosis of acute PE is arduous when presented as syncope² although when syncope is associated with hypoxia, it is an important clue for raising a suspicion for PE. Syncope in the setting of pulmonary embolism can be the result of three possible mechanisms: (Figure 1). First, occlusion of the pulmonary vascular tree causing right ventricular failure and impaired left ventricular filling, leading to a reduction in cardiac output and arterial hypotension, all of which result in compromised cerebral blood flow. Second is the appearance of arrhythmias associated with right ventricular overload. Another possible mechanism is that embolism can trigger a vasovagal reflex that leads to neurogenic syncope³. Severe hypoxemia secondary to ventilation-perfusion mismatch might be a contributing factor in the development of syncope¹. Acute Pulmonary Embolism (PE) is a common presentation in hospitals and is often a fatal disease⁴. PE has been estimated to be approximately 600000 cases, with 100000 deaths per year in USA. It can be a contributing factor in another 100000 cases⁵. True incidence of PE is unknown as more than half of all PE cases are probably undiagnosed⁵. Early diagnosis and treatment lowers mortality which otherwise can range from 8% to 40%⁶. Hypotension in PE may resolve after a short interval of time, in this situation, the syncope episode might easily be attributed to many other causes. The appropriate diagnosis can be made only if other signs and symptoms suggestive of PE are present⁷. In patients presenting with chest pain, presence of AF increases the probability of finding of PE⁸.

The clinical presentation of PE ranges from classic triad to absence of symptoms. Studies has shown that most patients with PE present more often with one of the following three symptoms, which in decreasing order of frequency are sudden onset dyspnea, chest pain, and cough⁹, other symptomatology include calf or thigh pain, swelling, tachycardia, rales, decreased breath sounds, an accentuated pulmonic component of the second heart sound, hemoptysis if pulmonary infarction or signs like tachypnea, and jugular venous distension. PE diagnosis remains difficult due to the variety of the signs and symptoms¹⁰. Patients presenting with classic triad of pleuritic chest pain, dyspnea and cough are rare in the clinical practice.

Clinicians should look astutely for different clues to make the diagnosis of pulmonary embolism in patients who have had a syncopal episode: (a) hypotension, tachycardia or transient arrhythmia (b) acute cor pulmonale findings on physical examination or electrocardiogram and (c) other signs and symptoms indicative of pulmonary embolism. Absence of other obvious causes of syncope and the presence of any of these findings should alert any clinician.

Routine laboratory findings are very nonspecific in PE. They include leukocytosis, increased erythrocyte sedimentation rate (ESR), and an elevated serum LDH or AST (SGOT). More important blood tests include D-dimer and arterial blood gas. A positive D-dimer is not synonymous with PE because of high false positivity in many conditions, but a negative high sensitive D-dimer can rule out PE in low risk category. Sensitivity of D-dimer is about 96.3%

and negative predictive value reached 94.4%¹¹. Acute PE is typically presented with respiratory alkalosis, hypoxia and increased A-a O₂ gradient, however results of blood gas analysis are normal in 10% of cases¹². In such cases, exercise-induced desaturation on pulse oximetry should trigger further investigations¹³. Brain natriuretic peptide (BNP) and cardiac troponin have limited usefulness in diagnosis of PE but they have important prognostic significance.¹⁴

PE is associated with electrocardiographic changes in rate, rhythm, conduction, axis and morphology. The most common finding is sinus tachycardia (31%). Classic ECG pattern of S1Q3T3 is found only in 6% of patients with PE¹⁵. Arrhythmias include atrial fibrillation, atrial flutter, atrial tachycardia and atrial premature contractions. A transient complete or incomplete Right bundle branch block (RBBB) is the most frequent conduction abnormality, followed by first-degree atrioventricular block. Right axis deviation is common but left and indeterminate QRS axes are also described in PE¹⁶. Pulmonary embolism is also associated with non specific changes of ST-segment, T-wave inversions in the right precordial leads and an increase of P-pulmonale more than 2.5mm in lead II¹⁷. The presence of atrial arrhythmias, RBBB, and ST-segment elevation or depression is associated with RV failure and poor prognosis¹⁷.

Echocardiogram (ECHO) is not routinely suggested in a patient suspected to have PE¹⁸. Increased levels of cardiac bio-markers and [brain natriuretic peptide](#) are an indication for echocardiogram because of imminent right heart injury. ECHO can visualize the right heart dysfunction, which consist of right ventricular (RV) hypokinesis, akinesia of the mid-free wall (McConnell's sign), RV dilatation, abnormal interventricular septum movement, tricuspid regurgitation and lack of inspiratory collapse of the inferior vena cava¹⁸.

The Pulmonary Embolism Severity Index (PESI) and the simplified PESI (sPESI) are the most used and well known prognostic scores¹⁹. The simplified PESI which is easier to remember and has the same significance as PESI assigns one point each, for the following: age >80 years, history of cancer, chronic cardiopulmonary disease, heart rate ≥ 110 beats per minute, systolic blood pressure <100 mmHg, and arterial oxygen saturation <90 percent. Zero score indicates a low risk for mortality, and a score of one or more indicates a high risk²⁰. Patients with right ventricular (RV) dysfunction have a twofold increase in PE-related mortality. Patients with persistent RV dysfunction at the time of discharge or presence of RV thrombus are more likely to have a recurrent PE, DVT and higher mortality compared to patients with no RV dysfunction or RV thrombus²¹. Presence of DVT in a patient with an acute PE increases the risk for mortality. Elevation of brain natriuretic peptide (BNP) or N-terminal pro-brain natriuretic peptide (NT-pro BNP) predicts RV dysfunction that by itself means higher mortality. 95 percent of patients with low serum BNP levels of less than 50pg/mL had a benign clinical course. Rise in serum troponin levels have shown to have increased risk of death in acute PE patients¹⁴.

A retrospective cohort study of 13,728 patients who were discharged over a two year period with a primary diagnosis of PE showed that hyponatremia is associated with increased mortality and higher chance of hospital readmission²². Elevated lactate levels may have some prognostic value

in predicting death in patients with acute PE. Further studies are required to validate lactate levels as an accurate predictor of death in acute PE. Studies have shown that pulmonary embolism is not associated with an increased risk of recurrence and/or death when it presents as a syncope²³.

Treatment options:

Before the diagnostic workup, we should start anticoagulation when acute PE is a possible diagnosis²⁴.

Stable/small PE	Low molecular weight heparin or Unfractionated heparin
Unstable/ massive PE	IV unfractionated heparin (UH)
Hemodynamic collapse	Consider Thrombolytics or embolectomy

The duration of anticoagulation depends on previous episodes of thrombotic events and presence of risk factors. Individual patients should be reassessed for continuing long-term anticoagulant treatment at periodic intervals. LMWH is suggested for long-term anticoagulation in patients with malignancy related thrombosis²⁴.

TABLE 1:

Poor prognostic factors in PE	
Symptoms	Calf or thigh pain, calf or thigh swelling
Signs	Hypotension
EKG abnormalities	Atrial Arrhythmias, RBBB, ST elevation or depression
Investigation findings	Presence of DVT
	RV dysfunction at discharge
	RV thrombus
	Elevated troponins
	High BNP or Pro-BNP
	Hyponatremia
	Elevated Lactate

Summary:

Syncope is a presenting symptom, not a diagnosis. We must look for an etiology or a mechanism in all cases¹². We report an unusual case of massive pulmonary embolism, presented with syncope and new onset atrial arrhythmia. Clinicians should be vigilant about different presenting symptoms, to make an early diagnosis and management of potentially lifethreatening condition.

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