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A Plumbing and Electrical Problem: An Unusual Cause of Syncope

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CARDIOVASCULAR CASE SERIES

A Plumbing and Electrical Problem

An Unusual Cause of Syncope

Information about a real patient is presented in stages (boldface type) to expert clinicians (Dr Maskoun and Dr Khan), who respond to the information and share their reasoning with the reader (regular type). A discussion by the authors follows.

Patient presentation: A 64-year-old obese male (body mass index 35.3) with no other medical history presented with acute left leg swelling, shortness of breath, and diaphoresis which started acutely during the previous day. On presentation to the emergency department, his blood pressure was 138/97 mmHg, he was tachycardic to 103, tachypneic with a respiratory rate of 24, and afebrile. The patient's oxygen saturation was initially 90% and improved to 96% on 2 L nasal cannula oxygen supplementation. His cardiac examination was significant for regular tachycardia without murmurs or rubs on cardiac auscultation. The lung fields were clear to auscultation despite his tachypnea. His abdominal and neurological examination was normal. His neck veins were flat without evidence of jugular venous distension or hepatjugular reflux. Asymmetrical, nonpitting left lower extremity swelling was present with intact pulses in all extremities. His ECG showed sinus tachycardia and right bundle branch block, and was without an S1Q3T3 pattern. His BNP (B-type natriuretic peptide) was normal and troponin was elevated to a peak of 497 ng/L (reference value <40 ng/L). His clinical presentation combined with unilateral leg swelling, hypoxemia, and sinus tachycardia made an acute pulmonary embolus diagnosis highly suspected. Anticoagulation was started for his suspected pulmonary embolism (PE). A computed tomography scan of the chest with contrast showed a right-sided, submassive PE, and duplex ultrasound showed a large, left-sided deep venous thrombosis (DVT) (Figure 1). Transthoracic echocardiography showed a preserved left ventricular ejection fraction with right ventricular (RV) enlargement and McConnell sign¹ (Figure 2). The RV basal diameter was moderately dilated at 5.1 cm (normal <4.1 cm), right atrial volume was normal, and the inferior vena cava (IVC) was not well visualized.

Dr Maskoun: In this acutely ill patient, it is imperative to initiate workup and risk stratification rapidly so treatment is not delayed. Based on the presentation in this previously healthy patient with a Well's score of 7.5 (>4 being high risk), acute pulmonary embolism is certainly high on the differential and appropriate medical therapy with anticoagulation needs to be started. Other conditions, such as isolated right-sided infarct and acute decompensated heart failure should be on the differential diagnosis but are less likely in the setting of the patient's euvoletic clinical examination, presenting ECG, and dyspnea in the setting of unilateral leg swelling. The S1Q3T3 ECG pattern has been initially described in pulmonary embolism, but later data shows that it is neither sensitive nor specific for acute PE.² Of note, sinus tachycardia and incomplete right bundle block

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Key Words: May-Thurner syndrome
■ pulmonary embolism ■ tachycardia,
ventricular ■ venous thrombosis

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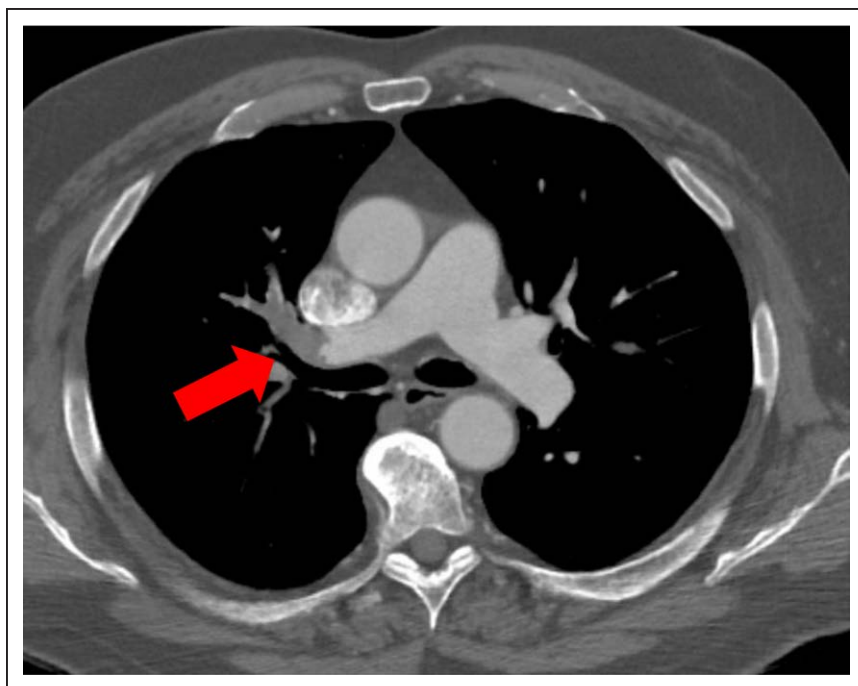


Figure 1. Computed tomography scan of the chest with intravenous contrast revealing bilateral pulmonary emboli, most significant in the right main pulmonary artery (arrow).

branch are the most sensitive ECG signs in acute PE.² Additionally, ST elevations in the right arm lead on the ECG >1 mm suggest poor prognosis in acute PE. Attention also needs to be paid to ST elevation in V1 which can be consistent with isolated RV infarct. Neither of these were present on the patient's presenting ECG. The only finding was sinus tachycardia which is a sensitive finding in acute PE but not specific. Ultimately, acute PE is an important diagnosis to make and treat early as it can be life-threatening without prompt therapy, and obtaining a computed tomography scan of the chest with contrast to solidify the diagnosis was appropriate, although it should not delay treatment. While obtaining a stat computed tomography scan is widely available in almost every emergency room in this country, this might be limited because of geography and other medical conditions. This is particularly true in the setting of the current unique coronavirus disease 2019 (COVID-19) pandemic. Acute myocardial infarction that requires emergent catheterization laboratory activation is less likely given absence of ischemic changes on ECG and the suspected DVT on physical examination. The echocardiogram offers useful information in this setting as well. The McConnell sign is a feature of RV free wall hypokinesis with apical hyperkinesis and is indicative of acute right heart failure/strain. Initially it was described as a sign of pulmonary embolus, but can be seen in other causes of acute right-sided heart failure, including isolated RV infarct and acute respiratory distress syndrome. Chronic RV dysfunction would not be expected to have a McConnell sign, and would show evidence of chronically elevated central venous pressure such as right atrial dilation, a distended IVC (not well visualized in this

patient), and global RV hypokinesis including in the apex which were not present in this case.

Patient presentation (continued): The patient was taken for an urgent pulmonary angiogram showing bilateral lower-lobe filling defects and bilateral catheter-directed thrombolysis catheters were placed (Figure 3). The patient's symptoms improved significantly, however 36 hours after presentation, he had 2 episodes of sustained monomorphic ventricular tachycardia associated with syncope requiring 2 separate direct current cardioversions with return to sinus rhythm with intermittent premature ventricular complexes (Figure 4). Both the axis of the VT and premature ventricular complexes after cardioversion were consistent with the location over the RV apex. Due to concern that the patient was having recurrent pulmonary emboli causing more RV strain and monomorphic ventricular tachycardia, an IVC filter was placed to help prevent further embolization.

Dr Maskoun: This was an unusual arrhythmia to be seen after a PE. The ventricular tachycardia (VT), premature ventricular complex morphology, clinical presentation, and echocardiogram findings all suggest that the current life-threatening arrhythmia was attributable to recurrent PE with RV strain. This VT is a left bundle branch block morphology with negative concordance suggesting origin at the ventricular septum or right ventricle, but there are several signs that help us localize the VT further. The negative inferior leads (II, III, and AVF) suggest an apical or posterior origin, and the positive AVR, AVL, and lead I also suggest an origin at the RV

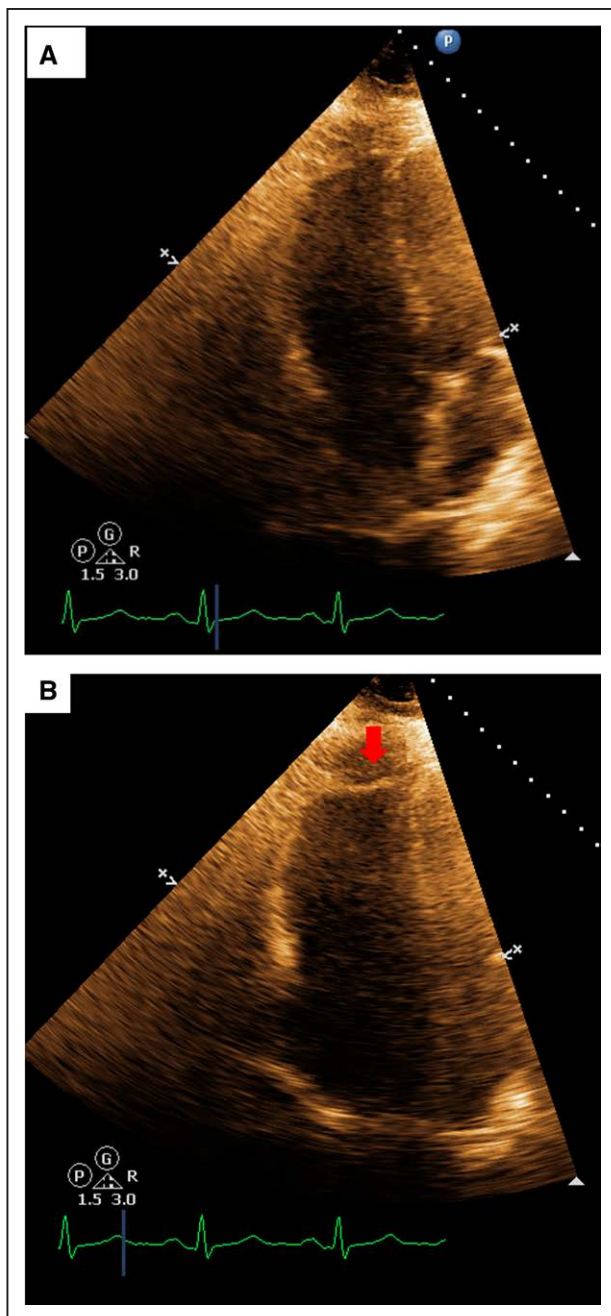


Figure 2. Transthoracic echocardiogram apical 4-chamber view with focus on the right ventricle.

The right ventricle shows significant dilation during diastole (A) and apical hyperkinesis (arrow) with basal hypokinesis during systole (B).

apex or apical septum. In addition, the negative deflection in lead III was greater than lead II and the positive deflection of AVL was greater than AVR, further demonstrating an RV apical origin, fitting the clinical presentation and echocardiogram findings. Managing most arrhythmias, whether focal or reentry, typically involves targeting the arrhythmia substrate, such as an accessory pathway, isthmus of re-entry, or clinical triggers like alcohol use or heart failure. We felt that the patient's relatively late presenting and serious arrhythmia was triggered

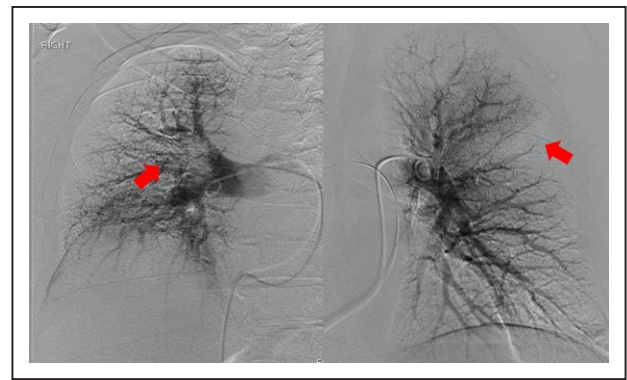


Figure 3. Bilateral pulmonary angiography obtained at the time of catheter-directed thrombolysis placement.

Bilateral filling defects are noted in the lobar pulmonary arteries compatible with pulmonary emboli (arrows).

by recurrent PE despite anticoagulation and catheter-directed thrombolysis, making placement of an IVC filter reasonable to prevent further embolization. Ventricular arrhythmias are sometimes seen because of myocardial reperfusion injury, however to our knowledge, this has not been currently reported in reperfusion lung injury.

Patient presentation (continued): The patient was continued on high intensity heparin for the duration of his catheter-based thrombolysis therapy and 72 hours afterward. He was then transitioned to apixaban 10 mg twice a day for 7 days, followed by 5 mg twice a day thereafter. The patient was started on amiodarone and discharged with a LifeVest with no recurrence of arrhythmia or symptoms. He was discharged home after 6 days with a plan for an outpatient electrophysiology study, and was continued on amiodarone for 1 month after discharge and then the medication was discontinued.

Dr Maskoun: Amiodarone and LifeVests are commonly prescribed for ventricular arrhythmias in the setting of ischemic cardiomyopathy. In asymptomatic, nonsustained VT after an acute myocardial infarction, the treatment should be management of the ischemia and optimal medical therapy. As per guidelines, if there is a potentially reversible cause to the VT, such as electrolyte disturbances or medications, they should be addressed instead of implantable cardioverter defibrillator placement. No data are available as to the benefit, if any, in patients with VT attributable to PE. The patient was very concerned about having another arrhythmic episode after leaving the hospital. While we felt the IVC filter would be protective of large recurrent embolism, a LifeVest was thought to be reasonable in the event of small emboli that might trigger additional VT.

Patient presentation (continued): A month after discharge he continued to have left leg swelling

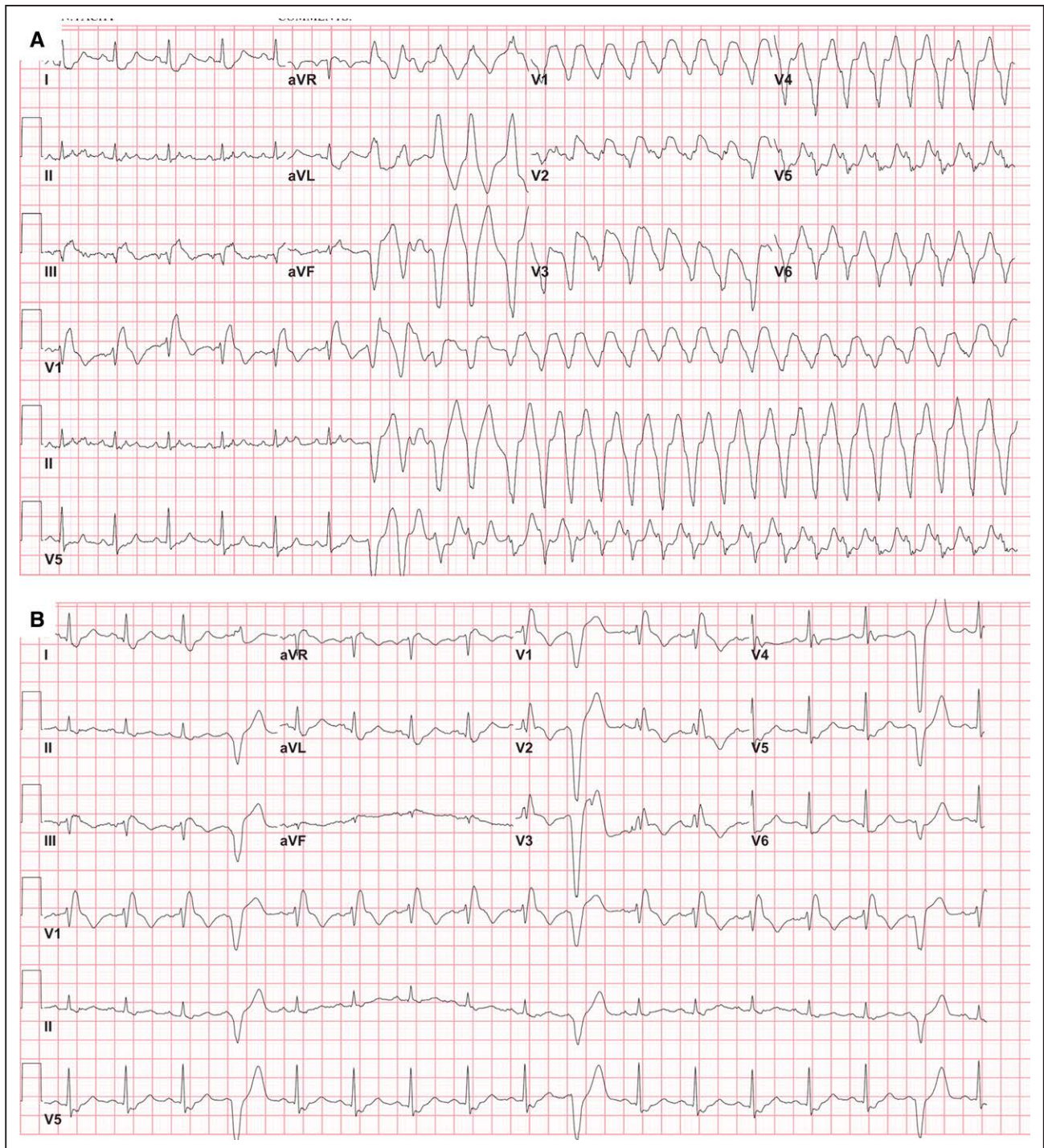


Figure 4. Twelve-lead ECG.

A, Twelve-lead ECG taken on initiation of a left bundle with a left superior axis ventricular tachycardia consistent with the right ventricular apex as the site of origin. **B,** Twelve-lead ECG immediately after cardioversion with frequent premature ventricular complexes with a similar axis to the previous ventricular tachycardia.

despite compliance with apixaban. He had no other trigger during this time for DVT such as long travel, recent surgery, or malignancy. Repeat left leg venous duplex ultrasonography showed residual extensive thrombus burden. He followed-up in the vascular surgery clinic where he was diagnosed with May–Thurner/iliac vein compression

syndrome.³ He received treatment with left iliofemoral vein thrombectomy and stent placement with significant improvement in his symptoms.

Dr Maskoun: When patients fail anticoagulation for DVT, this should be inspected closely. Noncompliance needs to be considered, but this was denied by

the patient. Alternative anticoagulation, such as warfarin or enoxaparin, may be considered, but a thorough investigation for cause of DVT persistence/recurrence needs to be evaluated. In this case, the patient had mechanical outflow obstruction of his left common iliac vein that improved with surgical management. May-Thurner syndrome typically affects the left leg because of the anatomic compression of the left common iliac vein by the overriding right common iliac artery. Recurrent or nonresolving left-sided DVT in the absence of other potential risk factors, such as hypercoagulability, immobility, or recent surgery, should trigger investigation for this treatable disorder.

Patient presentation (continued): Also on follow-up, the patient had a cardiac magnetic resonance imaging study showing no late gadolinium enhancement and normal RV size and function without evidence of scar. A nuclear stress test was negative for inducible ischemia. An electrophysiology study several months after his initial presentation while off amiodarone showed no inducible ventricular arrhythmias and an automatic implantable cardioverter defibrillator was not implanted. Recent follow-up ultrasound imaging shows patency of his bilateral iliac veins and no evidence of DVT recurrence or stent stenosis. He is currently following-up with interventional radiology with plans to have his filter removed in the near future

Dr Maskoun: After resolution of his acute issues, a cardiac magnetic resonance imaging study is helpful to rule out any previously undetected structural abnormalities of the heart or scar tissue. This also is helpful to evaluate for arrhythmogenic RV cardiomyopathy as a possible substrate/contributor to the patient's ventricular tachycardia. Also, this patient received an IVC filter which should be removed. This is typically done after the patient has tolerated anticoagulation for several weeks and there is no longer a concern for redevelopment of venous thromboembolism.

DISCUSSION

Sustained monomorphic ventricular tachycardia as a direct result of PE is very rare, and the best long-term

management of these arrhythmias is unclear. Classically, cardiac arrest as a result of PE is associated with pulseless electric activity arrest, with ventricular arrhythmias being very uncommon.⁴ Published case reports have shown VT as a result of large saddle pulmonary embolus.⁵ The mechanism of these arrhythmias is unclear, but suspected mechanisms include right heart strain from the acute changes in the RV myocardium because of increased RV afterload, leading to a McConnell sign on echocardiography and ischemic right VT. Alternatively, the arrhythmogenic tricuspid annulus can be irritated by clot-in-transit. Further research is needed to decide the best long-term management of these patients, and the need for different DVT management and automatic implantable cardioverter defibrillator implantation consideration.

ARTICLE INFORMATION

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Disclosures

None.

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