A 47-year-old black woman with no prior medical problems presented to the emergency department after a syncopal event associated with palpitations while walking. The patient noticed new onset of palpitations while walking that lasted about an hour. She subsequently had a syncopal episode. She denied dizziness, lightheadedness, dehydration, or seizure-like activity. Medications included antihistamines and oral contraceptives. On admission she was afebrile, heart rate 84 bpm, blood pressure 121/73 mmHg, respiratory rate 18 breaths per minute, and her oxygen saturation was 97% on room air. The physical examination was normal except for a small (1 cm) head laceration.

A noncontrast computed tomography of the head was negative for any acute intracranial findings. The chest radiograph showed no acute cardiopulmonary disease. The electrocardiogram showed normal sinus rhythm at a ventricular rate of 69 bpm, no evidence of ST segment changes, and an isolated T wave inversion in V2 (Figure 1).

She was admitted with high suspicion for cardiac syncope given her symptoms of palpitations before the event. Myocardial infarction was ruled out. A pro-B-type natriuretic peptide was normal and thyroid-stimulating hormone test was normal. A D-dimer level was not checked. Multidetector computed tomography of the head laceration showed normal endocardial border visualization demonstrating normal augmentation of LV function. However, there was significant dilation of the RV, with severe RV dysfunction and flattening of the interventricular septum, suggestive of RV pressure and volume overload (Figure 4; Movies I and II in the Data Supplement). There was an insufficient degree of tricuspid regurgitation to allow for estimation of RV systolic pressures. This information suggested an abnormality in the pulmonary vascular physiology, likely because of increased pulmonary vascular resistance and inadequate contractile reserve of the RV. In our clinical scenario, these findings were considered suspicious for an acute/subacute pulmonary embolism (PE).

Given concerns for PE, and a high pretest probability, a chest computed tomography angiogram was performed next that showed bilateral PE involving the right upper, middle and lower segmental/subsegmental pulmonary arteries as well as the left upper segmental and left lower segmental/subsegmental pulmonary arteries (Figure 5). Before this presentation, the patient had unlimited exercise tolerance. Given the new finding of palpitations on initial presentation and increased dyspnea with exercise during the stress test, we assume that this is a diagnosis of acute PE.

The patient denied any recent travel, trauma, or previous family history of a hypercoagulable state. She is up to date on her cancer screening. It was determined that the patient’s use of oral contraception was likely the cause of her developing the thromboembolic event. A lower extremity Doppler later did confirm acute, near complete thrombosis of the left popliteal vein. The patient was placed on full dose enoxaparin and discharged on warfarin with close outpatient follow-up. She has continued to do well.

The clinical diagnosis of PE can be challenging because of the vast range of clinical signs and symptoms that a patient may present with. A D-dimer is a good initial screening test for a PE. In our case, a PE was not initially considered, so a D-dimer was not checked. Multidetector computed...
tomography with intravenous contrast is now the most utilized diagnostic imaging modality for PE. Alternative imaging modalities include ventilation-perfusion (V/Q) lung scanning, transthoracic and transesophageal echocardiography. Resting transthoracic echocardiography in the setting of a known acute PE is used primarily for risk stratification. Echocardiographic findings related to increase RV afterload include RV dilatation, hypokinesis, tricuspid regurgitation, and interventricular septal wall flattening and septal motion toward the LV. Patients with concerns for a PE, or known PE, do not routinely undergo exercise stress echocardiography as it can be harmful, and therefore are not routinely used for the evaluation of a PE. Similarly, patients with acute thrombophlebitis or deep vein thrombosis are at increased risk of thromboembolism. Therefore, exercise stress echocardiogram is not a routine imaging modality used for the evaluation of PE or in patients with known acute thrombophlebitis or deep vein thrombosis.

Syncope is an infrequent presentation for PE, but may be a presenting symptom regardless of hemodynamics. Generally, evaluation of a patient with syncope and palpitations often includes a resting echocardiography to evaluate for underlying cardiac structural abnormalities. A stress echocardiogram was performed in our patient to evaluate for underlying ischemia or arrhythmias related to exertion, because her syncope occurred after exertion. On exercise stress echocardiography, in addition to evaluating for arrhythmias and signs of ischemia including new LV wall motion abnormalities, it is also important to assess the RV. In 1 study involving 2703 stress echocardiograms, abnormalities of the RV wall motion were present in 4% of patient and tended to occur in the setting of concomitant LV abnormalities. Patients with increased age, diabetes mellitus, hypertension, and known coronary artery disease were at higher risk of developing RV wall motion abnormalities. Evaluating the RV can be difficult, as in our case, given the technically difficult images and use of perflutren contrast.

There has been only 1 documented case of a PE diagnosed through exercise stress echocardiography, where it was performed in the setting of dyspnea on exertion for many months. When exercise-induced RV dilatation and hypokinesis may be seen on exercise stress echocardiography, its presence in the absence of LV abnormalities is unusual, and physicians should have a high suspicion for an abnormality of the pulmonary vasculature, specifically increased pulmonary vascular resistance. These manifestations become more evident when the cardiac output increases during stress and the RV lacks contractile reserve in the setting of increased pulmonary vascular resistance. When this is seen, differential includes both PE and pulmonary artery hypertension.

This case highlights the difficulty with diagnosing PE. Our patient had extensive bilateral PE; however, prognostic indicators such as RV size at rest and troponins were negative. This case emphasizes the high clinical suspicion and recognition of stress echocardiography findings that may be suggestive of PE in patients who may undergo an exercise stress echocardiogram for a different suspected diagnosis, such as coronary artery disease or valvular abnormalities.

Disclosures

None.

References

Figure 1. Admission 12-lead ECG showing normal sinus rhythm and no suspicious findings.

Figure 2. Two-dimensional echocardiogram in parasternal long (A) and short axis (B) images and apical 4 (C) and 2 chamber (D) images at rest.

Key Words: dyspnea • echocardiography, stress • pulmonary embolism • syncope • ventricular dysfunction, right
Figure 3. Patient’s stress ECG positive for exercise-induced myocardial ischemia (>2 mm ST segment depression in the inferior and lateral leads) at maximal stress of 103% maximum predicted heart rate and 5 metabolic equivalents.

Figure 4. Two-dimensional echocardiogram in parasternal long (A) and short axis (B) images and apical 4 (C) and 2 chamber (D) images immediately after treadmill exercise.
Figure 5. A, Chest computed tomographic angiogram after injection of intravenous contrast showing large left segmental pulmonary embolism. B, Chest computed tomographic angiogram after injection of intravenous contrast showing large right segmental pulmonary embolism.
Pulmonary Embolism Diagnosed From Right Heart Changes Seen After Exercise Stress Echocardiography
Brian C. Case, Michele Zemedkun, Amarin Sangkharat, Allen J. Taylor and Monvadi B. Srichai

Circ Cardiovasc Imaging. 2015;8:e003506
doi: 10.1161/CIRCIMAGING.115.003506

Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2015 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/8/8/e003506

Data Supplement (unedited) at:
http://circimaging.ahajournals.org/content/suppl/2015/07/20/CIRCIMAGING.115.003506.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Cardiovascular Imaging can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Cardiovascular Imaging is online at:
http://circimaging.ahajournals.org//subscriptions/
Supplemental Video Legends

**Video 1.** 2 dimensional echocardiogram in parasternal long (top images) and short (bottom images) axis views at rest (left side) and immediately post treadmill exercise (right side).

**Video 2.** 2 dimensional echocardiogram in apical 4 chamber (top images) and 2 chamber (bottom images) views at rest (left side) and immediately post treadmill exercise (right side).