

# A case of elevated troponins

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## The case

A 77-year-old woman was referred to our cardiology service from a local emergency department for further evaluation of palpitations and elevated troponins. The presumed diagnosis was a non-ST elevation myocardial infarction (NSTEMI). Her past medical history included a pre-syncope event in 1998, which resulted in some outpatient investigations. She conveyed to us that a treadmill exercise test and coronary angiogram had been normal. She had suboptimally controlled hypertension, but no other cardiovascular risk factors.

Four days prior to presentation, she experienced an episode of palpitations. This lasted for one to two minutes and resolved spontaneously. The palpitations re-occurred several more times throughout the day. She felt quite weak intermittently for the next three days, but did not experience any additional palpitations.

On the day of her admission, she experienced new symptoms after making lunch. When she sat down to eat, she began to feel very weak, and felt that she was breathing rapidly. She denied any chest pain or pressure, or any pain in her arms, neck or jaw at any time during these episodes. She also denied sweating, pallor, nausea or vomiting. Of note, she did report decreased exercise tolerance recently, as she had been inactive for the previous two months. This was on a background history of lumbar disc herniation with associated severe sciatica in her left leg, which limited her to sitting, or lying in bed for most of the time. She initially presented to her local emergency department. At this time her serum troponin was found to be elevated. An ECG demonstrated sinus tachycardia and some non-specific T-wave abnormalities in leads V1 and V2. As per their NSTEMI protocol, she received metoprolol, heparin and aspirin. She was then transferred to us.

## On examination

<b>Vital signs</b>	Pulse: 113 beats/minute, regular rhythm Blood pressure: 141/65mmHg Respirations: 20 breaths/minute Temperature: 36.7°C O <sub>2</sub> saturation: 98% on 2L O <sub>2</sub> by nasal cannula
<b>General inspection</b>	The patient appeared well and was resting comfortably in bed. No apparent pain, clinical distress or dyspnoea.
<b>Body mass index</b>	29.8kg/m <sup>2</sup>
<b>Neck</b>	No jugular venous distention. Normal carotid upstroke. No audible carotid bruits.
<b>Heart</b>	Palpable heave at the upper left sternal border. No chest wall tenderness on palpation. S1/S2 normal. Audible S4 at the left mid-sternum. No murmurs, clicks or rubs.
<b>Lungs</b>	Good air entry bilaterally. Breath sounds vesicular. No adventitious sounds.
<b>Abdomen</b>	Soft and non-tender, with no organomegaly, masses or pulsations. Bowel sounds present and normal. No renal artery bruits.
<b>Extremities</b>	Trace oedema to the ankle bilaterally. Dorsalis pedis pulses palpable and equal. No lumps or swellings.

We re-evaluated her troponin T, which was high at 0.21ng/ml (normal <0.01ng/ml). Coagulation studies revealed her D-dimers to be >2,000ng/ml (DVT rule out threshold ≤250ng/ml). Her ECG was essentially unchanged as compared to the ECG performed in her local emergency department. A chest x-ray was

uninformative. We calculated her modified Wells score for pulmonary embolism (PE) to be 6, indicating a high probability of PE. In light of her presentation, the results of the D-dimers made us highly suspicious for a PE. A contrast CT scan of the chest was ordered. A few hours later we had our

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answer: bilateral pulmonary emboli were present in the right and left pulmonary arteries extending into multiple lobar and segmental arteries (Figures 1a, b, c and d). Furthermore, there was enlargement of the right heart, and reflux of contrast into the hepatic veins, suggestive of acute right heart strain. The patient was anticoagulated with heparin and warfarin, and received physical and occupational therapy. She did well in hospital and was discharged to a skilled care facility for further reconditioning support before returning home.

## Discussion

PE is very common and a comprehensive discussion of diagnosis, management and follow-up can be found elsewhere. In this discussion, the emphasis will be on a few important learning points from this case. The relevance of this case discussion lies in the fact that PE is common and therefore important to recognise. The diagnosis of PE can be challenging, yet early diagnosis and treatment are necessary to reduce mortality. The mortality of untreated PE is 30% while mortality with treatment is between 2 and 8%.<sup>1,2</sup> The incidence of non-classical symptoms of acute coronary syndrome (ACS) is high in women. In a study of early warning symptoms of acute myocardial infarction in women, the most frequent acute symptoms were shortness of breath (57.9%), weakness (54.8%) and fatigue (42.9%). Acute chest pain was absent in 43%.<sup>3</sup> Given this fact, the non-specific symptoms in our patient's history, along with her elevated troponins, made it prudent to consider ACS. However, PE is a crucial diagnosis to make and should have been in the differential prior to her referral. The most common symptoms of PE in the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study are shown in Table 1.<sup>4</sup>

Despite this, PE may be asymptomatic. While our patient did not initially suffer any of the symptoms of PE outlined in Table 1, she did exhibit tachypnoea, tachycardia and a fourth heart sound (S4). She also had a palpable right ventricular (RV) heave from the straining right ventricle. Her S4 was a right-sided S4, representing an atrial kick against the stiff, straining RV. These findings underscore the fact that careful attention to clinical examination is warranted in these patients. The elevation in her troponins was probably the major misleading factor in the early cardiac focus of her workup. While elevated troponins are very specific for myocyte damage, in this case the myocyte damage was secondary to her right heart straining to pump blood into thrombus-clogged vessels. Thus, the first important point here is that an elevation in troponins should not automatically focus the differential on primary cardiac pathology. The second important point is that elevated troponins carry a significantly worse prognosis. In a meta-analysis by Becattini *et al*, 31% of PE cases involved elevated troponins. The mortality rate for patients with elevated troponins was 19.7% vs. 3.7% for those with PE without elevated troponins. It is important to note that the high mortality rate also included the subset of patients who were haemodynamically stable (odds ratio 5.46).<sup>5</sup> Thus, these patients need to be monitored closely for any signs of acute decompensation. The ECG in this patient was not particularly helpful as it revealed only sinus tachycardia (which in itself does not require an ECG for diagnosis). This certainly illustrates the rule rather than the exception. Sinus tachycardia is the only finding in most cases of PE.<sup>6</sup> There was T-wave flattening/inversion in leads V1 and V2,

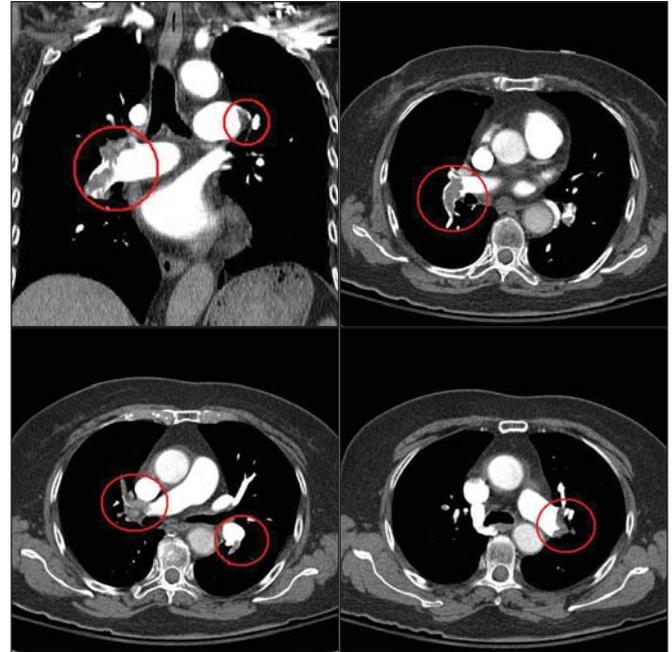


FIGURE 1: Contrast-enhanced CT images of the thorax (clockwise from top left): a) coronal view, demonstrating large hyperdensities consistent with pulmonary emboli at the bifurcation of the right pulmonary artery, and some clot in the left pulmonary artery; b) axial view, demonstrating hyperdense clot in branches of the right pulmonary arteries; c) axial view, demonstrating hyperdense clot in the left pulmonary artery; and, d) axial view, demonstrating hyperdense clot extending into the right and left lobar and segmental arteries.

Table 1: Most common symptoms/physical findings of PE in patients without pre-existing cardiopulmonary disease in the PIOPED Study.<sup>4</sup>

Symptoms	Signs
Dyspnoea (73%)	Tachypnoea (70%)
Pleuritic chest pain (66%)	Rales (crackles) (51%)
Cough (37%)	Tachycardia (30%)
Haemoptysis (13%)	S4 (24%)
	Loud P2 (23%)

which was probably, in retrospect, related to her PE, but no old ECGs were available for comparison. There are a few ECG changes that may be helpful in diagnosing PE. These include new right axis deviation, new right bundle branch block, new atrial fibrillation or flutter, or definite T-wave inversion in the right precordial leads (V1 and V2). These findings are suggestive of RV dysfunction and may carry a worse prognosis.<sup>7</sup> The classic pattern of an S-wave in lead I, a Q-wave in lead III and T-wave inversion in lead III (the so-called S1Q3T3 pattern) is highly suggestive of RV strain when present, but is only seen in about 12% of massive PEs

(i.e., those associated with systemic arterial hypotension).<sup>8</sup> The key laboratory finding in this case was the D-dimer elevation. D-dimers are elevated in 95% of patients with PE. The drawback is that they are highly non-specific, and can be elevated in patients without PE. For CT-experienced institutions (most institutions these days), the Christopher study validated a very simple and effective approach to working up a PE. The modified Wells score is used as a metric for stratifying patients into low or high clinical probability of PE prior to ordering investigations (Figure 2). In the study, out of 1,028 patients who had PE ruled out and received no anticoagulation, only five (0.5%) were found to have venous thromboembolism in the subsequent three months, none of which were fatal.<sup>9</sup>

As a final point, our patient was scheduled for follow-up malignancy screening. In older patients from the community presenting with their first PE, up to 20% will have an occult malignancy.<sup>10</sup>

In summary, PE may present with non-specific findings, which may hinder early diagnosis. Particularly in older women, the symptoms of PE overlap considerably with those of acute myocardial infarction. A careful history and physical examination is crucial. Elevated troponins are a common feature of acute PE and should be taken seriously, as they are associated with a significantly greater mortality rate, even in haemodynamically stable patients. A simple approach to diagnosis involves the use of the modified Wells score and D-dimers to guide the use of contrast-enhanced helical CT scan. Follow-up in older patients with first onset PE should include a screen for occult malignancy.

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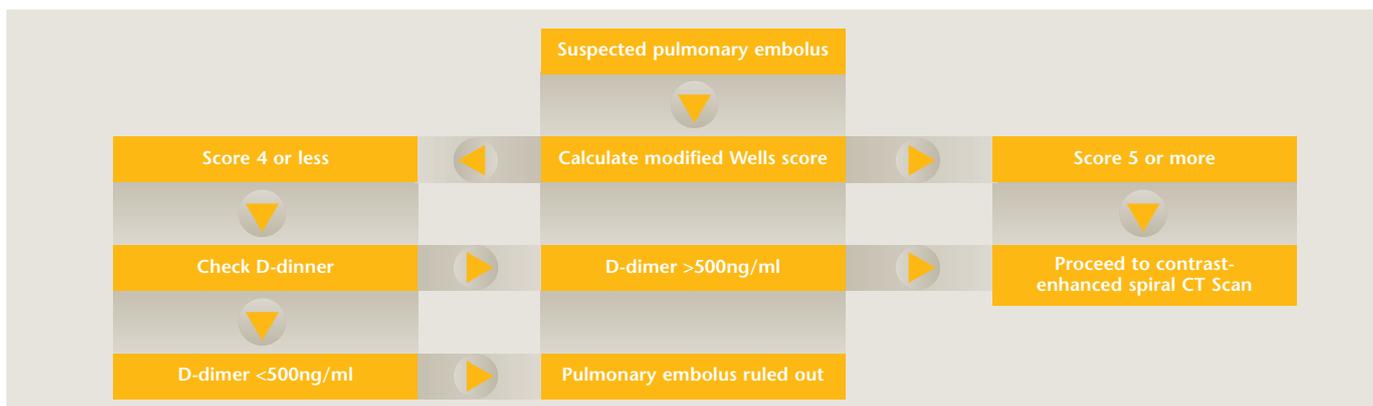


FIGURE 2: An approach to the diagnosis of PE as outlined by the Christopher Study.<sup>9</sup> The modified Wells score influences the use of D-dimers. The results of these two investigations guide the use of contrast-enhanced CT scanning.

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