QUESTION 1

A 65-year-old woman presents with a one-week history of progressive dyspnoea. On admission, there are signs of shock, a systolic murmur and an elevated jugular venous pressure. The ECG shows sinus tachycardia but no other abnormality. An antero-posterior chest X-ray shows cardiomegaly. The serum troponin I level is 0.5 mg/L [<0.1]. A computed tomography (CT) scan is shown below.

What is the most likely diagnosis?
A. Pulmonary embolism.
B. Right ventricular infarction.
C. Pericardial tamponade.
D. Myocarditis.
E. Acute mitral regurgitation.

Answer: C

The key here is that all the above if presenting as an emergency cannot be differentiated by the any of the presenting signs listed

A. Pulmonary embolism

CLINICAL SYMPTOMS / SIGNS — Specific symptoms and signs are not helpful diagnostically because their frequency is similar among patients with and without PE. In a large prospective study, the following frequencies of symptoms and signs were noted among patients without preexisting cardiopulmonary disease

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>73 percent</td>
</tr>
<tr>
<td>Pleuritic chest pain</td>
<td>66 percent</td>
</tr>
</tbody>
</table>
Cough | 37 percent
---|---
Hemoptysis | 13 percent

**Sign**
Tachypnea | 70 percent
Rales | 51 percent
Tachycardia | 30 percent
Fourth heart sound | 24 percent
Accentuated pulmonic component of second heart sound | 23 percent
Circulatory collapse | 8 percent


<table>
<thead>
<tr>
<th>Clinical Characteristic</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous pulmonary embolism or deep venous thrombosis</td>
<td>+1.5</td>
</tr>
<tr>
<td>Heart rate &gt;100 beats per minute</td>
<td>+1.5</td>
</tr>
<tr>
<td>Recent surgery or immobilization</td>
<td>+1.5</td>
</tr>
<tr>
<td>Clinical signs of deep venous thrombosis</td>
<td>+3</td>
</tr>
<tr>
<td>Alternative diagnosis less likely than pulmonary embolism</td>
<td>+3</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>+1</td>
</tr>
<tr>
<td>Cancer</td>
<td>+1</td>
</tr>
</tbody>
</table>


**Troponin** — Serum troponin I and troponin T are elevated in 30 to 50 percent of patients with a moderate to large pulmonary embolism. The presumed mechanism is acute right heart overload. The troponin elevations usually resolve within 40 hours with pulmonary embolism in contrast to the more prolonged elevation with acute myocardial injury.

**RADIOLOGY**

**Chest radiography** — Radiographic abnormalities are common in patients with PE; however, they are not helpful diagnostically because they are similarly common in patients without PE.

This site [http://www.msit.com/phys_art03.html](http://www.msit.com/phys_art03.html) provides some CT images of PE.

**B. Right Ventricular Infarction**

**CLINICAL**
- The classic clinical triad of right ventricular infarction includes distended neck veins, clear lung fields, and hypotension.
Year 2003 Paper two: Questions supplied by Tricia

- Infrequent clinical manifestations include right ventricular third and fourth heart sounds, which are typically audible at the left lower sternal border and increase with inspiration.

- On hemodynamic monitoring, disproportionate elevation of right-sided filling pressures compared with left-sided hemodynamics represents the hallmark of right ventricular infarction.

Differences between left and right ventricular myocardial infarction

<table>
<thead>
<tr>
<th></th>
<th>Left Ventricular MI</th>
<th>Right Ventricular MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>Pulmonary congestion</td>
<td>Clear lung fields</td>
</tr>
<tr>
<td></td>
<td>Third and fourth heart sounds</td>
<td>Right-sided third heart sound</td>
</tr>
<tr>
<td></td>
<td>New mitral regurgitation</td>
<td>New tricuspid regurgitation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypotension with distended neck veins</td>
</tr>
<tr>
<td>ECG</td>
<td>ST elevation in standard leads</td>
<td>ST elevation in V4R</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Commonly associated with inferior MI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Frequent atrioventricular block</td>
</tr>
<tr>
<td>Hemodynamic findings</td>
<td>Increased pulmonary capillary wedge pressure PCWP</td>
<td>Right atrial pressure (RAP) &gt;10mmHg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RAP: PCWP &gt;0.8</td>
</tr>
<tr>
<td>Specific Management</td>
<td>Fluid restriction</td>
<td>Fluid resuscitation</td>
</tr>
<tr>
<td></td>
<td>Preload and afterload reduction</td>
<td>Avoid Preload reduction</td>
</tr>
<tr>
<td></td>
<td>Reperfusion therapy</td>
<td>Reperfusion therapy</td>
</tr>
<tr>
<td></td>
<td>Ionotropic agents if necessary</td>
<td>Ionotropic agents if necessary</td>
</tr>
</tbody>
</table>

Imaging = TTE is the most appropriate

- In the appropriate clinical setting, a diagnosis of right ventricular infarction can be made using noninvasive techniques, or the patient may require right ventricular catheterization and hemodynamic monitoring.

- Echocardiography is useful as a modality to rule out pericardial disease and tamponade, which are the major differential diagnoses in the setting of a right ventricular infarction.

- Gated equilibrium radionuclide angiography and technetium 99m pyrophosphate scintigraphy are useful in diagnosing right ventricular infarction noninvasively (Sugimoto et al, 1996). In the case of radionuclide angiography, the right ventricle is demonstrated to be enlarged and poorly contractile, with a reduced ejection fraction. When technetium 99m pyrophosphate is employed, the right ventricular free wall is "hot," indicating significant infarction

C. Pericardial tamponade.
Cardiac tamponade is a clinical syndrome caused by the accumulation of fluid in the pericardial space, resulting in reduced ventricular filling and subsequent hemodynamic compromise. Cardiac tamponade is a medical emergency. The overall risk of death depends on the speed of diagnosis, the treatment provided, and the underlying cause of the tamponade.

Symptoms vary with the underlying cause and the acuteness of the tamponade. Patients with acute tamponade may present with dyspnea, tachycardia, and tachypnea. Cold and clammy extremities from hypoperfusion are also observed in some patients.
Distended neck veins are a common feature in patients with tamponade. Evidence of chest wall injury may be present in trauma patients. Tachycardia, tachypnea, and hepatomegaly are observed in more than 50% of patients with cardiac tamponade, and diminished heart sounds and a pericardial friction rub are present in approximately one third of patients.

The Beck triad or acute compression triad
- Described in 1935, this complex of physical findings refers to increased jugular venous pressure, hypotension, and diminished heart sounds.
- These findings result from a rapid accumulation of pericardial fluid. However, this classic triad is usually observed in patients with acute cardiac tamponade.

Pulsus paradoxus or paradoxical pulse:
- This is an exaggeration (>12 mm Hg or 9%) of the normal inspiratory decrease in systemic blood pressure.
- To measure the pulsus paradoxus, patients are often placed in a semirecumbent position; respirations should be normal. The blood pressure cuff is inflated to at least 20 mm Hg above the systolic pressure and slowly deflated until the first Korotkoff sounds are heard only during expiration. At this pressure reading, if the cuff is not further deflated and a pulsus paradoxus is present, the first Korotkoff sound is not audible during inspiration. As the cuff is further deflated, the point at which the first Korotkoff sound is audible during both inspiration and expiration is recorded. If the difference between the first and second measurement is greater than 12 mm Hg, an abnormal pulsus paradoxus is present.
- The paradox is that while listening to the heart sounds during inspiration, the pulse weakens or may not be palpated with certain heartbeats, while S1 is heard with all heartbeats.
- A pulsus paradoxus can be observed in patients with other conditions, such as constrictive pericarditis, severe obstructive pulmonary disease, restrictive cardiomyopathy, pulmonary embolism, rapid and labored breathing, and right ventricular infarction with shock.
- A pulsus paradoxus may be absent in patients with markedly elevated LV diastolic pressures, atrial septal defect, pulmonary hypertension, and aortic regurgitation.

Kussmaul sign
- This was described by Adolph Kussmaul as a paradoxical increase in venous distention and pressure during inspiration.
- This sign is usually observed in patients with constrictive pericarditis but occasionally is observed in patients with effusive-constrictive pericarditis and cardiac tamponade.

Ewart sign
- Also known as the Pins sign, this is observed in patients with large pericardial effusions.
- It is described as an area of dullness, with bronchial breath sounds and bronchophony below the angle of the left scapula.

The y descent
- The y descent is abolished in the jugular venous or right atrial waveform.
- This is due to an increase in intrapericardial pressure, preventing diastolic filling of the ventricles.
With a 12-lead electrocardiogram the following findings are suggestive but not diagnostic of pericardial tamponade.

- Sinus tachycardia
- Low-voltage QRS complexes
- Electrical alternans (also observed during supraventricular and ventricular tachycardia): Alternation of QRS complexes, usually in a 2:1 ratio, on electrocardiogram findings is called electrical alternans. This is due to movement of the heart in the pericardial space. Electrical alternans is also observed in patients with myocardial ischemia, acute pulmonary embolism, and tachyarrhythmias.

- PR segment depression

RADIOLOGY

Chest radiography findings may show cardiomegaly, water bottle–shaped heart, pericardial calcifications, or evidence of chest wall trauma

D. Myocarditis.

Physical: Physical findings can range from nearly normal examination findings to signs of fulminant CHF.

- Patients with mild cases of myocarditis have a nontoxic appearance and simply may appear to have a viral syndrome.
- Tachypnea and tachycardia are common. Tachycardia is often out of proportion to fever.
- More acutely ill patients have signs of circulatory impairment due to left ventricular failure.
- A widely inflamed heart shows the classic signs of ventricular dysfunction including the following:
  - Jugular venous distention
  - Bibasilar crackles
  - Ascites
  - Peripheral edema
- $S_3$ or a summation gallop may be noted with significant biventricular involvement.
- Intensity of $S_1$ may be diminished.
- Cyanosis may occur.
- Hypotension caused by left ventricular dysfunction is uncommon in the acute setting and indicates a poor prognosis when present.
- Murmurs of mitral or tricuspid regurgitation may be present due to ventricular dilation.
In cases where a dilated cardiomyopathy has developed, signs of peripheral or pulmonary thromboembolism may be found.

Diffuse inflammation may develop leading to pericardial effusion, without tamponade, and pericardial and pleural friction rub as the inflammatory process involves surrounding structures.

E. Acute mitral regurgitation.

**ETIOLOGY** — Although there are many causes of acute mitral regurgitation (MR), many of which can, under other circumstances, also cause chronic MR, there are only three basic mechanisms of acute native valve acute MR

1. Flail leaflet due to myxomatous disease (mitral valve prolapsed), infective endocarditis, or trauma
2. Chordae tendineae rupture due to trauma, spontaneous rupture, infective endocarditis, or acute rheumatic fever
3. Papillary muscle dysfunction or rupture due to acute myocardial infarction or severe ischaemia or trauma

Different mechanisms are responsible for acute MR in prosthetic valves:

1. Tissue valve leaflet rupture due to degeneration, calcification, or endocarditis.
2. Impaired closure of mechanical valve occluders due to valve thrombosis, infection, or pannus formation. With older generation mechanical valves, there were instances of strut fracture and disk escape, but these have not been reported with currently implanted valves.
3. Paravalvular regurgitation due to infection or suture rupture (often related to a calcified or scarred annulus).

Acute mitral regurgitation typically presents as a cardiac emergency with the sudden onset and rapid progression of pulmonary edema, hypotension, and signs and symptoms of cardiogenic shock. In some cases, the pulmonary hypertension leads to acute right-sided heart failure.

The presentation may not be as dramatic if acute MR is superimposed upon chronic MR or the patient is younger and physically fit. Such patients may present subacutely in the office or clinic, rather than in the Emergency Department. However, they may note a sudden and marked increase in symptoms of heart failure and a low output state, with increasing shortness of breath, dyspnea on exertion, fatigue, and weakness.

**Physical examination** — The patient with acute MR is often in pulmonary edema and there is evidence of poor tissue perfusion with peripheral vasoconstriction, pallor, and diaphoresis. The arterial pulse is often rapid and of low amplitude or thready due to the reduction in forward output. When there is an associated increase in right-sided pressure, the neck veins become distended; they may also become pulsatile with a marked “v” wave if the elevated right ventricular pressure leads to tricuspid regurgitation.

The chest radiograph usually shows a normal size cardiac silhouette, with severe left-sided congestive heart failure and pulmonary edema. An enlarged left ventricle and atrium may be present if chronic MR has been present prior to the acute event.

**ECG**
There are generally no electrocardiographic abnormalities specifically associated with acute MR. There may, however, be changes that reflect the etiology, such as an acute myocardial infarction, left ventricular hypertrophy, or P-mitrale reflecting underlying chronic MR.
Summary of presentation signs and symptoms related to options

<table>
<thead>
<tr>
<th></th>
<th>Pulmonary embolism</th>
<th>Right ventricular infarction</th>
<th>Pericardial tamponade</th>
<th>Myocarditis</th>
<th>Acute mitral regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progressive dyspnoea</td>
<td>yes</td>
<td>Yes</td>
<td>Yes</td>
<td>yes</td>
<td>yes</td>
</tr>
<tr>
<td>Shock</td>
<td>yes</td>
<td>Yes</td>
<td>Yes</td>
<td>yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Systolic murmur</td>
<td>yes</td>
<td>TR</td>
<td>Y</td>
<td>?</td>
<td>sometimes</td>
</tr>
<tr>
<td>Elevated jugular venous pressure</td>
<td>yes</td>
<td>Yes</td>
<td>Y</td>
<td>?</td>
<td>Yes</td>
</tr>
<tr>
<td>ECG sinus tachycardia only</td>
<td>Yes</td>
<td>Should be ST elevation</td>
<td>Y</td>
<td>Yes</td>
<td>Possible</td>
</tr>
<tr>
<td>CXR cardiomegaly</td>
<td>?pre existing</td>
<td>?</td>
<td>Y</td>
<td>Yes</td>
<td>possible</td>
</tr>
<tr>
<td>Increased trop I 0.5</td>
<td>Possible</td>
<td>Yes Inferior MI as mechanism</td>
<td>Y</td>
<td>possible</td>
<td>Ischaemia may be mechanism</td>
</tr>
<tr>
<td>CT – bilateral pleural effusion pericardial effusion</td>
<td>CT has a role No – thrombus identified</td>
<td>Usually echo but CT does have a role</td>
<td>? usually echo</td>
<td>? usually echo</td>
<td></td>
</tr>
</tbody>
</table>