A 82-year-old man presented to the emergency department with chest pain. His pain started one day before admission; it was intermittent, substernal, and pressure-like in nature. Gradually, the pain progressed and became constant. In addition, the patient developed shortness of breath and lower-extremity swelling.

Past medical history included hypertension, chronic kidney disease, severe chronic obstructive pulmonary disease (COPD), obstructive sleep apnea, gastroesophageal reflux disease, and prostate cancer status post-transurethral resection. His home medications include candesartan 24 mg daily, metoprolol tartrate 50 mg twice daily, furosemide 10 mg daily, esomeprazole 40 mg daily, fluticasone-vilanterol 200-25 μg/act inhaler with 1 puff daily, umeclidinium 62.5 μg/act with 1 puff daily, and albuterol as needed. He was not using a continuous positive airway pressure machine at home.

On arrival to the emergency department, he was in acute respiratory distress. Examination revealed an elderly man with a body mass index of 23.5 kg/m²; vital signs: temperature 36.4°C, heart rate 111 beats per minute; respiratory rate 27 breaths per minute; blood pressure 114/73 mm Hg; oxygen saturation (SpO2) 80% on room air. On examination, the patient was sitting upright, leaning forward, and breathing heavily. His head was normoecephalic and atraumatic. Eyes were equal, round, and reactive to light. Nose and mouth demonstrated normal moist mucosa. Chest auscultation was notable for diffuse crackles up to midlungs bilaterally. Cardiac auscultation was also significant for regular rhythm; tachycardia; and a soft, low-pitched systolic murmur. Abdomen was soft, nontender, and nondistended. Lower extremities demonstrated bilateral pitting edema up to the knees with intact peripheral pulses. He was placed on 2L supplemental O2 by nasal cannula.

Electrocardiography (ECG) showed ST segment elevations in leads III and aVF, with reciprocal ST segment depressions in leads I, aVL, V2, and V3. Initial troponin T was 385 ng/L. Point-of-care arterial blood gas showed pH 7.24 with pCO2 49 mm Hg and bicarbonate 26 mmol/L.

1. Given ongoing chest pain and ECG findings demonstrating ST elevations, which one feature of this patient’s history is most likely to change management?
   a) Presentation after 24 hours of onset of chest pain
   b) Advanced age
   c) Chronic kidney disease
   d) Chronic obstructive pulmonary disease
   e) History of prostate cancer

This patient was diagnosed with ST-elevation myocardial infarction (STEMI), a clinical syndrome that occurs after plaque rupture and thrombosis, resulting in abrupt cessation of coronary arterial blood supply. Rapid restoration of blood flow is the cornerstone of management via pharmacologic (ie, fibrinolysis) or percutaneous intervention (PCI). Poor outcomes correlate with total ischemic time or the time from onset of symptoms to reperfusion. As a result, guidelines emphasize early diagnosis and treatment of STEMI. Within the early phase of a STEMI, defined as a total ischemic time of less than 12 hours, reperfusion therapy can be either PCI or fibrinolitics, depending on PCI capability and transport time. The cutoff time of 12 hours was originally determined with respect to use of fibrinolitics,
which have a maximal benefit in the first hour of onset of symptoms but limited benefit after 12 hours. After 12 hours, primary PCI is the preferred option over fibrinolytics, even when travel time exceeds 120 minutes.

The remainder of the answers are additional prognostic factors but do not necessarily affect management. Advanced age is associated with increased risk of mechanical complications, acute heart failure, and inhospital mortality. Elderly patients often present with atypical symptoms, and delayed presentation is common. Up to age 85, reperfusion therapy improves mortality but may be limited by contraindications. However, there are no specific guidelines for elderly patients, owing to a limited number of clinical trials for this patient population. Given the clinical heterogeneity in this patient population, choice of treatment should be patient centered. Patients with renal insufficiency are at higher risk after STEMI for cardiovascular complications, including short-term inhospital mortality. However, reperfusion therapy is associated with reduction in mortality. Similarly, there is no change in management for acute MI, given his history of COPD or prostate cancer. It is important to avoid undertreatment and deviation from therapeutic interventions in patients with complex medical comorbidities.

The patient was diagnosed with STEMI based on ECG, initial troponin, and clinical presentation. He was given aspirin 324 mg, clopidogrel 600 mg, and started on a heparin infusion.

2. After diagnosing late-presenting STEMI, what would be the best next step in management?
   a) Transfer to cardiac intensive care unit (CICU) and monitor
   b) Transfer to CICU and coronary angiography in the next 24 hours
   c) Coronary angiography with possible intervention and transfer to CICU
   d) Immediate fibrinolytics and transfer to CICU
   e) Cardiac surgery consultation

For late-presenting patients without evidence of ongoing ischemia or other high-risk features, urgent PCI is not necessary. In this setting, transfer to the CICU to monitor for post-STEMI complications is adequate. Routine primary PCI can be considered within 48 hours of symptom onset. However, in late-presenting patients with ongoing ischemic symptoms, heart failure, hemodynamic instability, or life-threatening arrhythmias, a primary PCI strategy is strongly recommended. Given evidence of acute decompensated heart failure in this patient, coronary angiography should not be deferred for 24 hours. Rather, coronary angiogram with possible PCI should be performed emergently before transfer to the CICU. As previously mentioned, fibrinolytics are not the treatment of choice for late-presenting MI. Reasons for cardiac surgery consultation include mechanical complications following MI, unsuitable coronary anatomy for PCI with evidence of cardiogenic shock or large myocardial area at jeopardy, left main coronary artery stenosis, and multivessel disease. However, none of these indications had been identified at this point in the case.

As the patient presented to a PCI-capable hospital, he underwent coronary angiography. He was found to have diffuse multivessel coronary disease, with 100% obstruction of the right posterolateral segment by a thrombus, and a 95% obstructed ramus intermedius segment by a chronic discrete lesion. He underwent PCI with placement of a drug-eluting stent in the right posterolateral artery and placement of 2 overlapping stents in the ramus intermedius. Due to worsening respiratory failure, the patient was intubated and sedated, requiring vasopressors.

After coronary catheterization, the patient was admitted to the CICU because of continued mechanical ventilation and vasopressor requirement. Bedside transthoracic echocardiogram showed flail mitral valve leaflet with possible papillary muscle rupture. The left ventricle was normal size, with estimated ejection fraction of 65% and akinesis of the inferior wall. The right
ventricle was normal size with normal systolic function.

3. Which physical examination sign is most associated with the patient’s valvular heart disease?
   a) Harsh holosystolic murmur at the lower left or right sternal border
   b) Soft, decrescendo, low-pitched, systolic murmur
   c) Distant heart sounds
   d) Warm extremities
   e) Canon a-waves in jugular venous pulse

There are 3 main mechanical complications following a STEMI: ventricular septal rupture (VSD), papillary muscle rupture, and free wall rupture; VSD demonstrates a new harsh holosystolic murmur at the lower left or right sternal border accompanied by a palpable thrill and signs of acute biventricular heart failure. The defect creates a left-to-right shunt, increasing pulmonary blood flow and causing left ventricular (LV) overload. Compensatory vasoconstriction further exacerbates the left-to-right shunt. This may result in pulmonary congestion and dependent edema, but often there are little to no hemodynamic findings other than a murmur. The patient has papillary muscle rupture, which presents similarly with a new systolic murmur and sudden increase in left atrial pressure and volume, causing acute pulmonary edema. Unlike the holosystolic murmur of chronic mitral regurgitation, the murmur for an acute mitral regurgitation (MR) is soft and low pitched with a decrescendo caused by rapid equilibration in pressure between left atrium and LV during systole. Finally, free-wall rupture can cause cardiac tamponade secondary to hemopericardium. Characteristic examination findings for cardiac tamponade include distant heart sounds. Although an acute rupture often results in sudden death, a subacute rupture may allow for formation of a thrombus, sealing the perforation and creating a pseudoaneurysm. All 3 of these mechanical complications are possible causes of cardiogenic shock from LV dysfunction and decreased cardiac output.

Warm extremities are associated with high-output shock, such as from sepsis, and would not be expected with mechanical complications following an MI. Canon a-waves in the jugular venous pulse are often seen in complete heart block, caused by disassociation between atrial and ventricular contraction. Although heart block may be a complication of MI, it is not caused by papillary muscle rupture.

A transesophageal echocardiogram was performed for better anatomic clarification of papillary muscle rupture. It showed severe mitral valve regurgitation caused by rupture of the anterolateral papillary muscle and resultant flail P1 and A1 segments.

4. Occlusion of which coronary artery has resulted in papillary muscle rupture in this case?
   a) Left anterior descending artery
   b) Left circumflex artery
   c) Right coronary artery
   d) Ramus intermedius artery
   e) Obtuse marginal artery

The mitral valve apparatus consists of 2 papillary muscles originating from the LV wall, in the anterolateral and posteromedial positions. Each muscle attaches to both anterior and posterior mitral valve leaflets via chordae tendinea. The anterolateral papillary muscle has dual blood supply from the left anterior descending and left circumflex arteries. The posteromedial papillary muscle is supplied by only the right coronary artery. As a result, compared with anterolateral muscle, the posteromedial muscle is 6 to 12 times more likely to rupture, given its single blood supply. The ramus intermedius artery results from trifurcation of the left main artery. It can follow a course similar to the obtuse branches of the circumflex artery or the diagonal branches of the left anterior descending artery. Given the anatomy of the papillary muscle rupture, occlusion of the ramus intermedius artery most probably resulted in the patient’s mechanical complication. The obtuse marginal artery is one of the tributaries from the left circumflex artery.
This patient’s case is interesting, given the fact that the posteromedial papillary muscle was preserved despite total occlusion of posterolateral segment with associated inferior akinesis. However, the anterolateral papillary muscle has ruptured despite having a lower degree of coronary occlusion (95% of ramus intermedius artery) with preserved coronary flow, compared with posterolateral segment. The patient continued to require use of escalating vasopressors for cardiogenic shock. After identification of papillary muscle rupture, the patient was transferred to a quaternary care hospital for further management.

5. After diagnosing a mechanical complication following a STEMI, what would be the next best step in management for this patient?
   a) Temporary intra-aortic balloon pump (IABP)
   b) Transcatheter edge-to-edge mitral valve repair
   c) Surgical closure
   d) Percardiocentesis
   e) Medical management alone

As a general rule, mechanical complication following an acute MI requires urgent evaluation by cardiac surgery. For severe mitral regurgitation secondary to papillary muscle rupture, intra-aortic balloon pump (IABP) placement is the best option and improves mortality as a bridge to surgery. Counterpulsation from an IABP lowers afterload, thereby simultaneously improving forward flow to the aorta and reducing regurgitant fraction. Transcatheter edge-to-edge mitral valve repair may be considered in patients with papillary muscle rupture when surgical risk is prohibitively high, but there is no strong clinical evidence for this approach, and its benefit was only shown in case reports. Surgical closure is required for VSD. However urgent closure after diagnosis is associated with a mortality rate as high as 50%. Delaying closure after 7 days offers better prognosis by allowing for fibrosis of myocardium to take place, making the tissue stronger for suturing. Percardiocentesis in case of free-wall rupture should be considered, but the potential increase of the pressure gradient between the LV and the pericardium may also cause re-expansion of the rupture. Thus, pericardiocentesis should be considered in cases of acute hemodynamic collapse secondary to free-wall rupture as a bridge to urgent surgical intervention. While waiting for surgical intervention, medical management—such as inotropes and vasopressors—can be used to support hemodynamics in the setting of cardiogenic shock. If the arterial blood pressure is preserved, vasodilators may improve afterload and forward flow. In this case, because of cardiogenic shock, medical management (vasopressors) would only be used in conjunction with an IABP as a bridge to surgery.

On arrival to the quaternary care hospital CICU, the patient required higher doses of norepinephrine. Vasopressin was added and, because of worsening cardiogenic shock, he was sent to the cardiac catheterization laboratory for placement of an IABP. Cardiac surgery was consulted, but he was considered a poor candidate for surgery given his advanced age and medical comorbidities. After discussion with family over the risks and benefits of surgery, he was transitioned to comfort care. The patient died 2 days after presentation.

DISCUSSION
In this case, a patient had delayed presentation following an acute MI, resulting in a fatal mechanical complication. He had several comorbidities that increased his risk of mortality but, most importantly, his risk of mortality was substantially increased because of delayed presentation. Clinical presentation, including new murmur and acute heart failure, provided initial warning for a papillary muscle rupture. Prompt evaluation for mechanical complications is necessary before coronary catheterization. Early identification of a mechanical complication would result in emergent cardiac surgery consultation for consideration of simultaneous coronary bypass surgery and...
repair of mechanical defect. Moreover, the case highlights how quickly mechanical complications post-MI can decompensate. If no mechanical complications are present before catheterization, continued clinical surveillance for a new murmur is required for timely diagnosis.

Educating patients on the signs of a heart attack is crucial to avoid delayed presentation in patients with MI, particularly in cases of STEMI. On average, patients do not seek care until more than 2 hours after onset of symptoms.10 Possible reasons why patients present late are lack of knowledge of warning signs, atypical symptoms or stuttering presentation, visiting a primary care physician first, or taking medications other than nitrates.10 Health disparities also affect delays in care. At-risk patient populations, such as minorities, have increased risk for prehospital delay of more than 2 hours.10

Providing education and resources to the public for early recognition has been crucial for decreasing prehospital delay in patients. Fewer than 10% of patients after an acute MI recall speaking to a physician regarding what to do in case of a heart attack.11 As a result, the National Heart, Lung, and Blood Institute and the American Heart Association created the Act in Time to Heart Attack Signs campaign to educate patients and family members about heart attack risks, symptoms, and survival. The campaign recommends that primary care physicians, hospitalists, and cardiologists follow the TIME method to educate patients on heart attacks.

Talk to patients about their risks of developing heart attacks, concerning symptoms, and necessity of prompt action. Investigate any barriers that may prevent them from seeking prompt medical help. Make an action plan for a possible heart attack. Evaluate their understanding of the risk of avoiding or delaying treatment.

**POTENTIAL COMPETING INTERESTS**
The authors report no competing interests.

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**CORRECT ANSWERS:** 1. a. 2. c. 3. b. 4. d. 5. a.