Cardiogenic Shock: Review Questions

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QUESTIONS

Choose the single best answer for each question.

1. A 60-year-old man with a past medical history of hypercholesterolemia presents to the emergency department with 2 hours of crushing substernal chest pain radiating to his left arm, nausea, and diaphoresis. On examination, his blood pressure is 82/48 mm Hg, heart rate is 110 bpm, and oxygen saturation is 95% on 4 L of oxygen. He is in severe respiratory distress and has cold clammy extremities, an S_3 gallop, and bilateral crackles on auscultation. Electrocardiogram reveals ST elevation in the anterolateral leads and ST depression in the inferior leads. The patient is given aspirin, nitroglycerin, heparin, and intravenous fluids. Vasopressors are started to maintain blood pressure, but he remains hypotensive despite receiving 2 pressors. Which of the following is the most appropriate next step in management until the patient reaches the catheterization laboratory?
   (A) Add a phosphodiesterase inhibitor
   (B) Initiate cardiac glycosides
   (C) Insert an intra-aortic balloon counterpulsation
   (D) More aggressive fluid resuscitation
   (E) Sodium nitroprusside infusion

2. Which of the following steps has been shown to have a mortality benefit in patients with cardiogenic shock caused by to myocardial infarction (MI)?
   (A) Addition of glycoprotein IIb/IIIa inhibitors
   (B) β-Adrenergic agonists
   (C) Early cardiac catheterization followed by revascularization by percutaneous coronary intervention (PCI) or surgical revascularization
   (D) Initial medical stabilization with blood pressure control prior to catheterization
   (E) Thrombolytic infusion

3. A 65-year-old man with a history of type 2 diabetes presents to the emergency department with 4 hours of chest pain. Physical examination reveals a heart rate of 120 bpm and a systolic blood pressure of 62 mm Hg with a palpable pulse. Electrocardiogram reveals ST elevation in leads V_1 to V_4. The patient undergoes emergent cardiac catheterization followed by PCI. A pulmonary artery catheter is inserted for hemodynamic monitoring. Which of the following hemodynamic subsets satisfies the criteria for true cardiogenic shock in this patient?
   (A) Pulmonary capillary wedge pressure (PCWP) < 18 mm Hg; cardiac index > 2.2
   (B) PCWP > 18 mm Hg; cardiac index > 2.2
   (C) PCWP < 18 mm Hg; cardiac index < 2.2
   (D) PCWP > 18 mm Hg; cardiac index < 2.2

4. A 75-year-old woman with a past medical history of hypertension is admitted with acute anterior wall MI and is Killip class III on admission. She receives intravenous thrombolytic therapy, and her chest pain resolves. Seventy-two hours later, her chest pain suddenly recurs and she develops respiratory distress and becomes hypotensive. Physical examination reveals a new pansystolic murmur best heard at the left sternal border with thrill and S_3 gallop. Pulmonary capillary wedge tracings reveal some V wave prominence and oximetry reveals the following oxygen saturations: superior vena cava, 66%; inferior vena cava, 70%; right atrial, 70%; right ventricular, 80%; and pulmonary arterial, 82%. Which of the following is the most likely diagnosis based on these findings?

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(A) Cardiac rupture
(B) Mitral regurgitation due to papillary muscle rupture
(C) Ventricular aneurysm
(D) Ventricular pseudoaneurysm
(E) Ventricular septal rupture (VSR)

ANSWERS AND EXPLANATIONS

1. **(C) Insert an intra-aortic balloon counterpulsation.** Intra-aortic balloon counterpulsation is recommended for patients with MI when cardiogenic shock is not quickly reversed with pharmacologic therapy.[1] It is also used as a stabilizing measure prior to angiography and prompt revascularization.[1] The synchronous deflation and inflation of the balloon during the cardiac cycle reduces afterload during systole and augments blood flow in coronary circulation during diastole, respectively.[2] In addition to their positive inotropic effect, phosphodiesterase inhibitors also have some vasodilatory properties and should not be used in patients with low mean arterial pressure. Nitroprusside also has a vasodilatory effect and should not be used in low cardiac output states. Aggressive fluid resuscitation may be limited by acute pulmonary edema from left ventricular dysfunction in patients with cardiogenic shock. Digoxin can be used in shock to control heart rate but only if atrial arrhythmias exist.

2. **(C) Early cardiac catheterization followed by revascularization by PCI or surgical revascularization.** The SHOCK trial compared emergent revascularization for cardiogenic shock due to MI with initial medical stabilization and delayed revascularization.[3] The results of the study revealed a mortality benefit at 30 days that increased over time at 6 months and 1 year. The American College of Cardiology/ American Heart Association guidelines recommend early revascularization (either PCI or coronary artery bypass grafting) for patients aged 75 years or younger with ST elevation or left bundle-branch block who develop shock within 36 hours of MI and who are suitable for revascularization that can be performed within 18 hours of shock.[1] Patients admitted to hospitals without facilities for revascularization should be immediately transferred to a tertiary care center with such facilities.

3. **(D) PCWP > 18 mm Hg; cardiac index < 2.2.** Patients with cardiogenic shock due to MI have low cardiac output due to left ventricular dysfunction, resulting in a low cardiac index. The left ventricle's inability to pump forward causes pooling of blood in the pulmonary circulation, resulting in high PCWP. Answer A is not consistent with shock, answer B represents congestive heart failure without shock, and answer C correlates with hypovolemic shock.[4]

4. **(E) VSR.** It is important to recognize cardiogenic shock due to mechanical complications of MI. In this case, pulmonary artery catheterization with oximetry shows stepped-up oxygen saturation in the right ventricle and pulmonary artery that usually occurs with VSR. Although acute mitral regurgitation may cause stepped-up saturations on oximetry, it is usually seen in the peripheral pulmonary arteries and is associated with giant V waves on pulmonary artery tracings. Cardiac rupture usually has a very abrupt course with chest pain, syncope, and sudden death, whereas ventricular aneurysm or pseudoaneurysm alone may not cause a new-onset systolic murmur. VSR usually occurs after 2 to 5 days of acute MI but may also occur in the first 24 hours after MI. Echocardiography with color flow imaging can confirm the diagnosis.

REFERENCES

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