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An elderly man with syncope caused by right ventricular infarction and anomalous coronary vasculature

A 72-YEAR-OLD MAN presents to the emergency department after collapsing in his kitchen. He has no significant past medical history. An electrocardiogram (ECG) is obtained (FIGURE 1).

Physical examination

The patient is alert and oriented but anxious.

Vital signs. Oral temperature 35.4°C (95.8°F), blood pressure 94/63 mm Hg, pulse 57, respiratory rate 24, oxygen saturation by pulse oximetry 100% while breathing oxygen at 3 L/minute per nasal cannula.

Cardiac examination. Bradycardia with no murmurs, rubs, or gallops. Jugular venous distention 8 cm above the clavicle.

Chest. Clear to auscultation bilaterally without rales, rhonchi, or wheezes.

Extremities. No edema. Distal pulses 1 + out of 4, and thready.

Ten minutes after arrival, his pulse drops to 39 and his blood pressure drops to 57/35 mm Hg.

- **Pulse 39**
- **BP 57/35**
- **Lungs clear**
- **Jugular distention**
- **ST elevations in II, III, aVF**
- **ST depressions in V_1–V_4**

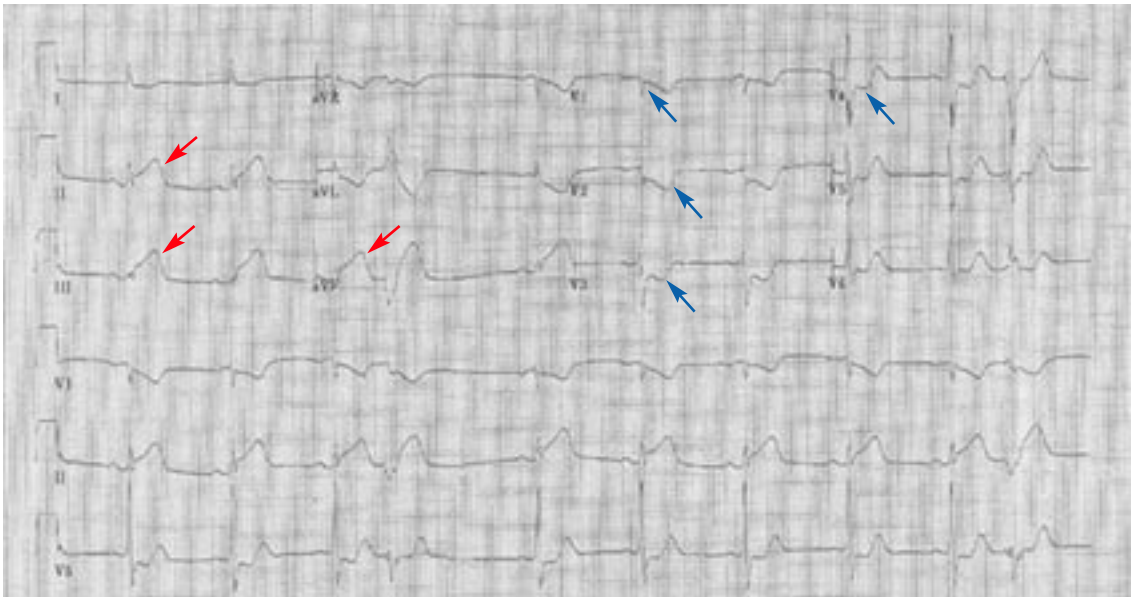

FIGURE 1. The patient's electrocardiogram on presentation to the emergency room. Note the ST-segment elevation in leads II, III, and aVF (red arrows) and the ST-segment depression in V_1, V_2, V_3, and V_4 (blue arrows).

MANAGING HYPOTENSION IN ST-SEGMENT ELEVATION INFARCTION

1 What would be the best initial therapy for this patient?

- ❑ Inotropic drugs
- ❑ Intravenous fluids
- ❑ Coronary reperfusion
- ❑ Pacing
- ❑ An intra-aortic balloon pump

Based on this patient's initial ECG showing ST-segment elevation in leads II, III, and aVF, and ST-segment depression in V_1, V_2, V_3, and V_4, the diagnosis of a right ventricular infarction must be considered, as it would influence treatment options. A right-sided ECG (in which the precordial leads are placed across the right side of the chest in a mirror image of the standard lead placement) would help clarify the diagnosis of a right ventricular infarction if it shows ST-segment elevation in leads V_3R and V_4R.

An ECG is essential to diagnose right ventricular infarction

Typical clinical features of right ventricular infarction include the triad of hypotension, jugular venous distention, and clear lung fields. However, an ECG is essential for diagnosis because these symptoms along with dyspnea can also be caused by a pericardial effusion or pulmonary embolism.

Another sign of a right ventricular infarction is severe hypotension that develops after patients receive nitrates for chest pain. This hypotension, which is due to the preload-dependent state of the right ventricle, typically resolves with aggressive volume resuscitation (see below). All of the treatments listed in this section are reasonable options.

Inotropic drugs improve contractility and hemodynamics in patients with hypotension by improving left ventricular-septal contraction,[1] but they should be used with caution because they may worsen preexisting myocardial ischemia. Dobutamine is preferred for patients with a right ventricular infarction: it has a better side-effect profile than milrinone and offers chronotropic stimulation. Dopamine is also an option.

Intravenous fluids increase ventricular preload and are the first step in managing a right ventricular infarction,[2] in which the noncompliant right ventricle is preload-dependent and any reduction in filling pressures may impair cardiac output. However, in left ventricular infarction, fluids should be given cautiously to avoid exacerbating pulmonary edema.

Even in a case of right ventricular infarction, giving too much fluid can also worsen cardiac output: aggressive volume resuscitation may compromise left ventricular diastolic function because of ventricular interdependence. Left ventricular filling may be compromised by right ventricular volume and pressure overload, leading to septal bowing toward the left ventricular cavity, ultimately decreasing cardiac output. Volume status can be difficult to clinically assess in critically ill patients with symptomatic hypotension.

Therefore, to optimize volume status, invasive hemodynamic monitoring with a pulmonary artery catheter is often appropriate. The pulmonary artery catheter allows one to measure the pulmonary artery wedge pressure and the cardiac index and detect mechanical complications early.

Coronary reperfusion improves left and right ventricular function in patients with right ventricular infarction. Compared with patients in whom reperfusion is attempted without success, those who undergo successful reperfusion have less hemodynamic compromise, shorter hospital stays, and lower rates of in-hospital mortality.[3] Even late reperfusion can help ventricular performance.

Pacing to optimize heart rate and rhythm should be considered in all patients with bradyarrhythmia or atrioventricular dyssynchrony. The right ventricle has a relatively fixed stroke volume and relies on heart rate to maintain adequate cardiac output. In patients with heart block, atrioventricular sequential pacing can dramatically improve ventricular performance.[4] A stiff right ventricle is sensitive to changes in volume, and loss of the atrial contribution to ventricular filling can seriously compromise cardiac output.

Intra-aortic balloon counterpulsation may not directly improve right ventricular hemodynamics, but by definition, patients with a right ventricular infarct have left ventricular ischemia. Therefore, an intra-aortic balloon pump can improve overall cardiac output in a hypotensive patient by improving

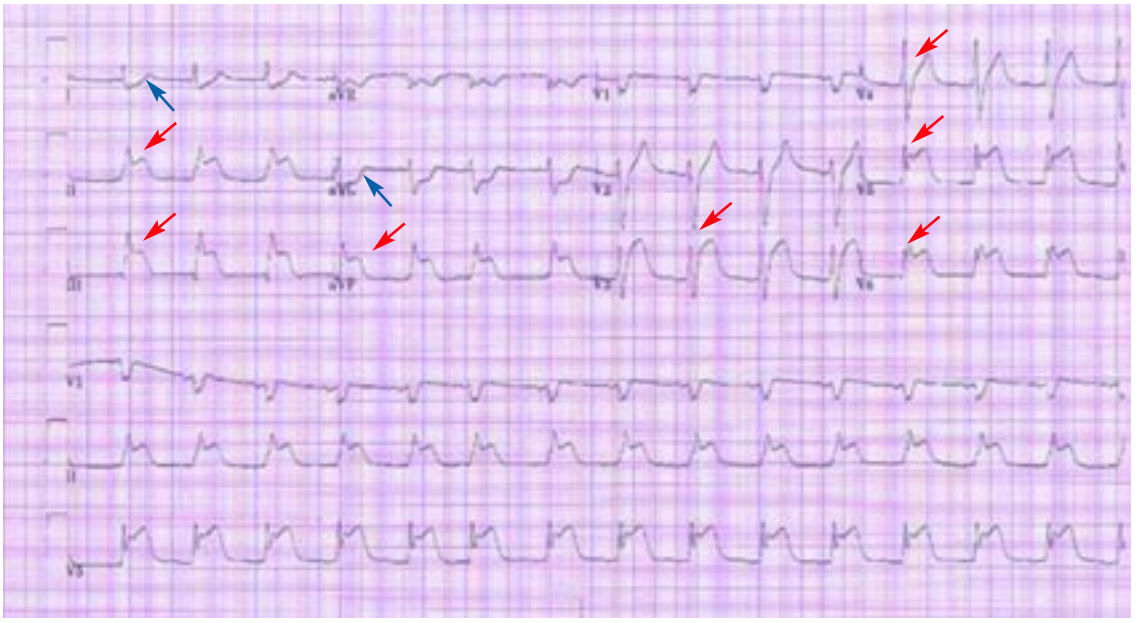

FIGURE 2. The patient's electrocardiogram approximately 45 minutes after the first one. Note the new ST-segment elevations in leads V_3–V_6 (red arrows) and pronounced ST-segment depressions in leads I and aVL (blue arrows).

coronary perfusion pressure but does not directly improve right ventricular function. Patients with concomitant left coronary artery disease that is not revascularized are also candidates for intra-aortic balloon pump insertion.

Case continued

The patient is given intravenous saline and a dopamine drip is started. He is intubated and externally paced. The pacing pads are disconnected after less than half an hour, and he is found to be in sinus rhythm.

WHAT IS THE NEXT STEP?

2 What is the most appropriate next step?

- ❑ An intravenous beta-blocker
- ❑ Thrombolytic therapy
- ❑ Cardiac catheterization
- ❑ Watchful waiting

Intravenous beta-blockers are not a good option, given the patient's hypotension.

Thrombolytic therapy could be considered but is typically less effective in patients with cardiogenic shock. Due to low blood pressure, the thrombolytic agent may not be adequately delivered to the coronary arteries and thus may not effectively treat an occlusion.

Cardiac catheterization is the best choice and should be done immediately. It can help to define the coronary anatomy and to treat occlusive atherosclerotic disease.

Watchful waiting would jeopardize this patient's likelihood of survival and is contraindicated so long as effective invasive medical therapy is an option.

By definition, patients with right ventricular infarction have left ventricular ischemia

Case continued: Coronary angiography

Another ECG is obtained and reveals new ST-segment elevations (**FIGURE 2**). The patient is taken for emergency left-heart catheterization, which shows:

- 100% proximal occlusion of the right coronary artery (**FIGURE 3**)
- An anomalous origin of the left coronary artery from the right sinus of Valsalva (**FIGURE 4**)
- 50% occlusion of the left main trunk
- 80% occlusion of the left anterior descending artery
- 80% occlusion of the first obtuse marginal artery.

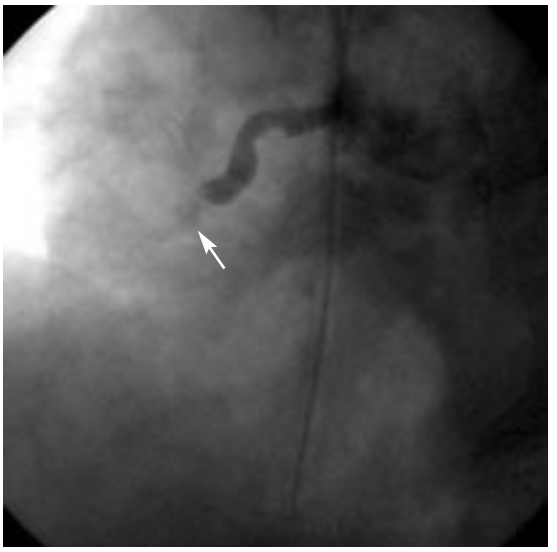

FIGURE 3. Angiogram revealing a totally occluded right coronary artery.

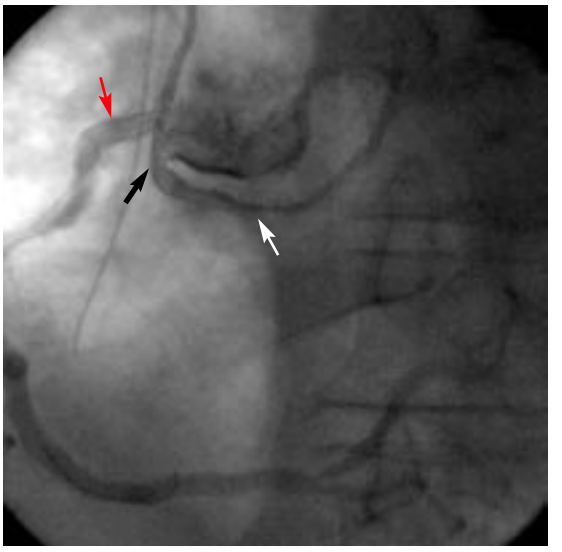

FIGURE 4. Anomalous origin of the left coronary system and a left main equivalent narrowing. Red arrow, right coronary artery; white arrow, left coronary artery stenosis; black arrow, anomalous origin of the left coronary artery.

Comment. These blockages explain the findings in the two ECGs: the right coronary artery obstruction is responsible for the initial inferior and posterior right ventricular infarct changes, and the jeopardized anomalous left coronary system is responsible for the anterior and lateral ST-segment elevations in the subsequent ECG.

Coronary anomalies are quite rare, occurring in about 1% of the population.[5,6] Left coronary arteries that arise from the right sinus of Valsalva are among the more infrequent of these anomalies, and when left coronary arteries traverse between the aorta and the pulmonary trunk, they are associated with sudden death among young people after exercise. Multiple factors are thought to contribute to sudden death in these cases, including compression of the coronary artery between the great vessels during exercise, angulation of the origin of the artery from the sinus, and vasospasm.

Transthoracic echocardiography is performed

The patient undergoes transthoracic echocardiography, which shows:

- Left ventricle—normal size and function
- Right ventricle—moderately dilated with severely decreased function
- Septal flattening
- No significant valvular disease.

The patient is diagnosed with an acute right ventricular infarction. His left ventricle is also jeopardized because of the anomalous left coronary artery.

COMPLICATIONS OF RIGHT VENTRICULAR INFARCTION

3 Which defect is associated with marked arterial desaturation?

- ❑ Ventricular septal defect
- ❑ Patent foramen ovale
- ❑ Tricuspid regurgitation
- ❑ Bradyarrhythmia and atrioventricular block

Coronary anomalies are rare, but some can be lethal

Ventricular septal defect is a complication that typically occurs 2 to 5 days after an infarction.[7] The septal defect creates a left-to-right shunt, further compromising the dysfunctional right ventricle, while cardiac output from the left ventricle falls and pulmonary edema develops. A clue is that patients with an acute ventricular septal defect can lie supine because pulmonary edema does not develop immediately. In contrast, patients with mitral rupture develop edema rapidly and cannot lie supine. Acute arterial desaturation is not characteristic of a ventricular septal defect, as blood flow would be from left to right.

Patent foramen ovale is the correct answer. Flow through a patent foramen ovale may be exacerbated by acutely increased right-sided pressures, causing a right-to-left shunt and hypoxemia. It should be suspected if hypoxia develops acutely and does not improve with supplemental oxygen.

Tricuspid valve regurgitation can be caused by papillary muscle rupture or ischemia, impairing right ventricular output. Dilation of the right ventricle and tricuspid valve annulus can also lead to functional valvular regurgitation. It is not typically associated with acute hypoxia.

Bradyarrhythmias and **atrioventricular block** can complicate right ventricular infarction. The mechanism can be either the Bezold-Jarisch reflex (high vagal tone inhibiting the heart rate) or ischemia of the atrioventricular node (in 90% of people, the blood supply to the atrioventricular node arises from a posteroventricular branch of the right coronary artery). Again, acute hypoxia is not associated with these arrhythmias.

The patient receives a bare metal stent, followed by grafting 11 days later

NATURAL HISTORY OF RIGHT VENTRICULAR INFARCTION

4 Which of the following statements is false regarding patients with right ventricular infarction?

- ❑ Half of inferior infarctions seen on ECG involve the right ventricle
- ❑ Patients frequently recover nearly normal right ventricular function
- ❑ Those with shock have an in-hospital death rate similar to that of patients with left ventricular shock
- ❑ Patients with right ventricular infarction are typically older and have a higher prevalence of previous myocardial infarction

Nearly half of inferior infarctions do involve the right ventricle. Fortunately, patients tend to recover right ventricular function almost completely within several months: echocardiographic and nuclear studies typically show full resolution of their depressed right ventricular ejection fraction. Even if right ventricular dysfunction persists, patients tend to continue to improve clinically.

However, a substudy of the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) registry of patients with suspected cardiogenic shock after acute myocardial infarction found that right ventricular infarction complicated by cardiogenic shock imposes a rate of in-hospital death comparable to that of left ventricular shock.[8]

The last answer above is false: patients with right ventricular shock are typically *younger* than patients with left ventricular shock and have a *lower* incidence of previous myocardial infarction, although they have similar rates of renal failure, peripheral vascular disease, prior revascularization, and cardiac risk factors such as hypertension, diabetes, and smoking.

Case continued: The patient recovers

The patient undergoes percutaneous placement of a stent in the proximal right coronary artery. The cardiologist uses a bare-metal stent rather than a drug-eluting stent so that coronary artery bypass grafting can be performed soon afterward. The left coronary lesions are not approached for stenting for several reasons: they are not in the artery implicated in the infarct, this patient's anomalous vasculature makes the procedure technically difficult, and surgery offers the most lasting benefit.

Almost immediately after revascularization, he develops unstable ventricular tachycardia, goes into cardiac arrest, and is resuscitated. He then goes into arrest four more times. An intra-aortic balloon pump is placed to maximize perfusion to the coronary arteries. A pulmonary artery catheter is placed to provide precise hemodynamic monitoring.

The patient is transferred to the cardiac intensive care unit. He remains critically ill and requires pressors and inotropic therapy to support the impaired right ventricle, but he is eventually weaned off these drugs and extubated, and the balloon pump is discontinued.

He undergoes a three-vessel bypass on the 11th hospital day. Soon after, he is discharged to a skilled nursing facility in good condition. He continues to do well and is discharged in good condition for cardiac rehabilitation.

TAKE-HOME POINTS

- Up to half of inferior myocardial infarctions involve the right ventricle.
- Typical features of a right ventricular infarction include hypotension, jugular venous distention, and clear lung fields.
- An ECG is essential for diagnosis of right ventricular infarction. ECG signs include inferior ST-segment elevation, ST-segment depression in V_1 and V_2, and, on a right-sided ECG, ST-segment elevations in V_3R and V_4R.
- Fluid resuscitation is the first step in treating right ventricular infarction, but overaggressive hydration can be harmful. A pulmonary artery catheter is helpful in monitoring volume. Inotropes improve contractility and hemodynamics in hypotensive patients. Coronary reperfusion has been shown to improve outcomes. Pacing should be considered in patients with bradyarrhythmia or atrioventricular dyssynchrony.
- An anomalous origin of the left coronary artery from the right sinus of Valsalva is very rare but can be associated with sudden death in young athletes.
- Patients tend to have near-complete recovery of right ventricular function within several months.
- Right ventricular infarction complicated by shock has an in-hospital death rate comparable to that of left ventricular shock.[8]

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