56-year-old man with a history of diabetes mellitus and hypertension presents with chest pain, emesis, pallor, and diaphoresis.

A 56-year-old man with a history of diabetes mellitus and hypertension presents with chest pain, emesis, pallor, and diaphoresis. The chest pain began the day before as intermittent, left-sided discomfort that subsequently became constant; over the past 2 hours, it has intensified.

Blood pressure is 115/78 mm Hg; heart rate, 65 beats per minute; and respiration rate, 30 breaths per minute. Neck veins are markedly distended; lung fields are clear. A 12-lead ECG (A) shows sinus rhythm with ST-segment elevation in leads II, III, and aVF; ST-segment depression is evident in leads I and aVL. These ECG findings are interpreted as acute inferior wall myocardial infarction (MI) with reciprocal ST-segment depression in the lateral leads.

The patient is given aspirin and sublingual nitroglycerin (0.4 mg) while an intravenous line is placed. Moments later, the patient vomits. Blood pressure is undetectable; heart rate is 90 beats per minute; ECG monitoring continues to show normal sinus rhythm. While intravenous saline is administered, a second ECG is obtained; it does not reveal interval change from the first. Then a third ECG, using right-sided precordial leads, is obtained (B).

To what diagnosis do these ECG tracings point? WHAT THE ECG SHOWS

This patient sustained an inferior wall MI with right ventricular infarction. The typical approach to diagnosis and management of acute coronary syndromes focuses on different anatomic segments of the left ventricle (eg, the inferior wall). Right ventricular MI is a distinct acute coronary syndrome.

Right ventricular MI is defined as hypotension, elevated jugular venous pressure, and shock in the presence of clear lung fields in patients with acute coronary syndrome. Clinically recognized right ventricular MI usually occurs in the setting of an acute left ventricular ST-segment elevation MI (STEMI). Isolated right ventricular MI is rare. ASSOCIATION WITH INFERIOR OR LATERAL WALL MI

In most patients with right ventricular MI, the inferior wall of the left ventricle is involved (usually in the form of a STEMI) as a result of occlusion of the right coronary artery proximal to the right ventricular branch. However, in a few patients (about 10% of those in whom right ventricular MI develops), the left circumflex artery supplies the right ventricle and a lateral wall STEMI occurs. The incidence of right ventricular MI varies significantly, depending on the method used to detect such injury. ECG studies have consistently shown that right ventricular MI occurs in about one third of patients with inferior wall STEMI. However, autopsy studies reveal that right ventricular MI occurs in more than half of patients with acute MI.
**CLINICAL FEATURES AND UNDERLYING PATHOPHYSIOLOGY**

Patients with right ventricular MI (Table) classically present with an inferior wall STEMI and typical symptoms and signs of an acute MI, and blood pressure that is low-normal initially. After administration of a vasodilator, such as nitroglycerin or morphine, significant hypotension develops (although on occasion, the patient may be hypotensive on presentation, before vasoactive medication is administered). Patients typically appear ashen and are diaphoretic; the neck veins are distended, and the lung fields are clear of vascular congestion. The ECG usually reveals a STEMI of the inferior and/or lateral wall (Figure 1); additional electrocardiographic imaging shows ST-segment elevation in the right ventricular distribution (Figure 2).

This characteristic clinical picture results from an acute failure of the right ventricle. With dysfunction of the right ventricle, left ventricular filling becomes largely dependent on preload, and the typical vasodilators used in patients with acute coronary syndromes have potent preload-reducing effects. Reduced left ventricular filling causes systemic hypotension, and less effective right-sided output results in jugular venous distention. The lung fields are clear (ie, there is no pulmonary edema) because the contractile dysfunction is proximal to the lungs. **CHARACTERISTIC ECG FINDINGS**
ECG Challenge: Hypotension in a Man With Acute MI
Published on Physicians Practice (http://www.physicianspractice.com)

Several findings on the standard 12-lead ECG suggest right ventricular acute MI, including ST-segment elevation in the inferior leads (II, III, and aVF) and in the right precordial chest leads, particularly lead V1 (see Figure 1). In fact, lead V1 is the only standard lead of the 12-lead ECG that directly images the right ventricle.

On occasion, coexisting acute MI of the posterior wall of the left ventricle may obscure the ST-segment elevation in lead V1 that is caused by right ventricular MI; this occurs in patients who have acute inferoposterior MI with right ventricular involvement. However, a particular pattern of the relative magnitudes of the ST-segment elevation in the various inferior leads—greater ST-segment elevation in lead III than in the other inferior leads—suggests right ventricular acute MI. This pattern of ST-segment elevation occurs because lead III reflects the activity of the right ventricle more directly than do the other inferior leads.

Use of right precordial leads. Nonstandard electrocardiographic imaging can also be useful when right ventricular MI is suspected. Both additional ECG leads and body surface mapping have demonstrated varying degrees of success as diagnostic aids in this setting. The addition of lead RV4 to the standard 12-lead ECG provides further objective evidence of right ventricular involvement: right ventricular MI can be diagnosed with 80% to 100% sensitivity if ST-segment elevation of more than 1 mm is seen in this lead. All the right-sided leads, RV1 through RV6, show evidence of right ventricular MI; however, use of lead RV4 alone has yielded rates of right ventricular MI diagnosis similar to those obtained when the entire array of right-sided ECG leads was used (see Figure 2). The ST-segment elevation in the right-sided leads is usually less pronounced than that seen on a standard 12-lead ECG in patients with left ventricular STEMI. This disparity results from the fact that the right ventricle is composed of considerably less muscle than the left ventricle; because there is less myocardium to manifest a current of injury, the ST segment is less elevated.

Body surface mapping. This recently developed— but not yet widely used—electrocardiographic technique involves placing numerous leads on a patient’s anterior and posterior chest, thereby enabling more complete visualization of cardiac electrical activity. Output from body surface mapping can be displayed in a 12-lead ECG format, in an 80-lead ECG format, and on color contour maps. The color contour maps can be displayed on a torso image or as flat maps. The body map approach to ECG imaging has increased the diagnosis of right ventricular MI in patients with acute coronary syndrome.

Importance of diagnosing right ventricular MI

Identifying right ventricular involvement is important because patients with coexisting inferior wall infarction have a large amount of jeopardized myocardium. Consequently, they are at increased risk for life-threatening complications, including high-grade atroventricular block, atrial fibrillation, symptomatic sinus bradycardia, atrial infarction (signaled by PR-segment displacement, elevation, or depression in lead II, III, or aVF), cardiogenic shock, and cardiopulmonary arrest. In fact, the complication rate seen in patients with this type of infarction is similar to that seen in patients with anterior wall acute MI. Aggressive therapy is often warranted to limit adverse events. Mortality and in-hospital complications were significantly reduced in patients with right ventricular acute MI (diagnosed on the basis of ST-segment elevation in lead RV4) who received fibrinolysis. Also, because nitrate-induced hypotension has been well described in patients with right ventricular MI, ST-segment elevation in lead RV4 should prompt cautious use of nitrates and other vasodilators as well as judicious administration of crystalloid infusions to avoid systemic hypotension.

Outcome of this case

In the ECG with right-sided leads (see Figure 2), ST-segment elevation was noted in leads RV1 through RV6; these findings are consistent with right ventricular MI in the setting of acute inferior wall MI. Further review of the initial 12-lead ECG revealed disproportionate ST-segment elevation in lead III.

After administration of 600 mL of normal saline, the patient's blood pressure was detectable at 105/60 mm Hg. He was transferred to the cardiac catheterization laboratory, where a proximal right coronary artery lesion with occlusive thrombus was detected and a stent was successfully placed. Hypokinesis of the right ventricle and inferior wall of the left ventricle was also noted. Serum troponin levels were elevated, confirming the diagnosis of acute MI. He had an uneventful recovery; good left ventricular function was restored.

References: REFERENCES:


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