Case Report

Cardiogenic Shock Due to Isolated Right Ventricular Infarction in an Elderly Woman

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A 78-year-old hypertensive woman with no prior history of ischemic heart disease arrived at the hospital complaining of weakness and profound fatigue. Four days earlier, she had experienced substernal chest pain associated with nausea and vomiting. A standard 12-lead electrocardiogram showed marked ST-segment elevation and negative T waves in leads V_1 and V_2. The patient was treated with anti-thrombotic therapy, dobutamine and dopamine infusions. Angiography showed proximal occlusion of a small, non-dominant right coronary artery and no clinically significant disease in the left coronary artery. Isolated right ventricular infarction accounted for the cardiogenic shock in this elderly patient. She received conservative medical treatment and was discharged in good condition.

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Right ventricular (RV) infarction is a strong independent predictor of low cardiac output, cardiogenic shock and in-hospital mortality in older patients with acute inferior myocardial infarction.[1,2] We describe a case of cardiogenic shock due to isolated acute RV infarction caused by the occlusion of a non-dominant right coronary artery in an elderly woman.

Case presentation

A 78-year-old woman with a history of hypertension was admitted to the hospital complaining of progressive confusion, weakness and profound fatigue developing over a few days. Four days earlier she had experienced substernal chest pain associated with nausea and vomiting over five hours.

Upon admission to the hospital the patient was confused, and her skin was cold and clammy. On physical examination, the blood pressure was 65/30 mmHg, the heart rate 52 bpm, the jugular venous pressure was elevated, and a hepato-jugular reflux was present. ST-segment elevation and negative T waves in leads V_1, V_2, V_{3R} and V_{4R} of the electrocardiogram (Figure 1) were consistent with massive RV infarction. The echocardiogram showed a dilated and akinetic right ventricle, with mild tricuspid regurgitation, paradoxical movement of the interventricular septum, and a normally contracting left ventricle. Hemodynamic measurements made during dobutamine administration showed a mean right atrial pressure of 15 mmHg, RV pressure of 28/15 mmHg, mean pulmonary capillary wedge pressure of 15 mmHg, and a cardiac index of 1.4 L/min/m^2. The serum creatinine kinase enzyme peaked at 434 IU/L.

The patient was placed on sc enoxaparine 60 mg bid, aspirin 160 mg/day and infusions of dobutamine 10 μg/kg/min, and dopamine 4 μg/kg/min. Normal saline was infused at a dose of 100 ml/h in order to maintain the central venous pressure between 12-16 mmHg. The patient was anuric on admission, and her serum creatinine concentration peaked at 4.1 mg/dl. The patient's clinical and hemodynamic condition progressively improved and the infusion of

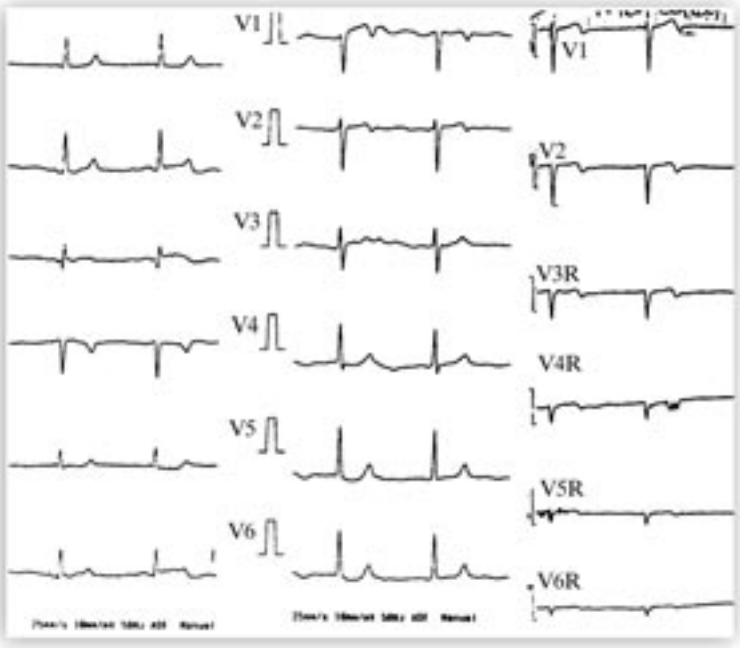

Figure 1. Electrocardiogram on admission.

inotropes was gradually discontinued. On day four, she developed atrial fibrillation associated with a rapid ventricular rate. Following spontaneous conversion to sinus rhythm a 7-second asystolic pause was observed and a temporary pacemaker was implanted. The patient underwent cardiac catheterization seven days after her admission, when her serum creatinine had returned to 1.2 mg/dl, mean right atrial pressure to 5 mmHg, RV pressure to 26/5 mmHg, mean pulmonary capillary wedge pressure to 9 mmHg, and cardiac index to 3.5 L/min/m^2. On coronary angiography, the left coronary system was dominant and patent (Figure 2) and a small right coronary artery was occluded proximally. The patient was managed medically and discharged on a regimen of aspirin 160 mg/day, metoprolol 25 mg bid, atorvastatin 10 mg/day.

Discussion

Right ventricular involvement is observed in 40% to 50% of patients presenting with acute transmural infero-posterior LV infarction, and is associated with an increased rate of hemodynamic instability and higher in-hospital mortality.[2,3] Isolated right ventricular infarction is an extremely rare phenomenon. Its electrocardiographic features may be misinterpreted, especially when a typical picture is lacking. Our patient's 12-lead electrocardiogram showed ST-segment elevation in the precordial leads V_1 and V_2, without any ST-segment elevation in the inferior leads. This case demonstrates that the electrocardiographic appearance of isolated right ventricular infarction may mimic an anterior wall infarct and can be easily missed if not suspected.

Cardiogenic shock caused by ischemia and infarction of the right heart is characterized by marked elevation of the right heart filling pressures, systemic hypotension and low cardiac output despite preserved LV systolic function.[3] The acute ventricular dilatation caused by occlu-

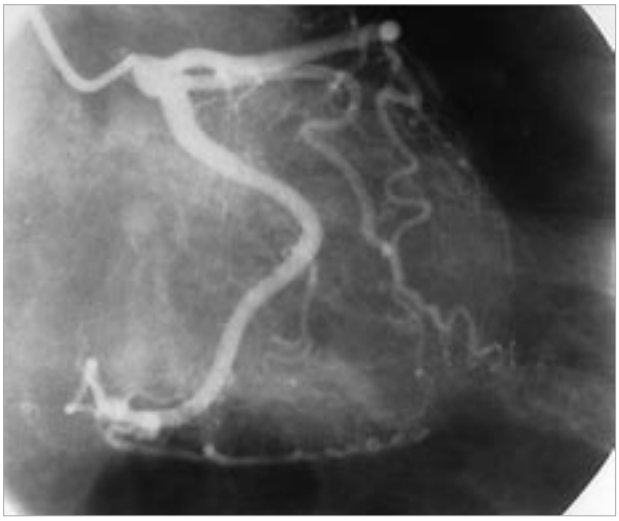

Figure 2. Selective left coronary angiogram in the right anterior oblique caudal projection demonstrating a patent dominant left coronary system.

sion of the RV coronary circulation results in elevated intrapericardial pressure, which, together with the increase in RV diastolic pressure, shifts the interventricular septum to the left, further limiting LV filling.[3-5] This "tamponade like" situation was caused by the equalization of diastolic filling pressures and was first observed on right heart catheterization. Furthermore, in the presence of depressed RV free wall contraction, the RV systolic pressure is partially generated by the mechanical displacement of the septum into the RV cavity during LV contraction.[4,5] The depressed systolic function of the septum caused by the usually associated LV infarction leads to a further deterioration of RV performance, while the insufficient loading of the LV results in a low output state.

In our patient, the occlusion of a non-dominant right coronary artery resulted in cardiogenic shock entirely attributable to RV dysfunction, since the posterior descending coronary artery supplying the septum originated from the left circumflex artery. Furthermore, the clinical manifestations observed may have been due in part to pathophysiologic changes during acute myocardial infarction that are specific to the elderly. Compared to the young, hearts of older age individuals are less compliant, have a smaller early filling volume, are more dependent on the contribution of atrial systole[6] and develop a higher ventricular end-diastolic pressure for any given end-diastolic volume. Our patient had had severely decreased cardiac output accompanied by increased right and also left ventricular filling pressures while under therapy with inotropes. Furthermore, cardiac output during stress in the elderly heart is primarily governed by the Frank-Starling mechanism. The catastrophic outcomes of RV infarction regularly observed in older patients are the consequence of several mechanisms that, combined with LV involvement, result in irreversible LV dysfunction.[7] In our patient, the pathologic process limited to the RV caused a profound decrease in cardiac output, with equalization of RV and LV filling pressures. The failure of cardiac output to increase after volume loading was most likely due to age-related loss of compliance of the LV and a tamponade-like situation caused by acute RV dilatation, perhaps aggravated by an age-related increase in pericardial stiffness. However, the infusion of inotropes for 4 days served as pharmacologic "counterpulsation" until the rapid return of better RV function, as has been previously reported.[7]

The ability of the RV to restore systolic function soon after the ischemic insult, even without reperfusion, along with the fact that the left ventricle was intact, might account for the patient's favorable outcome.

In conclusion, isolated RV infarction complicated by cardiogenic shock in an elderly patient may be reversed by conservative management, including the infusion of intravenous inotropes.

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