Case Reports

Unusual Electrocardiographic Presentation of Right Ventricular Myocardial Infarction

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Isolated right ventricular infarction is uncommon, but when it occurs its prompt recognition may alter therapy substantially. Electrocardiographic changes accompanying acute right ventricular infarction are variable and may be difficult to recognize. The case of a 40-year-old man who had right ventricular infarction with unusual electrocardiographic findings is presented. The clinical, hemodynamic, and electrocardiographic findings of right ventricular infarction are discussed. ***(Tex Heart Inst J 1996;23:305-9)***

In patients with acute myocardial infarction, a diagnosis of acute right ventricular infarction carries important prognostic information, influences pharmacologic therapy, and focuses the evaluation of cardiogenic shock.[1] Such a diagnosis should be considered in any patient with an infero-posterior current of injury on electrocardiography, or with ST-segment elevation in lead V_1 accompanied by an inferior wall current of injury. In a patient with acute inferior myocardial infarction, cardiogenic shock, clear lung auscultation, and engorged neck veins, the diagnosis of right ventricular infarction is almost certain. Electrocardiography using right-sided chest leads, especially V_4R, is recognized as a reliable means of diagnosing right ventricular infarction.[2-9] In fact, some[10] argue that the recording of a right-sided chest lead is an essential part of the initial evaluation of a patient with acute inferior myocardial infarction. Others maintain that analyzing the ST-segment vector from a standard 12-lead electrocardiogram (ECG), specifically searching for the effect of a right ventricular current of injury, is sufficient.[10] We report a case of isolated right ventricular infarction in which the electrocardiographic pattern simulated that of anterior wall myocardial infarction. Right ventricular infarction and its electrocardiographic manifestations are briefly discussed.

Case Report

A 40-year-old man was admitted to our institution with a 3-day history of atypical chest pain. Coronary atherosclerosis had been diagnosed at the time of an acute inferior myocardial infarction, 6 years prior to this admission. At that time, he underwent percutaneous coronary angioplasty (PTCA) of the distal right coronary artery. Two years later, he required PTCA of the mid left anterior descending coronary artery, after which he was able to discontinue anti-anginal medication.

During the 3-day period prior to admission, he experienced episodes of upper back and left shoulder "soreness" that lasted from 15 to 30 minutes and were not accompanied by dyspnea, diaphoresis, or nausea. There was no association with exercise. His physical examination showed nothing unusual. A resting ECG, which was unchanged in comparison with an ECG recorded 3 months earlier, revealed Q waves with T-wave inversion in leads III and aVF (Fig. 1). He was admitted to the hospital's telemetry unit and treated with aspirin, topical nitrates, and diltiazem.

Eight hours after admission, he developed severe, unremitting chest pain accompanied by ST-segment elevation in leads V_1 through V_5, II, and aVF (Fig. 2A). A diagnosis of acute myocardial infarction was made. Recombinant tissue-plasmin-

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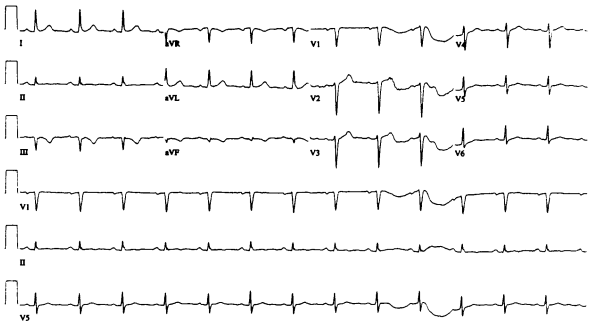

Fig. 1 *Twelve-lead electrocardiogram obtained at the time of hospital admission.*

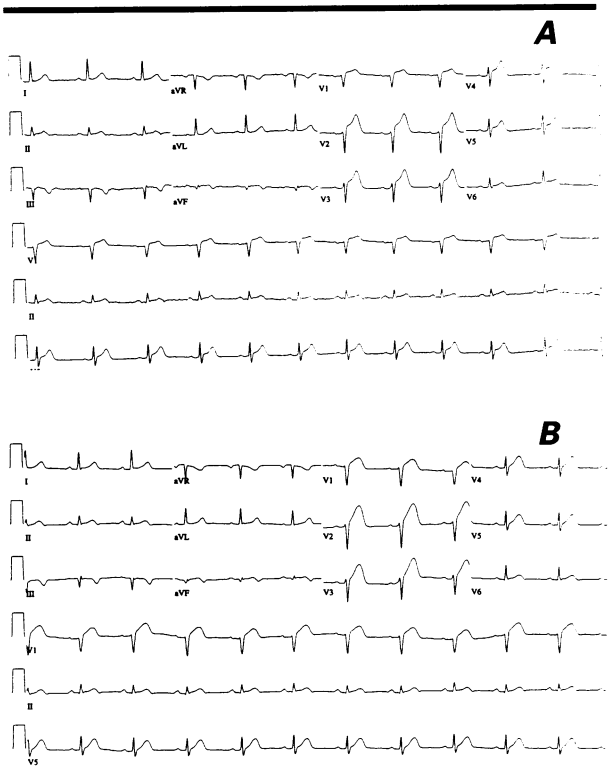

Fig. 2 ***A****) Anterior precordial ST-segment elevation, most prominent in lead* V_2*, is the most striking abnormality. Close scrutiny also reveals minor ST-segment elevation in leads II and aVF.* ***B****) After thrombolytic therapy, electrocardiographic evidence of acute injury was unchanged.*

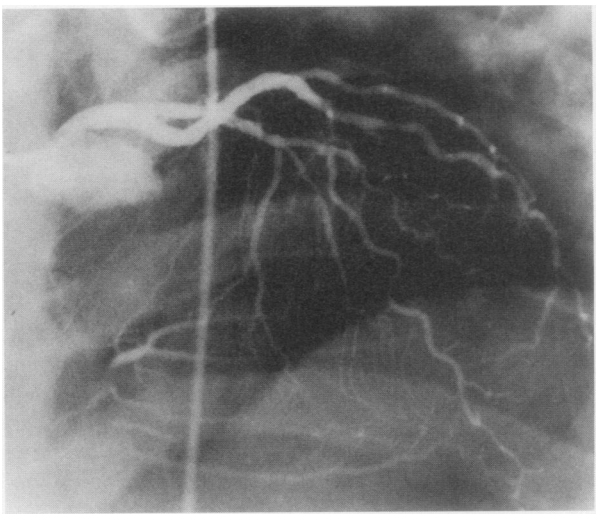

Fig. 3 *An antero-posterior view of the left coronary artery with cranial angulation reveals a mild narrowing of the left anterior descending coronary artery. More important, the posterior descending coronary artery and posterior left ventricular branches can be seen filling through collateral vessels.*

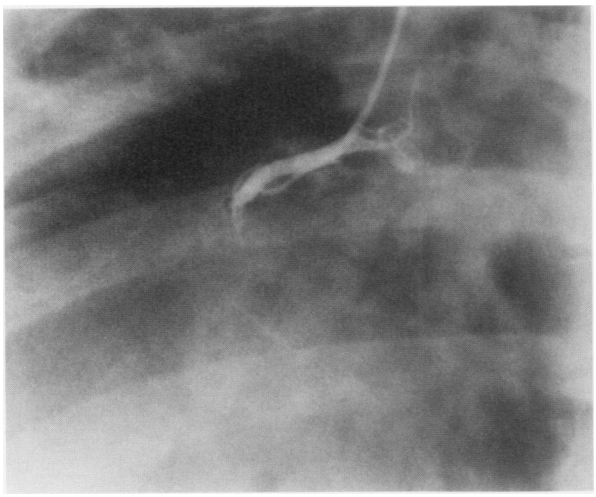

Fig. 4 *The right coronary artery was totally occluded before the origin of its right ventricular branches. The irregular borders and dye staining (not shown here) suggested that acute thrombus formation was the cause.*

ogen activator, intravenous heparin, and nitroglycerin were given. Ninety minutes after the initiation of thrombolytic therapy, the patient's pain had not resolved and ST segments remained elevated (Fig. 2B). The patient was taken to the cardiac catheterization laboratory where arterial access was achieved. Arterial pressure recording revealed a pulsus paradoxus of 36 mmHg. Angiography of the left coronary artery revealed mild narrowing of the mid left anterior descending coronary artery and collateral filling of the terminal right coronary artery distribution (Fig. 3). Angiography of the right coronary artery demonstrated thrombotic occlusion proximal to the branches that supplied the right ventricle (Fig. 4). After unsuccessful attempts to achieve patency with intracoronary urokinase, Abciximab (c7E3 Fab), and balloon angioplasty, a Palmaz-Schatz intracoronary stent was placed successfully. With restoration of coronary patency (Fig. 5), the patient reported complete relief of chest discomfort. Repeat electrocardiography revealed resolution of ST-segment elevation without development of anterior precordial Q waves (Fig. 6).

Serial creatine phosphokinase values reached a peak of 1,057 IU/dL, confirming acute myocardial infarction. Radionuclide ventriculography was per-

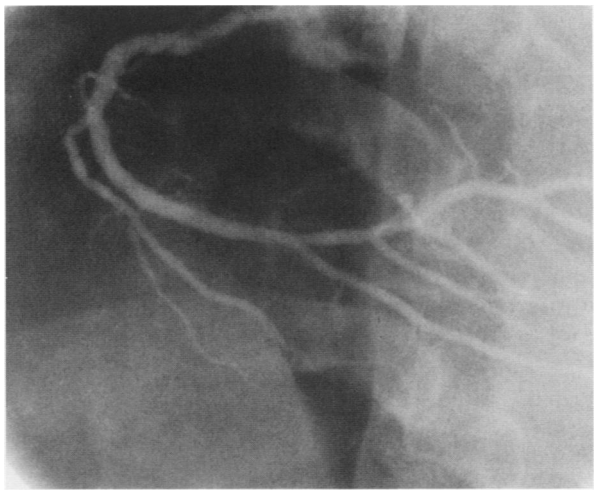

Fig. 5 *After numerous attempts with balloon angioplasty and eventually Palmaz-Schatz coronary stent placement, reliable patency of the right coronary artery was achieved.*

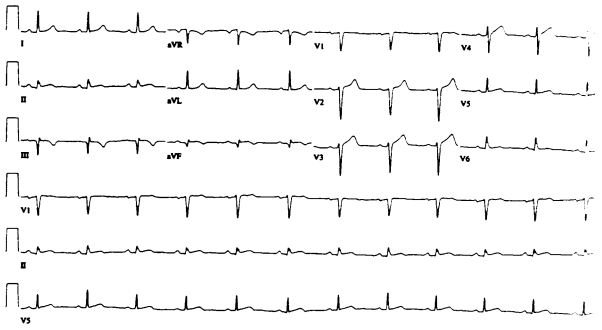

Fig. 6 *An electrocardiogram obtained after the angioplasty procedure reveals resolution of anterior ST-segment elevation without development of Q waves.*

formed the same day and revealed generalized right ventricular hypokinesis and moderate right ventricular dilation with a severely depressed ejection fraction. The left ventricular wall motion and ejection fraction were normal. Follow-up coronary angiography, performed 72 hours later, confirmed patency of the stented site.

Discussion

The pathophysiology, diagnosis, and treatment of right ventricular infarction have recently been reviewed in detail.[1] The right ventricle is protected from ischemia in some measure by its thin wall, low workload, and extensive sources of collateral blood supply. However, sudden occlusion of the right coronary artery proximal to its right ventricular branches, without antecedent development of collateral blood flow, may result in infarction of the free right ventricular wall. In most people, the right coronary artery is dominant, supplying the inferior left ventricle and the lower one third of the interventricular septum. The common vascular supply results in a close association between inferior infarction and right ventricular infarction.

Ischemic injury of the right ventricle may be difficult to detect during routine evaluation of a patient with a suspected myocardial infarction. The "classic" physical findings and hemodynamic aberrations resulting from right ventricular infarction may not be readily apparent. Without a clear, inferiorly directed current of injury (suggesting an associated right ventricular infarction) or prominent hemodynamic abnormalities, the diagnosis of right ventricular infarction might not be considered. Our patient had a predominantly anterior current of injury and was normotensive during nitrate exposure. The presence of pulsus paradoxus was not noted until the arterial pressure was recorded. As a result, our clinical suspicion was occlusion of the proximal left anterior descending coronary artery with anterior left ventricular ischemic injury. A review of the phenomenon of right ventricular infarction, highlighted by this case, is presented in order to assist in the recognition of similar cases in the future.

Impaired systolic function of the acutely infarcted right ventricular free wall reduces the right ventricle to little more than a conduit from the right atrium and venae cavae to the pulmonary artery. While a contracting interventricular septum may increase right ventricular pressure, the driving force maintaining pulmonary flow and cardiac output is increased systemic venous pressure supplemented by atrial contraction. As a result, right atrial pressures may equal or exceed left atrial filling pressures. Increased right ventricular wall stress produces right ventricular dilation, which is limited by the capacity of the pericardium. The enlarging right ventricle may physically limit left ventricular diastolic filling unless filling pressures are equal. The resultant physiology is much like that of pericardial constriction.[1]

Clinically, a patient with right ventricular infarction may present with jugular venous distention, normal auscultatory findings on lung examination, and hypotension. The requirement for high venous pressure to maintain adequate pulmonary flow and cardiac output results in extreme sensitivity to venodilators such as nitroglycerin.

A clinical diagnosis of right ventricular infarction may be made with certainty in patients with an inferior current of injury, hypotension, clear lung fields, and jugular venous distention. Fortunately, right ventricular infarction occurs in less than 25% of patients with acute inferior infarction. The finding of jugular venous distention alone is said to have a sensitivity of 88% and a specificity of 69%.[11] Hemodynamic data may confirm the diagnosis when right

atrial pressure is elevated and found to be equal to or greater than pulmonary artery occlusive pressure. In this setting the right atrial pressure recording may reveal a prominent Y-axis descent and frequently a diminished X-axis descent, indicating high right atrial pressure and the absence of right ventricular contractile function.

Electrocardiography is the most practical and easily accessible method for diagnosis of right ventricular infarction. In 1976, Erhardt and coworkers[2] reported the usefulness of a single right-sided chest lead. Heightened awareness of right ventricular infarction led to the exploration of a variety of electrocardiographic criteria, including ST-segment elevation in leads V_1, V_2, and V_2R through V_6R accompanying inferior infarction, and the ratio of the current of injury between leads III and II.[3-9,12] Lead V_4R provides the most reliable electrocardiographic evidence of right ventricular infarction: an ST-segment elevation greater than 0.1 mV in lead V_4R has a sensitivity of 88% and a specificity of 78%.[9]

Noninvasive imaging is useful in most patients in whom the diagnosis of acute right ventricular infarction is suspected but clinical criteria are not diagnostic. Echocardiography discloses right ventricular enlargement, free wall asynergy, paradoxical septal motion and, in severe cases, bowing of the interatrial septal wall to the left. Doppler evaluation of hepatic vein flow may also display diminished systolic flow and accentuated diastolic flow corresponding to the abnormal X- and Y-axis descents of the atrial pressure recording.[1]

Radionuclide ventriculography is the best available noninvasive method for evaluating right ventricular systolic function. In patients with suspected right ventricular infarction, the presence of a dilated right ventricle with a depressed ejection fraction is reported to carry a 92% sensitivity and 82% specificity for the diagnosis.[13] The use of dual isotope SPECT (single photon emission computed tomography) with technetium-99m pyrophosphate and thallium-201 appears reliable for the identification of even partial right ventricular infarction. Fernandez and colleagues[14] applied this unique method with overlay of the images obtained with each isotope, and found that 30 of their 190 patients with acute myocardial infarction had right ventricular involvement.[14] Sensitivity and specificity values of dual isotope imaging for the detection of right ventricular infarction have not been reported.

In the case presented here, acute myocardial infarction produced a current of injury primarily in the anterior precordial leads. On close inspection, however, slight ST-segment elevation was also recorded in leads II and aVF. This finding is consistent with an antero-inferior current of injury vector and is in contrast to the typical vector in acute anterior wall myocardial infarction, which has an antero-superior current of injury vector. Radionuclide ventriculography, performed less than 8 hours after angiography, confirmed the diagnosis of right ventricular infarction, revealing a dilated, hypokinetic right ventricle with a severely depressed ejection fraction. Normal left ventricular wall motion and systolic function suggested that this was an isolated right ventricular infarction. Although the right coronary artery was dominant, supplying the inferior and posterior left ventricle, protection by collateral blood supply probably accounted for the relative sparing from ischemic injury.

Anterior ST-segment elevation has been described in patients with right ventricular infarction.[15,16] In 1984, Geft and co-authors[16] reported a study of 69 patients with an anterior injury pattern who were treated with thrombolytic therapy. Of these, 5 were found to have an occluded right coronary artery, and most had inferior ST-segment elevation as well. Clues to the diagnosis were a coexistent inferior current of injury and an abnormal pattern of ST-segment evolution in the anterior leads. In patients with anterior left ventricular infarction, the ST-segment elevations were the lowest in lead V_1, increasing in amplitude toward V_5. On the other hand, patients with an occluded right coronary artery and apparent right ventricular infarction displayed the greatest amplitude ST-segment elevation in lead V_1, decreasing toward V_5.

The diagnosis of right ventricular infarction has become so closely associated with acute inferior injury that it may not be thought of as a distinct entity. Nevertheless, making this distinction may substantially alter therapy, especially in hemodynamically unstable patients. Inotropic support and mechanical assistance with intra-aortic balloon pumping are frequently provided to hemodynamically unstable patients with acute myocardial infarction. Both of these interventions, however, carry a substantial risk of potentially fatal complications, including exacerbation of rhythm disturbances, and arterial injury requiring surgical exploration. In contrast, hemodynamically unstable patients with right ventricular infarction will frequently respond to simple intravenous volume administration. Our experience demonstrates that right ventricular infarction may be difficult to recognize, requiring both a high index of suspicion for its presence and careful evaluation of current of injury vectors.

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