Electrocardiographic Changes Associated With Isolated Right Ventricular Infarction

MERVYN B. FORMAN, MB, MRCP, JULIA GOODIN, MD, BRIAN PHELAN, MD, HARRY KOPELMAN, MD, RENU VIRMANI, MD

Nashville, Tennessee

Isolated infarction of the right ventricle is an extremely rare entity. A patient is described with diffuse interstitial lung disease who developed ST segment elevation in inferior and anterior leads on a routine electrocardiogram and at autopsy was found to have an isolated right ventricular infarct involving approximately 70% of the right ventricular circumference without involvement of the left ventricle and septum. This case illustrates that isolated right ventricular infarction in the presence of cor pulmonale and right ventricular hypertrophy can produce an injury current in the limb and precordial leads of the electrocardiogram which mimics that seen in typical transmural infarction of the left ventricle.

Right ventricular infarction is usually associated with transmural inferior infarction of the left ventricle (1–4). Isolated infarction of the right ventricle is extremely rare with only a few cases having been described in early reports (5,6). We report a case of an isolated right ventricular infarction proven pathologically in a patient with cor pulmonale who presented with striking ST segment elevation in the anterior and inferior leads on a 12 lead electrocardiogram. This case demonstrates that a large infarction confined to the right ventricle can produce electrocardiographic changes that mimic the electrocardiographic pattern seen in acute anterior and inferior myocardial infarction.

Case Report

A 56 year old black man presented with a 6 month history of palpitations and shortness of breath. Diffuse interstitial lung disease was diagnosed at that time and he was treated with prednisone and isoniazid. He remained fairly asymptomatic with only occasional episodes of shortness of breath. He presented 36 hours before death to a local hospital complaining of palpitations and shortness of breath.

Clinical and electrocardiographic findings. His physical examination was unremarkable except for diffuse rhonchi and crepitations in both lung fields. A chest radiograph revealed diffuse interstitial lung disease. His initial electrocardiogram showed small R waves in leads II, III and aVF, an indeterminate QRS axis in the frontal plane and poor R wave progression in the precordial leads (Fig. 1A). He was admitted and treated with digoxin and diuretic drugs and his condition appeared to improve over the next 12 hours. On the following morning, he was found unconscious on the bathroom floor. He was deeply comatose with a pulse rate of 130 beats/min. Arterial blood gas determination on 25% nasal oxygen revealed a partial pressure of oxygen (Po_2) of 46 mm Hg and artificial ventilation was promptly instituted. An electrocardiogram done soon afterward demonstrated left axis shift, deep Q waves in leads III and aVF and marked ST elevation in the inferior (II, III and aVF) and precordial (V_1 to V_4) leads (Fig. 1B).

He was transferred to this medical center for further assessment and on arrival was unresponsive to painful stimuli with only occasional spontaneous respirations. The pupils were reactive with "doll's eye" movements and occasional decerebrate posturing was noted with painful stimuli. The blood pressure was 110/70 mm Hg on admission and jugular venous pressure could not be adequately assessed. However, a prominent third heart sound was present on auscultation. The chest examination and electrocardiogram were unchanged (Fig. 1B). Serum cardiac enzymes over the ensuing 36 hours revealed a peak total creatine kinase increase to 1,936 mg with a 13.4% MB fraction. He became progressively hypotensive in spite of large doses of dopamine and died 12 hours after admission.

Postmortem findings. Postmortem examination was performed on the following day. On in situ examination of

From the Departments of Cardiology and Pathology, Vanderbilt University Medical Center, Nashville, Tennessee. Manuscript received January 10, 1984; revised manuscript received April 10, 1984, accepted April 17, 1984.

Address for reprints: Mervyn B. Forman, MD, Division of Cardiology, Vanderbilt University School of Medicine, Nashville, Tennessee 37232.

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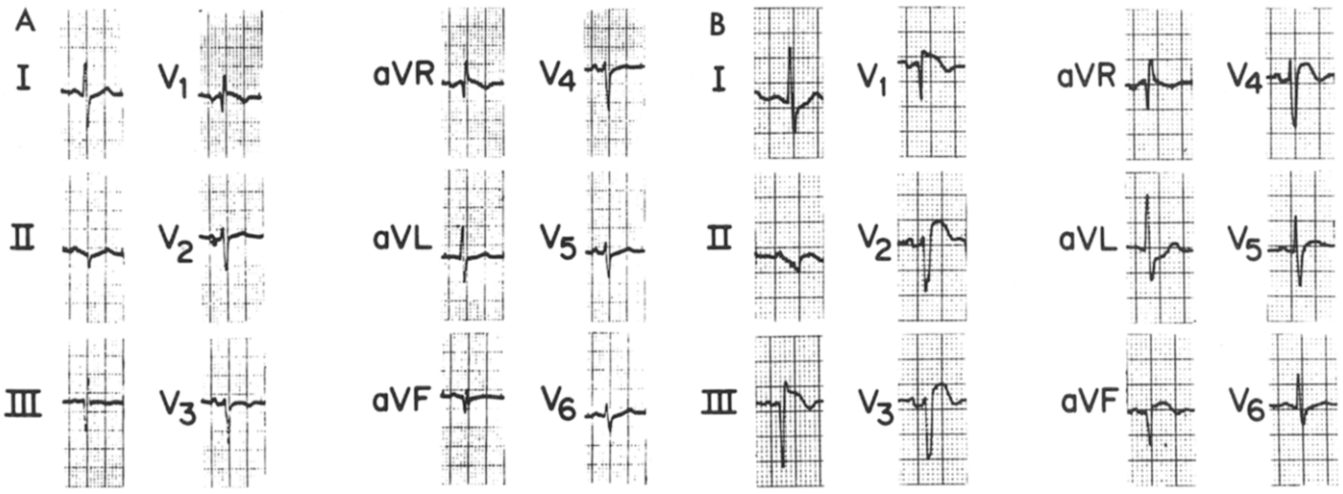

Figure 1. A, Admission electrocardiogram showing small R waves in leads II, III and aVF, an indeterminate QRS axis in the frontal plane, conduction defect of a right bundle branch variety and poor R wave progression in the precordial leads. **B,** Electrocardiogram obtained later reveals left axis shift, definite Q waves inferiorly and significant ST elevation in the inferior and precordial leads (V_1 to V_4).

the heart, it was noted that the right ventricle was rotated anteriorly; it was markedly dilated with transmural myocardial infarction of the lateral and inferior walls (Fig. 2), which by histologic examination was dated 1 to 2 days old. A portion of the anterior wall of the right ventricle that was not infarcted was hypertrophied. The left ventricle on gross and microscopic examination did not reveal areas of fibrosis or necrosis. Postmortem injection of the coronary arteries revealed a patent left coronary system (Fig. 3). A dominant right coronary artery showed arterial wall irregularities with up to 50% diameter reduction, and a nonocclusive thrombus was seen extending into the right marginal artery. On histologic examination of the four major coronary arteries, less than 25% cross-sectional area luminal narrowing was present despite the irregularities noted on the angiogram. The lungs showed extensive diffuse interstitial fibrosis with fibrointimal proliferation of muscular pulmonary arteries compatible with long-standing pulmonary hypertension without evidence of pulmonary emboli. Sections of the coronary and pulmonary arteries revealed no evidence of vasculitis.

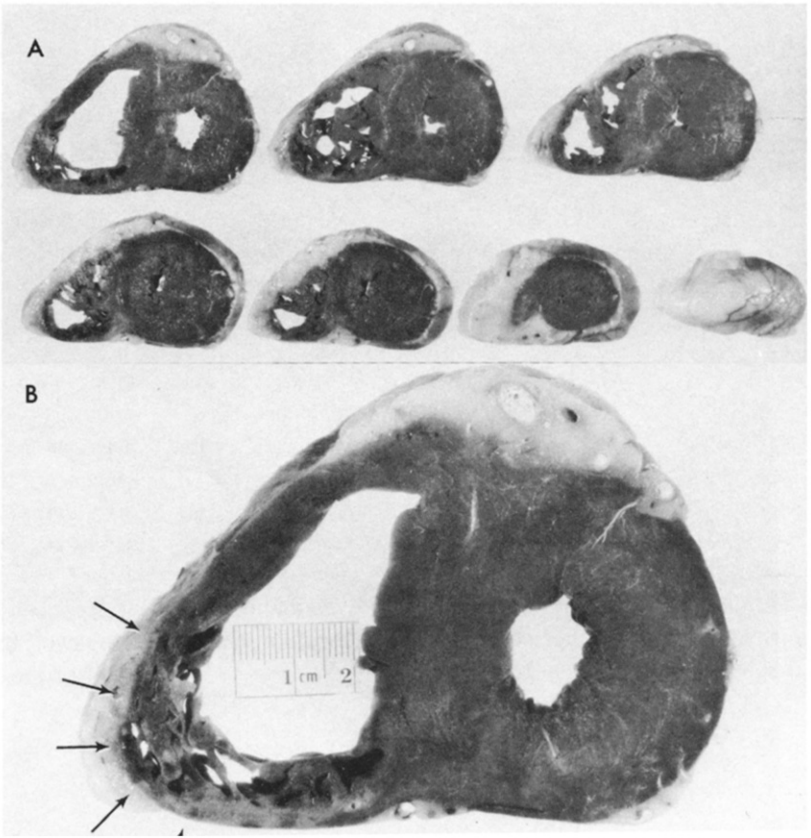

Figure 2. A, Slices of right and left ventricle showing acute transmural infarction of the right ventricle, involving the inferior and lateral walls and extending from base to apex. Note the absence of acute or healed infarction of the left ventricle. **B,** The basal slice showing acute infarction of inferior and lateral walls of the right ventricle **(arrows)** and right ventricular hypertrophy of the anterior wall of right ventricle.

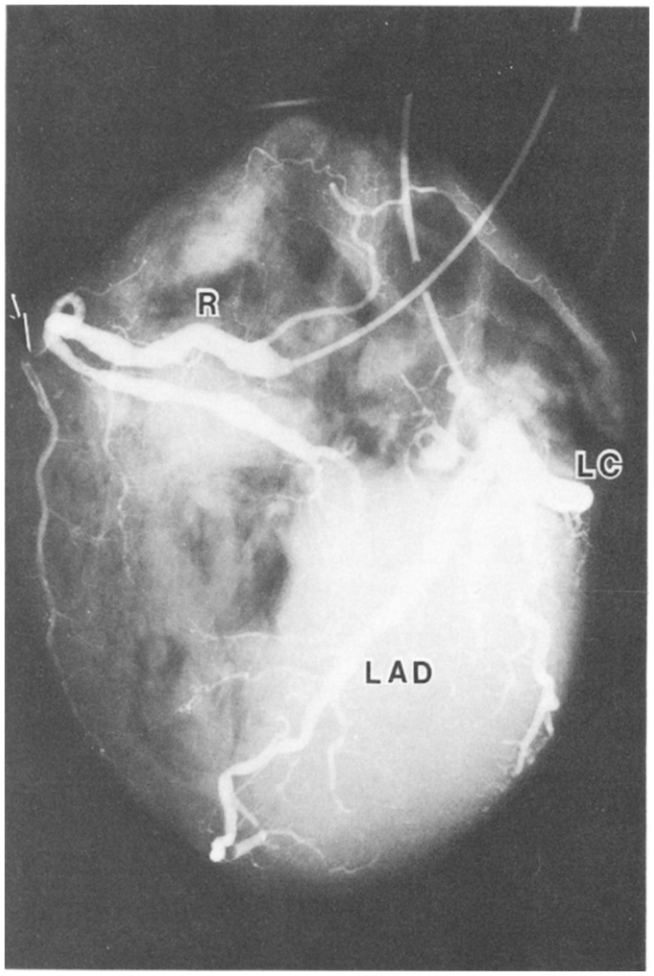

Figure 3. Postmortem coronary angiogram revealing irregularities in the right (R) and left coronary arteries without significant atherosclerosis. Note that the total occlusion of the right marginal artery (**arrow**). LAD = left anterior descending coronary artery; LC = left circumflex coronary artery.

Discussion

Incidence of right ventricular infarction. The clinical and pathophysiologic importance of right ventricular infarction was first described by Cohn et al. (7) in 1974. Since then, numerous studies (2,7–18) describing the clinical, hemodynamic, echocardiographic, electrocardiographic and scintigraphic features of this entity have been published. Most cases of right ventricular infarction are associated with damage to the inferior wall of the left ventricle (1). All patients in the study of Isner and Roberts (1) had at least 75% narrowing of the dominant artery supplying the inferior left ventricular wall. The association of right ventricular infarction with infarction of the inferior wall of the left ventricle has been further confirmed by echocardiographic and scintigraphic studies (2,3,13,17).

Isolated right ventricular infarction, although mentioned in early medical reports, appears to be extremely rare, with only two cases being reported in 160 autopsies (5). Wade (6) reported 11 cases of right ventricular infarction in 1959; three of these patients had isolated right ventricular infarction. In our patient, there was an isolated recent right ventricular infarction involving inferior and lateral walls approximating 70% of the right ventricular circumference.

Electrocardiographic diagnosis of right ventricular infarction. The value of the electrocardiogram in diagnosing associated right ventricular infarction has recently received attention (15–19). Chou et al. (15) found that ST segment elevation in the right ventricular leads V_3R to V_6R was a good predictor of associated right ventricular infarction with V_4R having the greatest sensitivity and predictive accuracy. In our patient, marked ST elevation was present in both the inferior and anterior precordial leads and was associated with the development of Q waves in inferior leads. To our knowledge, these electrocardiographic changes due to isolated right ventricular infarction have not been previously reported. We recognize that when the electrocardiogram showing injury current and Q waves (Fig. 1B) was taken, there could have been coexisting ischemia of the anterior and inferior aspects of the left ventricle. However, since 36 hours elapsed from the time this electrocardiogram was taken and the patient's death, we feel that infarction of the left ventricle would have been detectable on histologic examination had it been present. We speculate that since our patient had diffuse interstitial lung disease with cor pulmonale, the increased right ventricular mass and possibly the clockwise rotation simultaneously allowed detection of right ventricular ischemia or infarction in conventional electrocardiographic lead placements. At autopsy, transmural right ventricular myocardial infarction of the inferior and lateral walls was found.

Mechanisms of right ventricular infarction. The inferior wall of the right ventricle is usually supplied by the posterior descending branch of the right coronary artery (1). The lateral surfaces are supplied by the anterior marginal branch of the right coronary artery (20). The anterior wall usually has a dual blood supply from the conus branch of the right coronary artery and from the moderator branch artery arising from the left anterior descending coronary artery (1,20). The reasons for the rare occurrence of isolated infarction of the right ventricle are not well understood. Mechanisms include the smaller muscle mass of the right ventricle, the lower wall tension in the right ventricular cavity and the potential for collateral flow to the right ventricle (6,20). The partial occlusion of the right coronary artery and total occlusion of the marginal branch found in our patient could account for infarction of the lateral wall. However, the presence of extensive infarction of the inferior wall of the right ventricle could not be explained anatom-

ically. The right coronary artery and its posterior descending branch did not contain significant stenotic lesions. In addition, we were not able to detect any disease of the left coronary system. No mural thrombus was present in the left atrium or left ventricle. Histologic sections of the coronary arteries revealed no evidence of vasculitis. Since our patient had evidence of marked right ventricular hypertrophy and long-standing pulmonary hypertension, it is conceivable that an increase in extravascular compressive forces on the ventricle could have precipitated ischemia.

Conclusion. In our patient with cor pulmonale, displacement of the right ventricle anteriorly allowed detection of ischemia on routine electrocardiographic leads. Right ventricular infarction contributes to enzymatic estimates of infarct size among patients with inferior transmural infarction and accounts for the better prognosis than that for patients with anterior infarcts with similar enzymatic injury (11). However, our patient demonstrates that in some cases, massive right ventricular infarction may be associated with a high mortality.

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