Right Ventricular Infarction Mimicking Extensive Anterior Infarction*

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Two patients with inferior infarction complicated by right ventricular infarction are presented. Both manifested electrocardiographic changes involving the anterior chest leads with initial S-T segment elevation followed by loss of R waves and the development of QS complexes mimicking anterior infarction. Cardiac catheterization showed right coronary artery occlusion with normal left coronary system and anterior wall motion in each case. Radionuclide angiography showed dilated poorly contracting right ventricles. The ECG changes of "anterior infarction" in these patients were therefore due solely to right ventricular injury.

The diagnosis of right ventricular infarction is usually suspected when the jugular venous pressure is elevated in a patient with inferior myocardial infarction. It is known that electrocardiographic changes in V1 and V6 may reflect right ventricular infarction and V4R is frequently recorded when right ventricular involvement is suspected.

The two patients presented here demonstrate that more extensive anterior electrocardiographic changes including loss of R waves and the development of QS complexes, in the setting of inferior infarction, may represent right ventricular infarction and not infarction of the anterior wall of the left ventricle.

Case Reports

Case 1

A 37-year-old man presented with symptoms of acute myocardial infarction. Apart from raised jugular venous pressure, the examination was unremarkable. Chest x-ray film findings were normal. The electrocardiogram showed sinus bradycardia and first degree A-V block with evidence of acute inferior infarction. In addition, there was ST-segment elevation in the anterior chest leads from V1 to V6 (Fig 1). Equilibrium radionuclide angiography (ERNA) was performed and this showed a left ventricle contracting well, with an ejection fraction of 52 percent and posteroanterior hypokinesia. The right ventricle was markedly dilated and poorly contracting (Fig 2). The clinical course was uncomplicated apart from complete heart block necessitating the insertion of a temporary transvenous pacemaker. The electrocardiogram showed transmural inferior infarction as well as QS complexes from V4 to V6 (Fig 1).

Cardiac catheterization performed seven days after presentation confirmed elevated right ventricular end-diastolic pressure. The right coronary artery was totally occluded and the left coronary artery was normal. Left ventricular angiography showed inferior akinesia with normal movement of the anterior wall and septum. The patient was well on discharge from the hospital.

Case 2

This patient, a 51-year-old man, presented with acute, severe retrosternal chest pain. The jugular venous pressure was elevated and the rest of the examination was normal. Chest radiographic findings were normal. The electrocardiogram showed first degree A-V block, ectopic atrial rhythm, acute inferior infarction plus ST-segment elevation in V1 to V6 (Fig 3). The electrocardiogram evolved to show transmural inferior infarction and loss of R waves anteriorly. The ERNA study showed the left ventricle contracting well, with an ejection fraction of 67 percent. The right ventricle was dilated and contracted poorly. Cardiac catheterization after seven days showed a blocked right coronary artery, but normal left coronary system. The anterior wall and septum contracted well. The clinical course was complicated by pericarditis and complete heart block requiring temporary pacing. The patient was well on discharge from hospital.

Discussion

Right ventricular infarction is a known complication of inferior myocardial infarction. It is known that ST-segment elevation in the right precordial leads, especially V4R and V6, may in the setting of inferior infarction represent right ventricular involvement. It has recently been shown that ST-segment elevation due to right ventricular infarction may extend from V1 to V6 resembling the pattern of transmural ischemia of the anterior wall of the left ventricle. We are unaware of reports of loss of R waves or development of QS waves in the anterior chest leads in cases of documented right ventricular infarction without left ventricular anterior wall infarction.

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**FIGURE 1.** Case 1. A. The electrocardiogram on day 1 shows acute inferior infarction plus ST-segment elevation from V1 to V6. B. The electrocardiogram 10 days later shows transmural inferior infarction as well as QS complexes from V4 to V6.
In an early report, Roesler and Dressler\(^7\) described five patients whose electrocardiograms showed acute inferior infarction and simultaneous changes in the right precordial leads compatible with anteroseptal infarction. Post-mortem examination in all cases showed infarction of the interventricular septum that extended from the anterior to the posterior aspect of the heart and involved variable portions of the anterior or posterior wall adjacent to the septum. Septal infarction was excluded in our patients by demonstration of normal septal motion both on ERNA and biplane contrast left ventriculography.

We believe that the two cases presented above illustrate that these changes do occur without left ventricular anterior wall or septal infarction. They might be explained by progressive right ventricular dilatation and the development of clockwise rotation, such that leads \(V_1\) to \(V_5\), or \(V_4\), record right ventricular events as is sometimes the case in acute pulmonary embolism.\(^6\) The recognition of this electrocardiographic pattern has important prognostic and therapeutic implications.\(^8\)

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REFERENCES

Clinically significant cardiac amyloidosis may manifest as restrictive cardiomyopathy or, less frequently, as ischemic heart disease. Sudden death is relatively frequent, mainly caused by malignant arrhythmias such as asystole, marked bradycardia and sustained ventricular tachyarrhythmias. Amyloid deposits inside and around the conduction system have been described in a limited number of cases, correlating with conduction disturbances, and/or autonomic disorders. Accordingly, the clinicopathologic study of the present patient with cardiac amyloidosis, exhibiting arrhythmias and severe cardiocirculatory disorders, has been focused upon the specialized conduction system, the cardiac nerve plexus and, for the first time, upon the aortocoronary paraganglia.

**Case Report**

A 62-year-old man was admitted to our cardiac department because of dyspnea and dry cough.

**Investigation and Clinical Course**

The ECG showed first degree atioventricular (A-V) block (PR 0.22s), left anterior fascicular block, low QRS voltage potentials and anteroseptal pseudo-infarct aspect. Later, right bundle branch block supervened. Chest x-ray film demonstrated mild cardiomegaly with Kerley B lines at both costophrenic angles. The M-mode and 2-D echocardiogram showed increased thickness of the walls of both ventricles with sparkling granular appearance; the ventricles appeared hypokinetic without chamber dilation.

**Table 1—Electrophysiologic Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Values</th>
<th>1st Electrophysiologic Testing</th>
<th>2nd Electrophysiologic Testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max Sinus Node Recovery Time (Corrected) (msec)</td>
<td>450±50</td>
<td>1400 (450)</td>
<td>1760 (890)</td>
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<td>Sinusatrial Conduction Time (msec)</td>
<td>232±15</td>
<td>370 (395)</td>
<td>445 (not retested)</td>
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<td>Atrial Effective Refractory Period (msec)</td>
<td>273±9</td>
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<td>150 (not retested)</td>
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<td>Basal AH Interval (msec)</td>
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<td>160/min</td>
<td>130/min</td>
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<tr>
<td>Wenckebach Point (beats per min)</td>
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<td>395</td>
<td>75 (not retested)</td>
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<tr>
<td>AV Node Effective Refractory Period (msec)</td>
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<td>55</td>
<td>241±21</td>
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<td>Ventricular Effective Refractory Period (msec)</td>
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<td>300</td>
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