Normal Coronary Arteriogram in a Patient with Clinical Evidence of Myocardial Infarction*

Nicholas P. DePasquale, M.D., and Michael S. Bruno, M.D.

The case of a 61-year-old man in whom coronary arteriography was normal eight weeks after unmistakable clinical evidence of acute myocardial infarction is discussed to illustrate problems occasioned by the introduction of coronary arteriography. There is a growing tendency to consider the coronary arteriogram as the sine qua non for diagnosis of obstructive artery disease while disregarding clinicopathologic observations gathered and refined over many years. A normal coronary arteriogram in the presence of clinical evidence of coronary heart disease almost always elicits an explanation for the clinical findings other than the possibility that a good quality coronary arteriogram may fail to demonstrate significant coronary artery disease. The coronary arteriogram must be correlated with pathologic findings in many more patients before it can be accepted as the ultimate criterion upon which, not only diagnosis of coronary artery disease depends, but also evaluation of work capacity, disability compensation and other socioeconomic problems associated with coronary artery disease.

The value of coronary arteriography in the study of patients with known or suspected coronary heart disease is well established. Nevertheless, the increasing number of reports of normal coronary arteriograms in patients with strong clinical evidence of coronary heart disease is of growing concern to the clinician.1-9

This report describes a patient with unmistakable clinical evidence of acute myocardial infarction in whom coronary arteriography failed to demonstrate obstructive coronary artery disease and discusses some clinical implications of these findings.

**CASE REPORT**

The patient, a 61-year-old laborer, was well until December 1970 when he experienced retrosternal distress for the first time. An electrocardiogram registered a few days later displayed T-wave changes suggestive of myocardial ischemia, but no therapy was recommended (Fig 1). He was examined for the first time about four months later (April 22, 1971) following a two-hour episode of severe retrosternal pain. The arterial blood pressure was 120/80 mm Hg and the radial pulse was 92 beats per minute and regular. Moist rales

*From the Cardiovascular Service, Lenox Hill Hospital, New York City.

Reprint requests: Dr. De Pasquale, 100 East 77th Street, New York, New York 10021

<table>
<thead>
<tr>
<th>Date</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>12-5-69</td>
<td></td>
</tr>
<tr>
<td>12-4-70</td>
<td></td>
</tr>
<tr>
<td>4-22-71</td>
<td></td>
</tr>
<tr>
<td>4-23-71</td>
<td></td>
</tr>
<tr>
<td>10-7-71</td>
<td></td>
</tr>
</tbody>
</table>

**FIGURE 1.** Serial electrocardiograms showing development of complete left bundle branch block between April 22, 1971 and April 23, 1971.
were heard at both lung bases, but the neck veins were collapsed at 45° trunk elevation. The first heart sound was diminished in intensity. An atrial gallop sound and a soft, blowing, systolic murmur were audible at the mitral area. The liver was not enlarged and there was no peripheral edema. An electrocardiogram displayed increased abnormality of the ST segments and T waves as compared with the ECG registered on December 4, 1970 (Fig 1). He was admitted to the hospital, and the following day the electrocardiogram displayed complete left bundle branch block (Fig 1). Significant elevations in the serum concentrations of creatinine phosphokinase (CPK) and glutamic oxaloacetic acid transaminase (GOT) occurred between the fourth and tenth hospital day (Fig 2). Except for a brief period of Mobitz type I A-V block on the second day, the clinical course was uncomplicated, and he was discharged on the 23rd day.

Eight weeks after discharge a coronary arteriogram was performed, which failed to demonstrate obstructive coronary artery disease. A left ventriculogram showed minimal mitral regurgitation but no left ventricular dyskinesia. An electrocardiogram registered on October 7, 1971, 24 weeks after acute myocardial infarction, displayed persistence of left bundle branch block (Fig 1).

DISCUSSION

Because this patient performed heavy labor out-of-doors in all weather he was advised to seek less physical-demanding work. Although he had the same employer for more than 25 years, he could not be placed in a less strenuous job indoors. Subsequently he applied for disability. In spite of strong clinical evidence for myocardial infarction the medical director of a large insurance company refused to award disability compensation until a coronary arteriogram was performed. While the advisability of coronary arteriography was not debated, the implication that the diagnosis of coronary heart disease and determination of disability depended solely on the coronary arteriogram is disturbing. There appears to be a growing tendency to consider the arteriogram not only as indispensable in the diagnosis of coronary heart disease, but also as quantitatively accurate. However, the increasing number of reports describing angiographically normal coronary arteries in patients with unmistakable clinical evidence of coronary heart disease suggests that the coronary arteriogram is not the sine qua non for the diagnosis of coronary heart disease, nor is it a substitute for clinical judgment. Therapy as well as the determination of work capacity in the cardiac patient should be based upon all of the clinical findings, not upon a single diagnostic procedure, which although only recently introduced, is already generating a growing volume of disparate literature.

REFERENCES


