Thallium-201 Myocardial Perfusion Imaging in Infants and Children

Value in Distinguishing Anomalous Left Coronary Artery from Congestive Cardiomyopathy

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SUMMARY In infants and children, anomalous origin of the left coronary artery (ALCA) from the pulmonary artery may be difficult to distinguish from congestive cardiomyopathy (CCM) of other causes. We performed thallium-201 myocardial perfusion imaging in seven children with ALCA and in nine with CCM to study the usefulness of this technique in distinguishing between these lesions. Localized abnormalities of thallium uptake were present in each of the seven patients with ALCA, including two asymptomatic 4-year-old children. Thallium distribution was normal in five patients with CCM, diffusely irregular in three, and was absent in the lateral and posterobasal portions of the left ventricle in one patient. We conclude that thallium-201 imaging is a sensitive noninvasive method of detecting ALCA. However, perfusion abnormalities are not limited to patients with coronary artery abnormalities, and may be present in patients with myocardial ischemia or infarction of other causes.

INFANTS with anomalous origin of the left coronary artery (ALCA) from the pulmonary artery may be difficult to differentiate from those with congestive cardiomyopathy (CCM) of other causes. Both conditions may cause congestive heart failure and cardiomegaly, either with no cardiac murmur or the murmur of mitral insufficiency. The ECG frequently reveals evidence of myocardial infarction in patients with ALCA, but this is not a universal finding. Furthermore, patients with CCM may have electrocardiographic patterns that suggest infarction. Although the two conditions may be distinguished by aortography, this procedure is not without risk, especially in severely ill infants.

Thallium-201 myocardial perfusion imaging has been used to demonstrate areas of myocardial infarction and ischemia in adults with coronary artery disease. The present study was undertaken to determine the usefulness and limitations of thallium-201 myocardial imaging in differentiating infants and children with ALCA from those with CCM.

Methods

Patients

Thallium-201 myocardial perfusion scans were performed in 16 infants and children: seven had ALCA and nine had idiopathic CCM. In each patient the diagnosis was established by cardiac catheterization and angiography. The patients with ALCA were between 2 months and 4 years of age. Five were younger than 1 year of age and had signs and symptoms of congestive heart failure. The two older patients, each 4 years of age, were asymptomatic at the time of study but had experienced congestive heart failure in infancy. Two patients had mild mitral insufficiency.

The ECG showed the pattern of anterolateral infarction in four patients, left ventricular hypertrophy in two and left bundle branch block in one (table 1). The nine patients with CCM were between 1 month and 4 years of age. Each had evidence of congestive heart failure at the time of study and four had mitral insufficiency. The ECG revealed left ventricular hypertrophy in six, left bundle branch block in one, lateral myocardial infarction in one and isolated T-wave inversion (V₅ to V₇) in one.

Myocardial Imaging

Thallium-201 myocardial perfusion scintigrams were obtained in the anterior, 45° left anterior oblique (with a 15° cephalad tilt) and left lateral views. Imaging was performed with a mobile scintillation camera (Searle LEM) equipped with a low-energy converging collimator with a field of view of 25.5 cm at the collimator surface and 19.5 cm at 10 cm from the surface. Pediatric 201TI-thallous chloride dosages were calculated from a body weight nomogram based on a 1.5-mCi dose for a 70-kg adult. However, a minimum dose of 700 Ci was used to provide a statistically adequate number of counts in a practical imaging time. This dose of radiation is similar to that used in other diagnostic nuclear medicine procedures, and is considerably less than that used during cardiac catheterization and angiography.

Both raw and computer-processed images were interpreted without knowledge of the patient's history or angiographic data. Computer processing was ac-
Table 1. 

<table>
<thead>
<tr>
<th>Age</th>
<th>ECG</th>
<th>Thallium-201 scan (location of perfusion defect)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anomalous left coronary artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 mo</td>
<td>Anterolateral infarction</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>4 mo</td>
<td>Anterolateral infarction</td>
<td>Anterolateral, septum</td>
</tr>
<tr>
<td>5 mo</td>
<td>LBBB</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>6 mo</td>
<td>Anterolateral infarction</td>
<td>Anterolateral, apex</td>
</tr>
<tr>
<td>9 mo</td>
<td>Anterolateral infarction</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>4 yr</td>
<td>LVH</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>4 yr</td>
<td>LVH</td>
<td>Anterolateral</td>
</tr>
<tr>
<td>Congestive cardiomyopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 mo</td>
<td>Lateral infarction</td>
<td>Lateral, posterobasal</td>
</tr>
<tr>
<td>4 mo</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>10 mo</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>10 mo</td>
<td>LVH</td>
<td>Diffuse</td>
</tr>
<tr>
<td>16 mo</td>
<td>LVH</td>
<td>Diffuse, septum</td>
</tr>
<tr>
<td>16 mo</td>
<td>T-wave inversion (V₄ to V₇)</td>
<td>Diffuse</td>
</tr>
<tr>
<td>2 yr</td>
<td>LVH</td>
<td></td>
</tr>
<tr>
<td>4 yr</td>
<td>LVH</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: LBBB = left bundle branch block; LVH = left ventricular hypertrophy.

Complished by means of a PDP-11 computer, with a maximum background subtraction of 20%. Processed images were displayed in both black and white and seven-color formats. In each patient, the tracer distribution was judged to be either normal, diffusely patchy or irregular, or diminished in a segmental or localized distribution.

Results

Anomalous Left Coronary Artery

Localized myocardial perfusion abnormalities were present in each of the seven patients with ALCA (table 1). These abnormalities involved the anterolateral left ventricular wall in all patients (fig. 1), with additional abnormalities of the apex and septum in two patients.

Congestive Cardiomyopathy

Thallium distribution was uniform throughout the myocardium in five of the nine patients with CCM (fig. 2). In three patients, tracer concentration was diffusely irregular and uptake was further diminished in the septum of one of these patients (fig. 3). In one patient, a 1-month-old infant with severe congestive heart failure and an electrocardiographic pattern of lateral wall myocardial infarction, the thallium scan showed a perfusion defect involving the lateral and posterobasal portions of the left ventricle. Cardiac catheterization and angiography showed normal origin of the coronary arteries and diffuse myocardial hypokinesis.

Discussion

Infants with ALCA are asymptomatic in the newborn period, but most develop angina, congestive heart failure and anterolateral myocardial infarction as pulmonary artery pressure falls in the first or second month of life. Approximately 80% of infants with this anomaly die in the first year of life.14

Figure 1. Thallium myocardial perfusion scan in a 9-month-old infant with anomalous origin of the left coronary artery from the pulmonary artery. There is a localized area of diminished tracer concentration in the anterolateral wall, best seen in the anterior projection. ANT = anterior; LAO = left anterior oblique; LLAT = left lateral.
Because the myocardial distribution of thallium closely parallels regional myocardial perfusion, it seemed likely that thallium imaging would reveal abnormalities in patients with ALCA. The results of this study confirm this impression, and indicate that this technique is helpful in distinguishing ALCA from other causes of CCM in infancy. Each of our patients with ALCA, including the two asymptomatic 4-year-olds, had localized areas of diminished thallium uptake in the anterolateral wall of the left ventricle.

Perfusion abnormalities were also present in four of the nine patients with CCM. In three of these patients, the defects were small, diffuse and easily distinguished from those present in patients with ALCA. In the patient with ECG evidence of myocardial infarction, the thallium scan showed decreased tracer uptake in the lateral and posterobasal portions of the left ventricle. This patient, who had clinical and laboratory evidence of systemic viral infection and myocarditis, developed pulmonary edema and shock at 1 week of age. Myocardial infarction may have been due to hypotension and hypoxemia or, as described by Woods et al., to viral infection per se.

Previous investigators have used thallium-201 scans in evaluating infants and children with ALCA. Ferrer et al. reported perfusion abnormalities in three infants with ALCA and normal thallium distribution in three children with CCM. Finley et al. found anterolateral perfusion defects in three infants with ALCA; follow-up studies in two patients revealed reduction in the size of the defects, coincident with clinical improvement on anticongestive therapy. Scans in three patients with ALCA who had undergone operation were normal. Rabinovitch et al. showed an anterior area of absent tracer uptake in a 4-month-old infant with ALCA, but found no perfusion abnormality in an asymptomatic 13-year-old child with ALCA.

The results of the present study are similar to those of Bulkley et al., who used radionuclide imaging to study adults with ischemic and idiopathic cardiomyopathy. Patients with ischemic cardiomyopathy had larger perfusion defects by thallium scintigraphy, and had regional wall motion abnormalities on gated cardiac blood-pool scans. As in our study, one patient with normal coronary arteries had a large perfusion abnormality indistinguishable from that produced by coronary artery disease.

It appears, therefore, that thallium-201 imaging is quite sensitive but only moderately specific in the diagnosis of ALCA. With the exception of the asymptomatic adolescent reported by Rabinovitch et al., scans have consistently revealed perfusion abnormalities in children with ALCA. Although patients with primary myocardial disease generally have normal or diffusely irregular thallium uptake, areas of infarction or fibrosis may result in perfusion defects that can mimic those present in patients with ALCA. Likewise, although ALCA is the most common con-

FIGURE 2. Thallium scan in a 16-month-old girl with idiopathic congestive cardiomyopathy. Tracer distribution is homogeneous throughout the myocardium of the dilated left ventricle. ANT = anterior; LAO = left anterior oblique; LLAT = left lateral.

FIGURE 3. This thallium scan, from a 10-month-old girl with congestive cardiomyopathy, shows diffusely irregular tracer concentration, without discrete or segmental perfusion defects. Left ventricular dilation is also apparent. ANT = anterior; LAO = left anterior oblique; LLAT = left lateral.
genital coronary artery abnormality, lesions such as atresia of a coronary ostium, coronary artery fistula or premature coronary atherosclerosis might produce myocardial perfusion defects.

The present study reaffirms the value of the standard ECG as a screening test for the presence of coronary artery anomalies in infants. The ECG showed myocardial infarction in four of our seven patients with ALCA. The two patients in whom the ECG showed left ventricular hypertrophy were older (each was 4 years old) and may have had a pattern of infarction in infancy. If the ECG reveals myocardial infarction, angiography should be performed, and a thallium perfusion scan is not necessary to make the diagnosis. Subsequent studies are required to determine if the extent of the nonperfused area on the thallium scan can be used to determine prognosis or to select patients for operation. At present, thallium imaging is most likely to be useful in the patient with suspected ALCA and an atypical ECG, i.e., one showing left bundle branch block or left ventricular hypertrophy.

Early differentiation of patients with ALCA from those with CCM is important, because the former condition is potentially amenable to surgical therapy. Operative techniques have included ligation of the left coronary artery at its origin from the pulmonary artery, 4–16 saphenous vein grafting, 17–20 anastomosis of the left subclavian artery to the anomalous coronary artery, 21–23 and reimplantation of the origin of the left coronary artery into the aortic wall. 24 Although operative mortality is high in infants younger than 1 year of age, this could be potentially improved by earlier recognition of the anomaly and improvement in microvascular surgical techniques.

References

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