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Confirmation of Anomalous Origin of the Right Coronary Artery From the Left Sinus of Valsalva With Magnetic Resonance Imaging*

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Anomalous origin of the right coronary artery from the left sinus of Valsalva is a rare but clinically significant congenital abnormality, difficult to diagnose angiographically. We describe a patient in whom magnetic resonance imaging was used to delineate the anomalous course of the right coronary artery following angiographic demonstration limited by technical considerations. (Chest 1993; 104:1284-86)

RCA = right coronary artery

Anomalous origins of coronary arteries are found in 0.3 percent to 1.0 percent of patients undergoing coronary catheterization. They include origin of the circumflex from the right sinus of Valsalva with a 0.45 percent incidence, origin of the left anterior descending from the right sinus of Valsalva with a 0.2 percent incidence, and origin of the left main from the pulmonary artery with a 0.04 percent incidence. One of the least frequent anomalies is the right coronary artery (RCA) arising from the left sinus of Valsalva with a reported incidence of 0.02 percent to 0.16 percent. Although this anomaly is rare, its recognition is clinically important, especially its course, since it is associated with angina pectoris, myocardial infarction, syncope, and sudden death. We describe a patient in whom magnetic resonance imaging (MRI) was used to delineate the anomalous course of the RCA following angiographic demonstration that was limited by technical considerations.

CASE REPORT

A 54-year-old black man was admitted to the surgical service for excision of a right axillary hibernoma. Cardiologists physicians were consulted following surgery because the patient experienced chest pain. The patient admitted to having pain for the previous 4 to 5 months. The pain was dull, oppressive, retrosternal, and often radiated to the left arm. It was brought on with exertion and relieved with rest. It was associated with shortness of breath and diaphoresis. During the preceding four months, the patient had also experienced six bouts of post-exertional syncope. His medical history was significant for hypertension for five years.

Physical examination revealed blood pressure of 150/100 mm Hg with no orthostatic changes. An ECG showed early repolarization changes in lateral leads. Echocardiogram showed normal left and right ventricular size, thickness, and function. Exercise stress test was terminated 3 min and 49 s into exercise when the patient experienced typical chest pain at peak exercise. The pain resolved 2 min after cessation of exercise. The patient achieved only 69 percent of his maximum predicted heart rate. No diagnostic ECG changes were noted.

Cardiac catheterization was performed that showed normal hemodynamics, normal left ventricular systolic function, and 1+ mitral regurgitation. The coronary angiogram showed the following:

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significant cardiac morbidity and mortality are the left main coronary artery originating from the right sinus of Valsalva, and the RCA arising from the left sinus of Valsalva, coursing between the aorta and pulmonary artery. The high risk of these anomalies may be related to the slit-like opening of the vessels caused by their acute take-off angle from the aortic root that would tend to narrow with aortic dilatation induced by exercise or to the compression of the coronary artery between the great vessels.

Sudden death is not an uncommon presentation for this anomaly, occurring in up to 30 percent of patients according to one report. Therefore, in patients suspected of having this anomaly, every attempt must be made for its diagnosis and surgical correction. Angiographic demonstration of this anomaly is often difficult as exemplified by our case and also reported by others. Currently, there does not seem to be a consensus on an ideal noninvasive technique to diagnose or confirm suspected cases. Several cases have been described in which transesophageal echocardiography supplemented the information obtained from cardiac catheterization. Magnetic resonance imaging has been used to evaluate patency of bypass grafts and to evaluate various coronary artery anomalies such as aneurysm and fistulas. We believe ours is the first case in which MRI has been used to document an anomalous course of the right coronary artery.

Magnetic resonance imaging appears to be a promising tool in confirming the origin and course of anomalous coronary arteries in difficult cases.

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Bronchiectasis in a Child After Acrolein Inhalation

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Acrolein is an unsaturated aldehyde produced by combustion of many organic compounds. Massive exposure may lead to severe pulmonary disease and possibly death. We report a case of intoxication in a 2-year-old boy; an 18-month follow-up showed development of obstructive bronchiolar disease with diffuse bronchiectasis.

(Chest 1993; 104:1256-57)

Acrolein is produced during the combustion of a variety of organic substances: house fires, cigarette smoking, burning fatty acid.\(^1\) Combustion of the lipids present in vegetable oil also produces acrolein. Dangerous inhalations usually are avoided because acrolein is a very powerful irritant. In contrast, very young children may be exposed to acrolein for a long time, and domestic accidents represent a potential but rare case of intoxication. We report a case of a severe acrolein intoxication in a young boy who was confined in an area where acrolein was present due to the lack of parental care.

**CASE REPORT**

A previously healthy 27-month-old boy was admitted to the district hospital because of acute respiratory failure, after being confined for about 1 h in a kitchen where vegetable oil was burning on an electric hot plate and producing acrid smoke. The child was cyanotic, acutely dyspneic, and his respiratory rate was 58 breaths per minute; auscultation of the thorax revealed crackles. Intoxication by acrolein was suspected because of the nature of the smoke; the cooker was electric so carbon monoxide inhalation was unlikely. Arterial blood gas value analysis showed a pH level of 7.08, a PaO\(_2\) value of 47 mm Hg, and a PaCO\(_2\) level of 81 mm Hg. The chest x-ray film showed an alveolar consolidation with bull's wing distribution. The initial treatment consisted of oxygen therapy and administration of duretics and antibiotics. Respiratory distress improved during the first hours, but hypoxemia persisted. No acetylcysteine treatment was given.

Four weeks later, the child was transferred to our department. He still had tachypnea, had a productive cough, and needed continuous oxygen therapy (3 L/min). Auscultation of the thorax revealed crackles and wheezing. A chest x-ray film showed notable regression of pulmonary opacities. Fiberoptic examination showed moderate but diffuse tracheobronchial inflammation. The results of functional follow-up are summarized in Table 1.

Three months after the exposure to acrolein, the productive cough was unchanged. There were moderate signs of bronchitis and general overinflation evidenced on the chest x-ray film. Computed tomography (CT) showed focal overinflation with decreased perfusion without any dominant localization (Fig 1). Bronchiectasis was not detected.

Nine months after exposure, daily chest physiotherapy was still necessary, but oxygen was required only during the night. The chest x-ray film was unchanged and CT showed heterogeneous areas of overinflation with more marked focal emphysema. Some bronchiectasis and more diffuse thickened bronchial walls appeared in the lingula, middle lobe, and lower lobes.

Eighteen months after the exposure, overnight transcutaneous saturation allowed the cessation of oxygen therapy. The clinical course was marked by occasional infections and permanent productive cough. Boentgenogram analysis showed bronchial thickening and massive overinflation. The CT scan showed patchy areas of emphysema, localized atelectasis, thickened bronchial walls, and diffuse bronchiectasis (Fig 2).

**Table 1—Pulmonary Function Follow-Up**

<table>
<thead>
<tr>
<th>Pulmonary Function Data</th>
<th>4 Weeks</th>
<th>3 Months</th>
<th>9 Months</th>
<th>18 Months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory rate (%)</td>
<td>88</td>
<td>93</td>
<td>92%</td>
<td>93%</td>
</tr>
<tr>
<td>Oxygen saturation (%)</td>
<td>67</td>
<td>80</td>
<td>88</td>
<td>93%</td>
</tr>
<tr>
<td>Transfer factor (%)</td>
<td>65%</td>
<td>.</td>
<td>.</td>
<td>92%</td>
</tr>
<tr>
<td>Functional residual capacity (%)</td>
<td>79%</td>
<td>.</td>
<td>.</td>
<td>66%</td>
</tr>
<tr>
<td>Functional residual capacity, % of normal value</td>
<td>112</td>
<td>91</td>
<td>.</td>
<td>150</td>
</tr>
<tr>
<td>Specific airway resistance, % of normal value</td>
<td>568</td>
<td>568</td>
<td>.</td>
<td>385</td>
</tr>
</tbody>
</table>

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