INTRODUCTION
Sinus of Valsalva aneurysm (SVA) is a very infrequent cardiac anomaly (0.14-0.96% of cases of open-heart surgery). Although the cause may be acquired, most cases are congenital anomalies arising from a defect of the aortic media. SVA usually remains silent until it ruptures, although it sometimes produces different clinical manifestations, such as obstruction of the right ventricular outflow tract, aortic regurgitation, rhythm disorders and, more rarely, myocardial ischemia or necrosis. We present a case of congenital right SVA that presented as inferior acute myocardial infarction (AMI).

CLINICAL CASE
The patient was a 40-year-old white male who smoked, but had no other coronary risk factors. In childhood he had been advised of the presence of a systolic murmur, but until the day of admission it had been asymptomatic and had not been studied. The patient came to the hospital with prolonged chest pain that had appeared suddenly while at rest. In the emergency room he was diagnosed as inferior AMI with clinical and electrocardiographic criteria, which later was confirmed by the demonstration of enzyme elevation (CPK 3495, MB 362). The clinical evolution was uncomplicated and 7 days after admission the patient was transferred to our service for complete study. In the physical examination his blood pressure was 100/60 mm Hg and he had a precordial systolic ejection murmur, grade 3/6. The ECG showed sinus rhythm, 65 beats/min, with a QS pattern and negative T waves in leads III and aVF. The chest X-ray exam was normal. Transthoracic echocardiography showed a tricuspid aortic valve and a partially thrombosed anterior sinus of Valsalva aneurysm. The left ventricle was dilated (end-diastolic diameter...
66 mm), with mild inferior hypokinesia and an ejection fraction of 52%. Transesophageal echocardiography confirmed the presence of an aneurysm (57 × 55 mm in diameter) that was partially filled with thrombotic material and protruded into the right chamber (Figure 1). No intracardiac shunting or aortic regurgitation was detected. Coronary angiography (Figure 2) showed a partly thrombosed right SVA (55 × 35 mm diameter in the left anterior oblique view and 41 × 41 mm in the right anterior oblique view); the coronary arteries had no atherosclerotic lesions. No possible causes of acquired SVA were detected.

The patient underwent surgery with extracorporeal circulation. The right sinus of Valsalva presented an aneurysmal dilatation (4 × 3 cm) extending forward and partially thrombosed, which affected the ostium of the right coronary artery. Surgical correction included closure of the right coronary ostium with two interrupted stitches, reconstruction of the ascending aorta with a Dacron patch and implantation of a saphenous vein graft bypass from the aorta to the right coronary artery. The postoperative course was uneventful and the patient is alive and asymptomatic after 2 years of follow-up.

**DISCUSSION**

SVA can be acquired, secondary to infectious processes, degenerative, or traumatic. In these cases there is diffuse dilatation of the sinus of Valsalva, which can affect any single sinus, two or three sinuses at the same time, the ascending aorta, and even project into the pericardium. However, most cases are congenital SVA due to a defect in the aortic wall behind the sinus of Valsalva, consisting of a lack of continuity between the aortic media and valve annulus. In these cases, the right coronary sinus is most frequently affected (70% of cases) and the aneurysm usually forms digitiform protrusions into the atrium and/or right ventricle. The non-coronary sinus is affected in 29% of cases, and involvement of the left coronary sinus is exceptional. Congenital SVA may be associated with septal or valve defects.

The most frequent complication of SVA is rupture, opening into the right atrium or ventricle or, as occurs in some acquired cases, the pleural or pericardial cavity. The diagnosis of ruptured SVA is relatively easy to establish because of the severe clinical picture that accompanies the condition. However, diagnosing a SVA before rupture is much less likely because it is usually silent. An unruptured SVA shows no clinical manifestations when it becomes infected, produces embolism, or compresses neighboring structures during its expansion. The most frequent complications are obstruction of the right ventricular outflow tract, aortic regurgitation, conduction disorders, and, more rarely, myocardial ischemia due to compression of the coronary arteries (Table 1).

The reduction of coronary flow due to SVA is an uncommon manifestation. In these cases, affectation of the coronary circulation can be potentially lethal because aneurysmal expansion may be very rapid, so emergency surgery must be considered. There is more risk of myocardial ischemia with left SVA than with right SVA. The reason for this may be that the central part of the left atrial coronary sinus is directly exposed to the pericardium and aneurysms originating here can protrude between the left atrium and pulmonary trunk, compressing the trunk and branches of the left coronary artery. In contrast, obstruction of the ostium of the right coronary artery is usually due to the presence of a thrombus or syphilitic involvement, and
less frequently to mechanical deformation of the os-
tium and/or proximal compression.8

In our case, the SVA was probably congenital (pos-
sible acquired causes were excluded) and the first cli-
nical manifestation was inferior AMI. In the interna-
tional literature, only 44 cases of SV A accompanied
by myocardial ischemia or necrosis have been repor-
ted. Of them, 28 involved the left sinus of V alsalva, 12
the right sinus, and 4 both. AMI was the first clinical
manifestation in 14 of the 28 cases of left SV A, but
only in 3 of the 12 right SV A (Table 1). Our patient,
consequently, is the fourth case in which AMI was the
first manifestation of an unruptured right SV A and
the first described in the Spanish literature.

Myocardial ischemia secondary to SV A can be a
sign of poor prognosis. As indicated by Faillace et al.,7
aneurysmal dilatation may occur very rapidly, and
eyarly diagnosis and surgical treatment is mandatory.
The surgical technique depends on the anatomical
constraints and ranges from a simple patch closing the
aneurysmal orifice9 to complete reconstruction of the
aortic root. The prognosis seems to improve if aortic
valve replacement and aortocoronary bypass can be
avoided.10 After surgical repair, the prognosis is
usually good and the risk of recurrence is rare.

REFERENCES
1. Takach TJ, Reul GJ, Duncan M. Sinus of Valsalva Aneurysms or
1.573-1.577.
2. Sakakibara S, Konno S. Congenital aneurysm of the sinus the
Valsalva. Anatomy and classification. Am Heart J 1962; 63:
405-424.
3. Jugdutt BJ, Fraser RS, Rossall RE, Lee SJ. Ruptured aneurysms
4. Brandt J, Jogi P, Luhrs C. Sinus of Valsalva aneurysm obstruc-
tion coronary arterial flow: case report and collective review of
5. Fishbein MC, Obma R, Roberts WC. Unruptured sinus of
6. Caballero J, Arana R, Calle G. Aneurisma congénito del seno de
Valsalva roto a ventrículo derecho, comunicación interventricular

TABLE 1. Cases of acute myocardial infarction as a manifestation of aneurysm of the sinus of Valsalva

<table>
<thead>
<tr>
<th>Author and bibliographic reference</th>
<th>Sinus involved</th>
<th>Mechanism</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chipps, 1941</td>
<td>Left</td>
<td>Compression of LCT, DA and CX</td>
<td>Rupture</td>
</tr>
<tr>
<td>Traffelet, 1957</td>
<td>Right</td>
<td>Occlusion of proximal RC</td>
<td></td>
</tr>
<tr>
<td>Eliot, 1963</td>
<td>Left</td>
<td>Compression of LCT, DA and CX</td>
<td>Bicuspid AoV</td>
</tr>
<tr>
<td>Olsen, 1969</td>
<td>Left</td>
<td>Compression of DA</td>
<td>Calcified AoS. Pneumonia.</td>
</tr>
<tr>
<td>Bulkley, 1975</td>
<td>Right</td>
<td>Occlusive thrombus in the RC ostium</td>
<td>Aor. Sudden death</td>
</tr>
<tr>
<td>Cupo, 1981</td>
<td>Left</td>
<td>Unknown</td>
<td>NCS also aneurysmal</td>
</tr>
<tr>
<td>Hiyamuta, 1983</td>
<td>Left</td>
<td>Compression of LCT and DA</td>
<td>Aor.</td>
</tr>
<tr>
<td>Brandt, 1985</td>
<td>Left</td>
<td>Compression of LCT, DA and CX</td>
<td>Previous AoVR</td>
</tr>
<tr>
<td>Ishide, 1985</td>
<td>Right and non-coronary</td>
<td>Compression of RC</td>
<td>Moderate Aor. Weber-Christian disease</td>
</tr>
<tr>
<td>Faillace, 1985</td>
<td>Left</td>
<td>Compression of LCT</td>
<td>Aor. Rupture into LV</td>
</tr>
<tr>
<td>Ince, 1986</td>
<td>Left</td>
<td>Compression of LCT</td>
<td></td>
</tr>
<tr>
<td>Nakano, 1986</td>
<td>Left</td>
<td>Compression of LCT and CX</td>
<td></td>
</tr>
<tr>
<td>Koike, 1991</td>
<td>Left</td>
<td>Partial occlusion of the left coronary</td>
<td>Myotic aneurysm</td>
</tr>
<tr>
<td>Tahir, 1995</td>
<td>Left</td>
<td>Occlusion DA (anterior LV aneurysm)</td>
<td>Clinical manifestations of angina</td>
</tr>
<tr>
<td>Tall, 1996</td>
<td>Left</td>
<td>Compression of LCT, DA and CX</td>
<td>ALE</td>
</tr>
<tr>
<td>Ferreira, 1996</td>
<td>Left</td>
<td>Compression of CX. Associated vasospasm?</td>
<td>During stress test with dobutamine</td>
</tr>
<tr>
<td>Wiemer, 1996</td>
<td>Left</td>
<td>Compression of DA and CX</td>
<td>Congenital AoS. IE</td>
</tr>
</tbody>
</table>

ALE indicates acute lung edema; Aor, aortic regurgitation; AoS, aortic stenosis; AoV, aortic valve; AoVR, aortic valve replacement; CX, circumflex artery; DA, left descending anterior artery; IE, infectious endocarditis; LA, left atrium; LCT, left coronary trunk; LV, left ventricle; MI, mitral regurgitation; MS, mitral stenosis; NCS, non-coronary sinus; RC, right coronary artery; RV, right ventricle.