

Sinus of Valsalva Aneurysm as a Cause of Acute Myocardial Infarction

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Aneurysmal dilatation of one or more of the sinuses of Valsalva (SVA) is a rare cause of coronary insufficiency. We describe one case of unruptured and partially thrombosed right sinus of Valsalva aneurysm of which the first sign was acute inferior myocardial infarction in a 40-year-old man while reviewing the literature, we found 44 reported cases of sinus of Valsalva aneurysm, complicated by myocardial ischemia or infarction. In 28 cases the left coronary sinus was involved, in 12 cases the right one, and in 4 cases both of them. Myocardial ischemia is a potentially ominous prognostic sign in SVA patients. The poor outcome with conservative treatment leads us to consider the patient for emergency surgical therapy.

Key words: *Aneurysm. Valsalva. Ischemia. Myocardial infarction.*

Aneurisma del seno de Valsalva como causa de un infarto agudo de miocardio

La dilatación aneurismática de uno o más de los senos de Valsalva (ASV) es una causa rara de insuficiencia coronaria. Describimos un caso de aneurisma de seno de Valsalva derecho, parcialmente trombosado y no roto, en un varón de 40 años, cuya primera manifestación fue un infarto agudo de miocardio inferior. En una revisión de la bibliografía hemos encontrado 44 casos descritos de ASV como causa de isquemia o infarto de miocardio: en 28 casos el seno de Valsalva implicado era el izquierdo, en 12 casos el derecho y en 4 casos ambos. La aparición de isquemia miocárdica en pacientes con ASV es un signo de mal pronóstico. La mala evolución con tratamiento conservador lleva a considerar la intervención quirúrgica urgente.

Palabras clave: *Aneurisma. Valsalva. Isquemia. Infarto de miocardio.*

INTRODUCTION

Sinus of Valsalva aneurysm (SVA) is a very infrequent cardiac anomaly (0.14-0.96% of cases of open-heart surgery).¹ Although the cause may be acquired, most cases are congenital anomalies arising from a defect of the aortic media.² SVA usually remains silent until it ruptures,³ 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).

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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



Fig. 1. Transesophageal echocardiogram (longitudinal plane at the aortic root) showing aneurysmal dilatation of the anterior sinus of Valsalva.

LA indicates left atrium; Ao, aortic root; LVOT, left ventricular outflow tract.

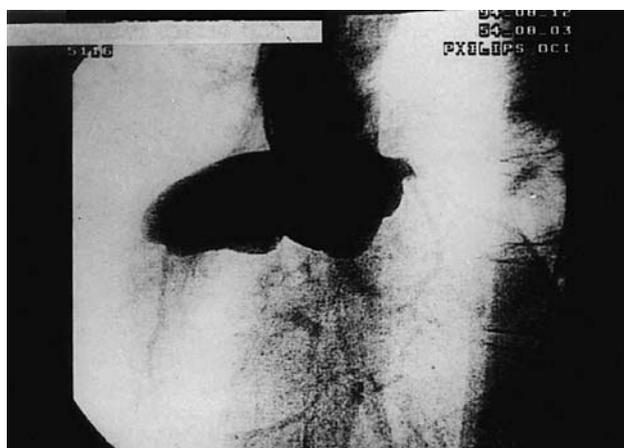


Fig. 2. Aortography: left anterior oblique view showing a saccular dilatation located at the aortic root in the right sinus of Valsalva (55 × 35 mm in diameter). The aneurysm forms a digitiform protrusion into the right atrium. The lower intensity of the contrast in the aneurysmal lumen is due to the presence of a thrombus. On the left side of the figure, the lesion-free right coronary artery is visible. Selective catheterization was not possible.

66 mm), with mild inferior hypokinesia and an ejection fraction of 52%. Transesophageal echocardiography confirmed the presence of an aneurysm (57 × 55 mm) 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.² 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.^{4,5} Congenital SVA may be associated with septal or valve defects.⁶

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.³ 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, affection of the coronary circulation can be potentially lethal because aneurysmal expansion may be very rapid, so emergency surgery must be considered.⁷ There is more risk of myocardial ischemia with left SVA than with right SVA.^{4,8} 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.² In contrast, obstruction of the ostium of the right coronary artery is usually due to the presence of a thrombus or syphilitic involvement, and

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

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

ALE indicates acute lung edema; AoI, 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.

less frequently to mechanical deformation of the ostium and/or proximal compression.⁸

In our case, the SVA was probably congenital (possible acquired causes were excluded) and the first clinical manifestation was inferior AMI. In the international literature, only 44 cases of SVA accompanied by myocardial ischemia or necrosis have been reported. Of them, 28 involved the left sinus of Valsalva, 12 the right sinus, and 4 both. AMI was the first clinical manifestation in 14 of the 28 cases of left SVA, but only in 3 of the 12 right SVA (Table 1). Our patient, consequently, is the fourth case in which AMI was the first manifestation of an unruptured right SVA and the first described in the Spanish literature.

Myocardial ischemia secondary to SVA can be a sign of poor prognosis. As indicated by Faillace et al,⁷ aneurysmal dilatation may occur very rapidly, and early 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.

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