Aspirin-induced asthma is a distinct clinical syndrome. It starts with vasomotor rhinitis which leads to chronic nasal congestion and later to the formation of nasal polyps. Bronchospasm follows with the development of intrinsic asthma.\(^3\) Rhinitis and asthma often precede the appearance of aspirin or other nonsteroidal anti-inflammatory drug (NSAID) sensitivity which is the hallmark of the syndrome.\(^4,5\)

The diagnosis of the AIA syndrome was made in our patient because of the history of precipitation of acute asthma after ingestion of ibuprofen, a NSAID, and aspirin. Although the precise mechanism of the sensitivity to aspirin in AIA remains unknown, because these patients are also sensitive to all the NSAID, and these drugs also inhibit prostaglandin synthesis, the mechanism most likely involves the effect of these drugs on arachidonic acid metabolism.\(^6,7\)

Arachidonic acid metabolism may also be abnormal in patients with PVA. In 1983, Lewy et al\(^8\) reported that thromboxane B\(_2\) levels were elevated at rest and during ischemia in patients with PVA, whereas they were normal in those with vasocclusive angina. Although Lewy's observations have not been consistently confirmed,\(^9,10\) other data have suggested a role for arachidonic acid metabolism in PVA. High dose aspirin has been shown to increase the number of anginal attacks and to reduce the capacity for exercise and to provoke exercise-induced coronary arterial spasm\(^11\) in patients with PVA. Aspirin does not have a similar effect on coronary hemodynamics or symptoms in patients with typical exertional angina.

In our patient, the diagnosis of coronary artery spasm during the acute asthmatic attack was made by the characteristic ECG findings of ST segment elevation with pain which subsequently normalized. The relatively normal coronary angiogram and the positive stress test in the presence of minimal anatomic disease further supports the probability of coronary spasm.

Of interest in this case is the association of coronary artery spasm with an acute attack of aspirin-induced bronchospasm. Since both events can be precipitated by aspirin, care should be taken in the interpretation of chest pain in patients with aspirin sensitivity and ECG changes of coronary artery spasm should be sought in these patients and especially in those with chest pain during acute asthmatic attacks precipitated by aspirin.

Because arachidonic acid metabolism seems to be abnormal both in AIA and PVA, and since both events occurred simultaneously in our patient after using 600 mg of aspirin, it is possible that both syndromes might have a common pathway and it is possible that coronary artery spasm is a common feature of the AIA syndrome. The confirmation of this possibility will require further studies in patients with this syndrome.

**REFERENCES**


**Congenital Aneurysm of the Left Sinus of Valsalva*\(^\text{a}\)**

**Report of a Patient with 19-Year Survival without Surgery**

Lisa Warsinger Martin, M.D.;\(^1\) Irene Hsu, M.D.;\(^2\)

Harry Schwartz, M.D.;\(^3\) and Alan G. Wasserman, M.D.\(^4\)

An unruptured aneurysm of the left sinus of Valsalva was diagnosed by angiography in a 16-year-old male subject 19 years ago. A recent repeated angiographic study documented no change in the aneurysm over the 19-year interval, and the patient has remained asymptomatic. We are aware of no other long-term follow-up reports of patients with this lesion who have not had surgical correction. This case emphasizes the controversy regarding the need for prophylactic surgical correction of an unruptured aneurysm.

An aneurysm restricted to the left aortic sinus of Valsalva is extremely rare;\(^1,3\) its natural history is not known. As in the case of the more commonly encountered aneurysm of the right sinus of Valsalva, diagnosis of an uncomplicated unruptured aneurysm is rarely made.\(^1,3\)

**CASE REPORT**

The patient is a 35-year-old white man whom we have observed for the past nine years. A systolic murmur was initially heard at 14 months of age. At 16 years of age, the patient was evaluated for aortic stenosis. Left cardiac catheterization revealed no gradient across a bicuspid aortic valve. Right cardiac catheterization was normal. The left ventriculogram disclosed an aneurysm of the left sinus of Valsalva (Fig 1), with the left coronary artery arising from the apex of the

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FIGURE 1. Left ventriculogram showing aneurysm of left sinus of Valsalva. The patient was advised to continue unrestricted activities but to avoid "extreme" physical effort and trauma to the chest and to observe SBE prophylaxis. The patient has remained asymptomatic and enjoys unrestricted activities.

Physical findings during our nine-year follow-up have remained unchanged. The patient is a well-developed man with normal body habitus and normal chest wall configuration. The blood pressure is 110/70 mm Hg in the left arm (blood pressure in right arm has been difficult to obtain since the catheterization in 1966). There is no abnormality of jugular pulse. Carotid upstrokes are slightly delayed bilaterally. A grade 3 systolic murmur is present in both carotid arteries. The cardiac impulse is somewhat sustained. No thrill is present. A systolic ejection click is present. The second heart sound is single, and there is a third heart sound at the apex. A grade 3-4 coarse ejection systolic murmur is heard at the second right interspace and is mid-peaking. It radiates into both carotid arteries. During some examinations over the years, a whiff of a diastolic murmur has been heard at the third left interspace; this murmur was described at the initial evaluation in 1966. The findings from the balance of physical examination are unremarkable.

The electrocardiogram and chest x-ray film have remained normal. The M-mode echocardiogram of March 15, 1976 revealed thickened but pliable aortic leaflets and a probable bicuspid aortic valve.

In March 1985, we restudied our patient, limiting the invasive procedure to right cardiac catheterization and an aortogram. The right cardiac pressures were normal, and no shunts were detected. Aortic root injection demonstrated mildly restricted cusp mobility and trivial aortic insufficiency. The aneurysm of the left sinus of Valsalva (Fig 2) involved the origin of the left main coronary artery. There was an area of opacification in the floor of the aneurysm, possibly representing a calcified thrombus. The remainder of the coronary circulation was normal. Comparing our aortogram with the left ventriculogram of 1966, there appeared to be no change in the size of aneurysm. A two-dimensional echocardiogram was obtained and revealed the aneurysm.

DISCUSSION

The few reported cases of an aneurysm of the left sinus of Valsalva illustrate the rare occurrence of this congenital anomaly. There are a few case reports of rupture, of coronary ischemia, and of myocardial infarction; however, the frequency of formation of an aneurysm and the natural history are unknown. An unruptured aneurysm of the sinus of Valsalva is rarely diagnosed clinically, and as in our patient, diagnosis is at times made fortuitously during angiography for suspected congenital aortic stenosis or ventricular septal defect, with which it can be associated. Therefore, the frequency of unruptured aneurysm may be greater than previously reported.

There are no long-term follow-up reports of cases of untreated aneurysm of the left sinus of Valsalva. Our patient, to the best of our knowledge, appears to be the first such individual in whom a documented surgically uncorrected aneurysm of the left sinus of Valsalva has been present for 19 years. The patient was not advised to undergo surgery, in view of the apparent stability of the aneurysm and because surgical correction would have necessitated replacement of the aortic valve, as well as coronary bypass or reimplantation of the left coronary artery. The opacification in the floor of the aneurysm was believed to represent a calcified thrombus. Thrombi in aneurysms of the right sinus of Valsalva have been noted, but there are no reports to our knowledge, of a thrombus in an aneurysm of the left sinus of Valsalva, and the

FIGURE 2. Recent aortogram. Note possible thrombus in floor of aneurysm.
need for anticoagulation is unknown. We are continuing to follow the patient with serial two-dimensional echocardiograms in an attempt to detect any enlargement of the aneurysm.

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REFERENCES


Fatal Paroxysmal Supraventricular Tachycardia in an Infant*

G. F. Buja, M.D.; D. Corrado, M.D.; P. A. Pellegrino, M.D.; A. Nava, M.D.; and G. Thieme, M.D.

A 37-day-old infant, without associated congenital anomalies, died following a 48-hour episode of supraventricular tachycardia. The histopathologic findings of nodoventricular and fasciculoventricular Mahaim's fibers in a setting of persistent fetal dispersion were consistent with an anatomic substrate for a reentry circuit at the specialized A-V junction.

Supraventricular tachycardia (SVT), the most common symptomatic arrhythmia in infants and children, is usually well tolerated. Nonetheless, fatal cases have been reported, but clinical profiles have rarely been correlated with the histopathologic background.

We report postmortem findings in a 37-day-old female infant without associated congenital anomalies, who died following a 48-hour episode of SVT.

CASE REPORT

The patient was the product of a full-term uncomplicated pregnancy and delivery; birth weight was 3.0 kg, and Apgar score was 9 at five minutes. Both parents were alive and well, and no family history of heart disease or arrhythmia was recorded. The baby was well until

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FIGURE 1. A (upper): ECG surface recording of paroxysmal, narrow QRS tachycardia with rate of 320 beats per minute. B (lower): Reversion of supraventricular tachycardia to sinus rhythm after a slowing of rate to 210 beats per minute.

the age of 34 days, when she started to refuse food, and her parents also noticed palpitation, restlessness, and hypotonia. Her clinical condition progressively worsened in the following two days, and she was hospitalized; at admission, signs of cardiogenic shock were present. Chest x-ray film revealed 3+ cardiomegaly with pulmonary venous congestion. The ECG tracing (Fig 1, upper) showed a regular SVT of 320 beats per minute, with narrow QRS: neither P nor atrial flutter waves were seen on the surface ECG including right precordial leads. The child was immediately intubated and assisted ventilation was initiated following episodes of apnea. During the intubation maneuvers, the SVT spontaneously and abruptly reverted to a sinus rhythm with a rate of 120 beats per minute, following a very short period of slowing of SVT rate to 210 beats per minute, with no evidence of P or atrial flutter waves (Fig 1, lower).

Soon after intubation, assay of arterial blood gas levels disclosed the following values: pH, 6.88; PO2, 73; PCO2, 31, and BE, -28.7. The patient was then taken to the intensive care unit where she died two days later, never having completely regained consciousness, with irreversible acidosis and anuria despite peritoneal dialysis. Continuous heart monitoring showed persistent sinus rhythm.

At autopsy, severe systemic and pulmonary venous congestion were observed together with features of plurivisceral shock. Extra-cardiac causes of death were excluded. No gross congenital heart anomaly was observed, and histologic examination ruled out any

FIGURE 2. Mahaim's fibers (arrow) join the compact atrioventricular node (AVN) with the crest of the ventricular septum (nodooventricular bypass fibers) (Azan-Mallory, original magnification ×48).