Aortic-Left Atrial Communication

A Diagnostic and Therapeutic Problem

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In recent years, aneurysms of the sinus of Valsalva with communication between the root of the aorta and intracardiac chambers as well as communication between the aorta and pulmonary artery, mediastinum, pericardium, superior vena cava, or pleural cavity have been reported fairly frequently. Only rarely has an aortic-left atrial communication been reported.1-3 The present report concerns a young woman with chronic rheumatic heart disease with severe mitral insufficiency, moderately severe aortic insufficiency, and a communication between the noncoronary sinus of Valsalva and the left atrium. The reasons for reporting this case are: (1) An aortic-left atrial communication is a rare lesion. (2) Sinus of Valsalva aneurysms with fistulous communications have only uncommonly been reported with rheumatic heart disease.2 4 (3) In the presence of aortic and mitral insufficiency, the diagnosis of such a lesion can be extremely difficult. (4) Such a lesion may present special problems for the cardiovascular surgeon at the time of cardiopulmonary bypass.

Report of Case

The patient was a 28-year-old Puerto Rican female who was referred to the hospital because of rheumatic heart disease and progressive symptomatology. She had rheumatic fever at age 18 and did fairly well until 1957 when atrial fibrillation and congestive heart failure developed. In 1958, she had her first child and after this she did poorly with bouts of atrial fibrillation and congestive failure. During the past 1½ years, she had had a progressive downhill course with marked easy fatigability, weakness, severe dyspnea on exertion, and frequent attacks of atrial fibrillation.

Physical examination revealed a chronically ill female in no distress. The pulse was 88 and irregular, blood pressure 120/70/50 mm Hg in both arms. The peripheral pulses were collapsing in quality. The cardiac apex was in the sixth intercostal space at the anterior axillary line with a prominent left and right ventricular heave and a systolic apical thrill. The second heart sound was palpable. A grade III-IV/VI pansystolic blowing murmur was present at the apex with radiation to the axilla and left infrascapular area. A grade II-III high-pitched blowing diastolic murmur maximal in the right second intercostal space was heard well also along the lower left sternal border. A grade I-II systolic ejection murmur was heard along the lower left sternal border. The second heart sound was moderately increased in intensity and narrowly split. There was a prominent protodiastolic filling sound followed by a very short diastolic rumble at the apex.

Routine laboratory studies including complete blood count, urinalysis, and serology were normal. A sedimentation rate, C-reactive protein and antistreptolysin-O (ASO) titer were negative or within normal limits.

The electrocardiogram revealed atrial fibrillation, right axis deviation, digitalis effect, and evidence of biventricular hypertrophy.

Cardiac x-ray series showed marked cardiac enlargement of all chambers, especially the left atrium, and passive pulmonary congestion.

Cardiac Catheterization

The patient was considered to have marked mitral insufficiency and moderately severe aortic insufficiency and underwent left and right heart catheterization prior to surgery. There was no step-up in oxygen saturation. Arterial saturation was 98%. Pressure in the pulmonary artery was 42/22 with a mean of 25 mm. The mean right atrial pressure was 5 mm. No systolic gradient was present across the pulmonary valve and no
diastolic gradient across the tricuspid valve. The retrograde left heart catheterization revealed an aortic pressure of 105/60 with a mean of 80 with no systolic gradient across the aortic valve. The left atrium was not entered, and the pulmonary arterial wedge pressure could not be obtained. The cardiac output by the Fick method was 3.4 L per minute (index of 2.1 L). A cineangiogram with injection into the left ventricle revealed marked mitral regurgitation, a very large left atrium, and rather limited motion of the mitral valve. Injection into the aortic root demonstrated at least moderate regurgitation of dye, some of which was directed eccentrically rightward and inferiorly in the left anterior oblique projection. The results of the catheterization indicated rather marked mitral insufficiency, aortic insufficiency, and mild to moderate pulmonary hypertension.

Surgical replacement of the mitral and possibly the aortic valve as well was recommended.

Operative Report

Initial exploration revealed incompetence of the aortic root in diastole, and digital exploration of the left atrium showed overwhelming mitral incompetence. Marked enlargement of all chambers was present.

The aortic cusps were scarred and grossly deformed with the noncoronary one being greatly dilated and displaced into the ventricle. A large sinus aneurysmal orifice was then noted in the depth of this sinus which had ruptured into the left atrium. Replacement of the aortic valve with a Starr-Edwards valve restored competence to the aorta and the cuff of the valve effectively was utilized to repair the aortic-left atrial fistula. Further repair was effected by buttressing the left atrial aspect of the lesion with Teflon felt after excising the “grape-like” sac so characteristic of this lesion.

The mitral valve was typically rheumatic and was replaced. Effective cardiac activity, however, was never obtained although circulation was supported for approximately 1 hour following repair.

Pathological Findings

Postmortem examination revealed the presence of visceral passive congestion. The 550-g heart showed marked dilatation of all chambers, and both the aortic and mitral valve prostheses were securely placed. After removal of the prosthetic valves at the base of the noncoronary sinus of Valsalva a 5 to 6 mm circular defect was noted which communicated with the left atrium. The orifice in the left atrium was approximately 4 mm in diameter (fig. 1). The right and left sinuses and coronary vessels were unremarkable.

Microscopic studies of the myocardium showed numerous Aschoff bodies in various stages of development.

Discussion

Sinus of Valsalva aneurysms and fistulae have been associated with syphilitic aortitis, bacterial endocarditis, Marfan’s syndrome, coarctation of the aorta, penetrating wounds, and dissecting aneurysms of the aorta, and may occur iatrogenically following repair of a ventricular septal defect; sinus aneurysms may certainly occur as an uncomplicated con-

Figure 1

(A) Aortic root with the valve cusps removed and the arrow pointing to the orifice of the aorto-atrial communication. It arose from the noncoronary sinus of Valsalva. (B) Atrial aspect of the communication. The mitral and aortic valves have been removed.
genital malformation. The unruptured aneurysms may be symptomless and discovered only incidentally in the course of x-ray examination. Ruptured aneurysms present usually with pain, signs of congestive heart failure, and murmurs of a continuous or to-and-fro nature.

Rheumatic heart disease has only uncommonly been associated with a sinus aneurysm. Whether the valvular disease was related in any way to the communication in our patient is unknown. We postulate, however, that the rheumatic mitral valvular insufficiency permitted such gross enlargement of the left atrium so as to allow the noncoronary sinus to become adjacent thereto. Normally this sinus is in relation to the septal wall of the right atrium. We further estimate that 40% of the aortic insufficiency was due to the fistula.

The diagnosis of an aortic-left atrial communication such as occurred in this patient, particularly in the presence of acquired aortic and mitral valvular insufficiency is very difficult. An aortic-left atrial communication produces a left-to-left shunt and should theoretically present with a continuous murmur, signs of aortic runoff, and left heart enlargement in the absence of other cardiac disease. The findings of elevated pressure and moderately large V waves on the left atrial pressure tracing like those of mitral insufficiency would be helpful.

Injection of contrast material into the aortic root should afford the best means of diagnosis, particularly if there is no associated mitral or aortic valvular regurgitation. Ideally, passage of the catheter from the aortic root into the left atrium would be diagnostic.

Summary

A case of aortic-left atrial communication is presented in a patient with chronic rheumatic heart disease. The rarity of this lesion, especially in association with rheumatic heart disease, is stressed and the diagnostic and therapeutic problems inherent in such a lesion are briefly discussed.

Acknowledgment

The authors wish to express their appreciation to Drs. C. C. Welch, H. J. Rubenstein and R. H. Sueper, and to Mrs. Helen Bradford for their assistance in preparation of this paper.

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Circulation. 1966;34:52-54
doi: 10.1161/01.CIR.34.1.52

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/34/1/52

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