False Aneurysm of the Left Ventricle Secondary to Bacterial Endocarditis with Perforation of the Mitral-Aortic Intervalvular Fibrosa

By Elliot Chesler, M.B., M.R.C.P. (Edin.), Michael E. Korns, M.D., Gerald E. Porter, M.D., Cesárr N. Reyes, M.D., and Jesse E. Edwards, M.D.

SUMMARY

In an infant with bacterial endocarditis of the aortic valve a mycotic false aneurysm of the left ventricular outflow tract was a complication. The unusual nature of the case relates to the fact that the mouth of the false aneurysm lay in the mitral-aortic intervalvular fibrosa, the junctional tissue between the mitral and aortic valves. The close relationship between this structure and the epicardium was responsible for the peculiar location of the aneurysm in the epicardium between the aorta and the left atrium. Rupture of the false aneurysm led to a fatal hemopericardium.

Additional Indexing Words:
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Infarction of left parietal lobe

ERSIVE MYCOTIC ANEURYSM involving the region of the outflow tract of the left ventricle is an occasional complication of bacterial endocarditis. When the membranous septum or aortic sinuses are affected, extension of infection and rupture into a chamber of the right side of the heart may occur. In contrast, presentation of a left ventricular mycotic aneurysm into the epicardium is rare. This communication describes a case of bacterial endocarditis in which mycotic perforation of the outflow tract of the left ventricle resulted in a false aneurysm in the epicardium.

In this case of primary aortic valvular bacterial endocarditis a point of special interest was the site of secondary infection. This occurred in the junctional zone between the aortic and mitral valves, a zone designated as the "mitral-aortic intervalvular fibrosa." Infection of this region with its peculiar anatomic relationship to the epicardium was responsible for the rare position of the aneurysm to be described.

Report of Case

Clinical Features

The patient was a 4-month-old, white, female infant who, at the age of 3 months, was treated for severe generalized atopic dermatitis. A month later, she was admitted to the hospital with fever, the temperature being 104°F. Physical examination revealed mild residual dermatitis but no other abnormalities. Urinalysis gave negative results. The concentration of hemoglobin was 10.7 g/100 ml of blood. The total leukocyte count was 26,000 cells/mm³ of blood. A blood culture yielded numerous beta hemolytic streptococci. Penicillin was administered intramuscularly, and the fever subsided rapidly. A week after admission a systolic murmur was detected for the first time and this persisted until the time of discharge from the hospital 10 days later. Oral administration of penicillin was continued on an out-patient basis.
A week after leaving the hospital, the infant developed seizures involving the right side of the body. A few days later, after a spell of severe coughing and crying, she died suddenly.

**Pathologic Features**

A recent hemorrhagic infarct was found in the left parietal lobe of the brain.

There was a massive hemopericardium, the source of which was a ruptured false aneurysm situated at the base of the left ventricle (fig. 1a) and which will be described in greater detail. The aortic valve was composed of three cusps. The lowermost aspect of the posterior one third of the left aortic cusp contained a perforation measuring about 2 mm in diameter. The edges of the perforation were slightly thickened with tan granular material. Beneath the perforation of the cusp and in the zone of continuity between the anterior mitral leaflet and elements of the aortic valve there was a second perforation. The latter was round, measured about 8 mm in diameter, and exhibited smooth surfaces. This perforation represented the mouth of the aforementioned false aneurysm of the left ventricle (fig. 1b).

The aneurysm was roughly pear-shaped and measured approximately 3.5 by 2.5 cm. It presented in the transverse pericardial sinus between the posterior wall of the ascending aorta, an-
Low-power photomicrographs of sections stained for elastic tissue. Each × 2. Sections of normal (a) and from the case reported (b) made in same plane through the aorta (Ao.), the left aortic cusp (L.A.C.) and the anterior mitral leaflet (A.M.). In the normal (a) the tissue joining the aorta and mitral valves is readily apparent as the mitral-aortic intervalvular fibrosa (M.A.I.V. F.). Abutting this structure is a wedge (E.W.) of epicardial fat. This tissue lies between the left atrial wall, the beginning of the aorta and the mitral-aortic intervalvular fibrosa. L.V. = left ventricular cavity. In the case reported (b) there is a perforation in the mitral-aortic intervalvular fibrosa which represents the mouth of the false aneurysm (An.) illustrated in the gross specimen (fig. 1). The false aneurysm lies within the area normally occupied by the epicardial wedge. In this plane the section passes to one side of the perforation in the left aortic cusp.

It was of interest to compare the region of the aneurysm in the case under consideration with the same site in normal hearts. In the normal, the left half of the posterior aortic cusp and the adjacent one third of the left aortic cusp are continuous with the anterior leaflet of the mitral valve. The junctional zone between the elements of the two valves is formed by fibrous tissue which is called the “mitral-aortic intervalvular fibrosa.” Abutting the superior aspect of the mitral-aortic intervalvular fibrosa is a wedge of epicardial fatty tissue. In this way, there is within a relatively confined region rather close...
Figure 3

Diagrammatic representation of the features shown in figure 2. (A) corresponds to the normal (fig. 2a) while (B) corresponds to the features in the case reported (fig. 2b). P = pericardial cavity. In the normal the surface of the visceral pericardium lies near the epicardial wedge. In the case reported, distention of the epicardial wedge by the aneurysm has occurred. Perforation of the roof of the aneurysm led to hemorrhage in the pericardial sac.

anatomic relationship between the left wall of the left ventricular outflow tract and the epicardium (figs. 2a and 3a).

Gross and histological examinations in our case indicated that the perforation between the aortic and mitral valves was through the mitral-aortic intervalvular fibrosa and that the position of the aneurysm was primarily in the related epicardial fatty wedge (figs. 2b and 3b).

Histological examination showed that the wall of the false aneurysm consisted of a narrow layer of vascular connective tissue having the appearance of granulation tissue containing neutrophilic leukocytes and lymphocytes in irregular concentration. In some areas, the wall was attenuated, and there were focal aggregates of exudate which consisted of fibrin, polymorphonuclear leukocytes, and nuclear debris. In some of these areas there was incipient organization of the exudate (fig. 4a).

The margin of the perforation in the base of the left aortic cusp showed an inflammatory reaction similar to that in the lining of the aneurysm and granulation tissue was deposited upon the surface of the cusp (fig. 4b).

The epicardium of the anterior wall of the left ventricle was minimally thickened and contained occasional plasma cells and lymphocytes. A small epicardial branch of the left coronary artery contained an embolus composed of fibrin and a small amount of nuclear debris.

The valves other than the aortic were normal. The atria and the right ventricle were normal.

Comment

False aneurysms of the left ventricle are rare and may occur as results of trauma, myocardial infarction, and bacterial infection. False aneurysms which result from myocardial infarction usually occur in the anterior wall or the apex; those resulting from infection, however, have a predilection for the base of the left ventricle as these usually result from aortic valvular bacterial endocarditis.

The mitral-aortic intervalvular fibrosa is an area in the outflow tract of the left ventricle of strategic importance. Here the aortic wall as well as the left part of the posterior aortic wall and the adjacent third of the left aortic wall are connected with the base of the anterior mitral leaflet. This structure is in
juxtaposition with the apex of a deep epicardial wedge containing fat. This wedge intervenes between the posterior wall of the aorta and the left atrium; the mitral-aortic intervalvular fibrosa thus separates the outflow tract of the left ventricle from the pericardial cavity (fig. 3a). Infections involving this part of the left ventricular outflow tract may perforate into this pericardial wedge.

Reported cases of bacterial endocarditis in which false aneurysms of the left ventricle have resulted from perforation of the mitral-aortic intervalvular fibrosa are rare. In these, the wall of the aneurysm is usually described as consisting of connective tissue, organized blood clot, and granulation tissue. This appearance is consistent with the suggestion that a slow seepage of blood into the epicardial wedge had taken place and resulted in the false aneurysm; eventually the aneurysm ruptured into the pericardial sac because of the high pressure in the left ventricle.5,6

The structure of the false aneurysm in our case is suggestive of a more rapid process. The wall of the aneurysm was thin and composed largely of granulation tissue. The postulated sequence of events is as follows.

The primary infection was in the left aortic cusp. Following perforation of this structure, regurgitant and infected blood struck the mitral-aortic intervalvular fibrosa. The latter structure became secondarily infected. This,

Figure 4

in turn, led to perforation of the intervalvular fibrosa. Hemorrhage and infection then extended into the epicardial wedge (fig. 3b). The hemorrhage evaginated the epicardial wedge to form the false aneurysm situated between the aorta and the left atrium. It is postulated that the hemorrhage was contained within the epicardial sac for only a short time before the rupture, since there was no evidence of clotting of blood in the sac.

References

Fifty Years Ago—ECG Diagnosis of Coronary Disease

The changes in the T-wave . . . were so characteristic in dogs watched for several days, that similar changes in the wave in man might reasonably be supposed to be due to similar lesions. In fact, one case in man, which will be reported later, was observed in which a clinical diagnosis of coronary thrombosis was made by Dr. James B. Herrick which was verified later at necropsy. The T-wave of the electrocardiogram of the patient ran a course similar to that of the dogs previously described. In other cases believed to be coronary thrombosis similar changes in the electrocardiogram have been seen but no verification of the diagnosis has been made, the patients either living or no necropsy having been obtained.—Fred M. Smith: The Ligation of Coronary Arteries with Electrocardiographic Study. Arch Intern Med 22: 27, 1918.
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ELLIOT CHESLER, MICHAEL E. KORNS, GERALD E. PORTER, CESAR N. REYES and JESSE E. EDWARDS

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