THE LEFT VENTRICULAR ANEURYSMS that involve the fibrous body of the atrioventricular ring are distinct from the muscular left ventricular aneurysms that may occur in association with myocardial infarction. This unusual type of aneurysm was first described in studies of African Negroes, and a congenital weakness in the fibrous atrioventricular ring was postulated as a possible cause. Additional instances have since been reported, including 3 in Caucasian patients. One case was traumatic in origin, 1 myotic in origin, and 1 had no definite cause.

This report describes a patient with a small aneurysm of the left ventricle which involved the fibrous atrioventricular body. Fungal endocarditis involving the posterior mitral valvular leaflet had eroded into the subjacent atrioventricular ring, thereby resulting in a mycotic aneurysm.

CASE REPORT

A 15 year old boy had been in good health until January 1964, when there was an abrupt onset of fever, shaking chills, delirium, vomiting, malaise and pain in the fingertips. He was admitted to a hospital by his family physician, who detected an apical systolic cardiac murmur that had not been heard previously. Blood cultures revealed hemolytic Staphylococcus aureus. Treatment consisted of penicillin administered intravenously, and after four days the patient was transferred to the University of Iowa Hospitals. No past history of rheumatic fever or chorea could be obtained.

At physical examination on admission he appeared acutely ill, with sweating, shaking chills and rapid respirations. The temperature was 102.8°F, orally, the blood pressure 100/40 mm. Hg, and the pulse rate 90/min. There was neither cyanosis of the skin nor clubbing of the nails. Petechiae were found on the hands, feet and abdomen; furuncles were present on the back. The pharynx was hyperemic. There was no cervical vein distention and the peripheral arterial pulses were normal. The lungs were clear to examination. The heart was not enlarged, but an apical systolic thrill could be felt. The first and second cardiac sounds were normal, and no gallop sounds were heard. A grade 3/6 pansystolic blowing cardiac murmur was heard at the apex with radiation into the left axilla. There was no hepatosplenomegaly nor edema.

The hemoglobin was 10.5 gm./100 ml., white blood count 17,800 cells per cu. mm. with 2 per cent nonsegmented neutrophils, 75 per cent segmented neutrophils, 2 per cent eosinophils, 16 per cent lymphocytes, 4 per cent monocytes and 1 per cent myelocytes. The urine had a specific gravity of 1.010, and an acid reaction, and it contained several white and rare red blood cells. An antistreptolysin-O titer was 50 Todd units, and the C-reactive protein was 3+. Blood and urine cultures were sterile, and throat culture showed normal flora and Candida albicans. The electrocardiogram was normal, and the thoracic roentgenogram showed a normal cardiac configuration with normal pulmonary vasculature. There was a small right pleural effusion.

Treatment initially consisted of intravenously administered penicillin, methicillin and chloramphenicol, and the patient became afebrile. This therapy was discontinued after two weeks, and oral administration of oxacillin was begun. Three days later he again became febrile, and the spleen became palpable for the first time. Intravenous administration of penicillin and methicillin was reinstituted and continued for four weeks, during which time blood cultures were sterile. The boy again became afebrile and was discharged with no medication after two months' hospitalization. He was followed up as an outpatient and remained asymptomatic.

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Cardiac catheterization was performed in December 1965, 23 months after his initial illness. The intracardiac pressures (mm. Hg) were as follows:

<table>
<thead>
<tr>
<th>Site</th>
<th>Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary artery “wedge”</td>
<td>28/19 (10)</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>25/10</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>27/4</td>
</tr>
<tr>
<td>Right atrium</td>
<td>6</td>
</tr>
<tr>
<td>Ascending aorta</td>
<td>120/80 (94)</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>126/7</td>
</tr>
</tbody>
</table>

It was concluded from the study that the patient had moderately severe mitral valvular regurgitation. A persistent left superior vena cava draining into the coronary sinus was an incidental finding.

The boy underwent operation for correction of the mitral valvular regurgitation in June 1966. The anterior mitral leaflet was large and thickened and the chordae tendineae were intact. A defect, 1.5 cm. in length, was present in the posterosmedial aspect of the posterior mitral leaflet and allowed severe mitral regurgitation to occur. A graft of autologous fascia lata was sutured over this defect and an annuloplasty of the posterosmedial mitral commissure was performed. After left atrial closure a slight systolic thrill could be palpated over the left atrium.

After operation, oxacillin, 4 gm., and streptomycin, 1 gm., were given daily. Although numerous blood and wound cultures were sterile, daily temperature elevations to 101 or 102° F were noted. Streptomycin was discontinued on the fifth, and oxacillin on the eighth postoperative day. Twenty-four hours later treatment with ampicillin, 250 mg. administered orally every 6 hours, was begun. On the fourteenth postoperative day the patient experienced severe pain in the left groin, and a tender pulsatile mass was noted at the site of the femoral arterial cannulation. This was diagnosed as an aneurysm of the femoral artery, and at operation it was found that the cardiac bypass suture line was disrupted, thus forming a false aneurysm. The segment was resected and replaced with a saphenous vein graft. Routine cultures of the tissue were sterile. Methicillin, given intravenously, and streptomycin, intramuscularly, were administered for two weeks, at which time streptomycin was discontinued. The methicillin was continued for a total of four weeks. A grade 2/6 pansystolic apical murmur was present at the time of discharge.

Final Admission: The patient re-entered the University of Iowa Hospitals five months later with a complaint of severe low back pain of three weeks' duration. He was uncomfortable but was in no distress. The temperature was 101° F. orally, and the pulse rate was 84 and regular. Petechiae of the left palpebral conjunctiva and splinter hemorrhages of the fingernails were observed. There was a marked overactivity of the apical impulse, and no thrills were present. A grade 3/6 pansystolic murmur was present at the apex. The spleen and liver were not enlarged. There was marked lordosis of the lumbar spine with severe tenderness over the spinous processes of the third to fifth lumbar vertebrae. Brudzinski’s and Kernig’s signs were present.

The hemoglobin was 13.5 gm./100 ml., the white blood count was 13,000 cells per cu. mm., with 69 per cent segmented neutrophils, 2 per cent eosinophils, 21 per cent lymphocytes and 8 per cent monocytes. The urine had a specific gravity of 1.016, acid pH, 1+ protein, 2+ Meyer’s reaction for blood, and numerous red blood cells. The cerebrospinal fluid was normal. Numerous blood cultures for aerobic bacteria and for fungi were sterile. An electrocardiogram and thoracic roentgenogram were normal. Lumbosacral spine roentgenograms revealed destruction of the inferior surface of the first and the superior surface of the second lumbar vertebrae with decrease in the disc space (Fig. 1A). Temperatures of 102 to 103° F. orally occurred daily.

Treatment consisted of numerous antibiotics, including penicillin, methicillin, oxacillin, streptomycin, colistimethate, chloramphenicol, cephalothin, vancomycin, novabacillin and sulfadiazine. There was no appreciable alteration of the progressively deteriorating, wasting, painful and febrile course. Except for two blood cultures that revealed a flavobacterium species, numerous blood cultures were sterile. On March 29, 1967, an infected and degenerated disc was removed from the first and second lumbar interspace. Cultures of this tissue showed a klebsiella-aerobacter organism and an aspergillus species, but microscopic sections of the disc failed to show the fungus and it was considered to be a contaminant.

The patient’s condition deteriorated. Numerous petechiae and painful embolic purpura appeared on the hands (Fig. 1B). On April 27, 1967, a left hemiparesis appeared, followed rapidly by stupor and death.

NECROPSY FINDINGS

There were diffuse punctate hemorrhages over the entire body. Acute bilateral bronchopneumonia was found. An embolus occluded the right middle cerebral and anterior communicating arteries and had caused a recent right cerebral infarct. There were recent infarcts of spleen, small bowel and both kidneys. The serosal surfaces of the bowel were covered with petechiae, as were the renal surfaces.

The heart weighed 460 gm. A healed pericarditis produced minor adhesions between the visceral and parietal pericardium. The coronary arteries were patent and normal. A persistent left superior vena cava drained into the dilated coronary sinus. The right atrium and ventricle were normal.

The left atrium was moderately dilated and, on opening, a huge mass of verrucous material was revealed (Fig. 2A). The mitral valve was opened by cutting across the anterior mitral leaflet near the ventricular septum to better evaluate the origin of the mass. This revealed a large vegetation arising from the posterior leaflet near the posterosmedial commissure.
Figure 1. A, lumbar spine, lateral roentgenogram. An expanding lesion of the intervertebral disc had caused erosion of the adjacent lumbar vertebral surfaces (point of arrows). B, large necrotic and painful embolic lesion of palm.

(Fig. 2B) in the location of the fascia lata graft inserted nine months earlier. The vegetations consisted of one large mass and numerous smaller nodules which hung, legume-like, attached to the chorda tendineae of the mitral valvular leaflets.

Probing the ventricular surface of the posterior mitral leaflet revealed an aneurysmal orifice (Fig. 2C) that penetrated the fibrous atrioventricular ring posteriorly and had as its posterior wall the coronary sinus. A material similar to the vegetation filled the aneurysm.

Microscopic examination of sections taken sagittal to the aneurysmal orifice revealed the fibrous atrioventricular body of the posterior mitral leaflet to be totally destroyed (Fig. 2D). The posterior mitral leaflet was replaced by amorphous eosinophilic material and debris, and no evidence could be found of the fascia lata graft.

Sections of the ventricular myocardium revealed small microabscesses, often with foreign body giant cells. With a fungal stain these abscesses consistently revealed mycelia. Similar microabscesses were present in almost every organ, and cultures of these abscesses grew Aspergillus terreus.

Comment

The majority of left ventricular aneurysms develop as a result of myocardial infarction. Other documented causes include trauma,7 mycotic infection,8 syphilis,9 tuberculosis,10 rheumatic myocarditis11 and periarteritis nodosa.12 Distinct from these aneurysms are the left ventricular aneurysms which involve the membranous septum and the atrioventricular ring. The latter have been virtually confined to Negroes and have been termed annular subvalvular aneurysms.2-4,13 Layman and January6 proposed fibrous body aneurysm as a more appropriate term because any left ventricular aneurysm must have its origin subvalvularly, but only this type of aneurysm dissects the fibrous body of the atrioventricular ring. The exact cause is not known, but most authors5-13 concur that a basic defect in structure involving the atrioventricular fibrous skeleton must be present. Chesler et al.14 have postulated that atrioventricular ring
Figure 2. Sections of heart. A, left atrium. The atrium was opened posteriorly, exposing a large verrucous mass filling the atrial cavity. B, left atrium and ventricle. The huge verrucous mass almost entirely filled the left atrium, and was attached to the posterior mitral leaflet. Smaller masses were attached to the chordae tendineae. C, orifice of fibrous body aneurysm. The orifice (point of arrows) was exposed by lifting the posteromedial papillary muscle (PPM). D, microscopic section taken sagittal to the orifice of the aneurysm. The left atrium (LA) was separated from the left ventricle (LV) by an aneurysmal sac filled with amorphous debris. The posterior mitral leaflet (PM) was replaced by amorphous eosinophilic tissue.

aneurysms arise from congenital fibrous diverticulae which are the result of endocardial retention clefts.

In our case the aneurysm resulted from destruction of the atroioventricular body by an infective process (Aspergillus terreus). The first definitive instance in which a fibrous body aneurysm resulted from an infective process (Brucella suis endocarditis) was reported by Layman and January.6 We cannot be certain that in the present case the aneurysm did not result from a previous underlying defect, but the
intimate proximity of the orifice to the area of infection strongly suggests its mycotic origin.

Fungal endocarditis is rare and difficult to diagnose. Jamshidi et al. reviewed 14 cases of fungal endocarditis complicating cardiac surgery. The infecting organism was candida in all cases, and most were fatal. Recent reports describe four additional cases of candida endocarditis and the first case of aspergillus endocarditis known to occur as a postoperative complication of open heart surgery. The present case is the twenty-first example of fungal endocarditis after open heart surgery and the second example caused by aspergillus.

**SUMMARY**

This report describes a patient with a small aneurysm of the left ventricle which involved the fibrous atrioventricular body and was caused by aspergillus endocarditis. Aneurysms of the fibrous atrioventricular ring are distinctly different from other types of aneurysms. The majority have been confined to young adult African Negroes. The etiology is uncertain, although a structural weakness in the atrioventricular ring has been postulated. In our patient fungal endocarditis (Aspergillus terreus) occurred after open heart surgery for mitral regurgitation and involved the mitral valvular leaflets. Erosion into the subjacent atrioventricular ring resulted in the mycotic aneurysm.

**REFERENCES**