Mycotic False Aneurysm of the Aorta Following Aortic Valvular Prosthesis

A Case Report

By Nobuhisa Baba, M.D., and Trevor L. McKissick, M.D.

Since the introduction of the Starr-Edwards ball valve into cardiac surgery in 1961, a number of successful operations have been recorded. This is a case report of a mycotic false aneurysm of the ascending aorta resulting in fatal incompetence of the prosthetic valve.

Case Report

A 39-year-old white man with acute rheumatic fever at the age of 19 years remained asymptomatic until 3 years before demise, when he started to have fainting episodes associated with chest pain and one year later symptoms of congestive heart failure.

Examination at the Ohio State University Hospital showed signs of aortic stenosis, and cardiac catheterization revealed a pressure gradient of 90 mm. water across the aortic valve. He underwent open-heart surgery for the placement of a Starr-Edwards prosthetic valve in the aortic ostium. The postoperative course was complicated by anemia and wound infection with a coagulase-positive Staphylococcus but he responded to treatment fairly well. Four months after the operation the patient developed fatigue, occasional fever, and chills. One week before his last admission he noted petechiae, dyspnea, and orthopnea. Five months after the operation he was again admitted to University Hospital.

On physical examination at this admission the patient was slightly dyspneic, the temperature was 100.2 F., and the blood pressure was 112/40. The neck veins were minimally distended at 30° position. The lungs revealed bilateral rales, particularly in the bases. The heart rate was regular at 87 per minute. The point of maximal impulse was in the sixth intercostal space at the left anterior axillary line. There were a grade-V/VI holosystolic regurgitant murmur throughout the precordium radiating to the left axilla, a grade-III/VI systolic ejection murmur in the right second intercostal space radiating toward the apex, and a grade-III/VI high-pitched blowing diastolic murmur along the left sternal border, intense in the third intercostal space. A prominent opening snap was heard. The liver edge was felt two fingerbreadths below the right costal margin. Pretibial and pedal edema was present, and petechiae were seen over the extremities. The nail beds showed splinter hemorrhages.

The patient's hemoglobin was 7.9 Gm. per 100 ml., the hematocrit level 25 per cent; reticulocytes 3.3 per cent; total leukocytes 10,400 per mm. The half-life of Cr51-tagged erythrocytes was 7½ days, and the blood smear showed pyknocytosis. The blood urea nitrogen was 39 mg. per 100 ml. All 12 blood specimens grew a coagulase-negative Staphylococcus. On radiologic examination the prosthesis was in place. The electrocardiogram suggested incomplete bundle-branch block and left ventricular hypertrophy.

The patient was given 40 million units of intravenous penicillin daily and his fever came down. One week after admission the chest wound started to drain and again a coagulase-positive Staphylococcus was cultured. A radiologic examination suggested widening of the ascending aorta due to an aortitis. Three weeks after admission the anterior chest abscess was noted to pulsate, and an attempt at incision and drainage was given up. On the forty-sixth hospital day the patient's temperature suddenly rose to 102 and petechiae spread over the skin. A radiologic examination at this time showed excessive rocking motion of the prosthesis, indicating loosening of the valve from its attachment. The valve was seen at an angle of 70°. His blood pressure changed to 110/0, and an electrocardiogram showed second-degree heart block. He died on the forty-ninth hospital day.

Autopsy Findings

On the chest wall of the body was a partially healed scar running from the suprasternal notch to the xiphoid process, showing a few areas of breakdown from which a very small amount of exudate was drained. The subcutaneous tissue contained small abscesses. In the right pleural cavity were 50 ml. of serous fluid; in the left, 75 ml. The entire pericardial cavity was loosely

From the Department of Pathology, The Ohio State University, Columbus, Ohio.

Circulation, Volume XXXI, April 1965
fibrosed. The heart, including the artificial valve and an aneurysm, weighed 780 Gm. The left atrium was moderately dilated. The left ventricle measured 18 to 20 mm. in thickness; the right, 3 to 4 mm. The myocardium of the posterior wall showed several patchy areas of fibrosis. The artificial valve was movable and was sutured to the annulus only in the posterior sinus, and in the sinus the Nylon sutures had been torn from the wall leaving defects. Six millimeters above the commissure between the posterior and left sinuses was an organized thrombus on the aorta with a central depression having the conformation of the surface of the plastic ball-valve. This thrombus probably formed when the prosthesis moved slightly to the left. The widest separation of the prosthesis from the aortic wall was in the right sinus. Also in the right sinus wall there was a triangular defect 3 cm. in width and in height at and above the level of the prosthetic ring. An irregular fibrin mass 2 cm. in diameter was attached on the artificial ring. The defect formed the opening of a false aneurysm between the ascending aorta, the remaining right atrium, and the right lung. The aneurysmal space measured 5 by 3 by 2 cm. and contained fresh and clotted blood. The outer wall consisted of dense post-surgical fibrinous adhesions, and the inner surface was lined by organized thrombi (fig. 1). There were also many organized fibrin masses on the endocardium of the subvalvular region although no defect was seen in this area. The base of the triangle lay along the previous valvular annulus. The previous surgical incision was intact and endothelium covered the Nylon sutures at the incision of the aorta, which ran vertically 1 cm. to the left of the rupture (fig. 2). The rest of the aortic root was slightly widened and showed only small atheromatous plaques. Neither coronary ostium was involved. The right artery was very hypoplastic. The anterior descending branch of the left coronary artery showed a pin-hole lumen 3 cm. from the ostium. The ball valve was partially endothelialized. The other valves showed no deformities. The annular circumferences of the mitral and tricuspid valves were increased.

The right lung weighed 500 Gm., the left 450 Gm. Both lungs showed edema, congestion, and focal atelectasis. The liver weighed 1,850 Gm. and showed moderate congestion. The spleen weighed 300 Gm. and showed areas of mycotic infarction. The right kidney weighed 150 Gm. and the left kidney 300 Gm.; they showed severe congestion and cloudy swelling.

The verrucous material attached to the aorta and the prosthesis was mainly fibrin with rare aggregates of neutrophils; no bacterial colonies were seen. There were several pieces of hyalinized tissue with occasional calcification originating from the annulus of the valve. This mass was not endothelialized. The aorta showed moderate intimal ulceration, but the most remarkable changes were seen in the outer media and the adventitia; these changes consisted of heavy proliferation of chronic granulation tissue with many capillaries, fibroblasts, and histiocytes. Occasional hemosiderin deposits were found. In the area of rupture the media appeared abruptly torn and the edge was surrounded by the granulation tissue; no degenerative process was noted in the elastic laminae. The base of the aneurysm was composed of dense, partially hyalinized fibrous tissue covered with a thick layer of fibrin which contained numerous

---

**Figure 1**


**Figure 2**

Root of aorta. 1: intact incision line. T: thrombotic material on prosthesis indicating both bacterial endocarditis and presence of aortic insufficiency at this portion. Arrow: opening of aneurysm.
Mycotic False Aneurysm

pus cells. Many pigment-laden macrophages were also seen (fig. 3).

The sections from lungs, liver, spleen, and kidneys confirmed the gross interpretation. No embolic focal glomerulitis was present.

Discussion

Mycotic aneurysm of the aorta is relatively uncommon, as shown by Parkhurst and Decker, who reviewed 22,792 autopsies at the Boston City Hospital performed between 1902 and 1951 and found only 12 cases of bacterial aortitis, nine of which were associated with aneurysm. Earlier, Auerbach reviewed 135 cases of purulent aortitis, including 15 cases of his own. Aneurysm formation was seen in 58 of them. Edwards showed several case illustrations of mycotic aneurysms associated with bacterial endocarditis. One case had mycotic aneurysms originating from the posterior aortic sinus, much as the aneurysm in our case was located.

Most of the mycotic aneurysms in the ascending aorta were associated with bacterial endocarditis of the aortic valve, although mycotic aneurysm may be observed following syphilitic aortitis, atherosclerosis, and dissecting aneurysm. Some lesions in the high ascending aorta, as reported by Merkel, may be secondary to remote trauma. Isolated mycotic aneurysms are rare and are usually associated with chronic septicemia, remote bacterial infections, or congenital malformations, such as hypoplasia and coarctation. The organisms reach the aorta through the vasa vasorum, with or without embolism, and the intima is relatively uninvolved.

Aneurysm formation following aortic valve surgery is either traumatic or mycotic. Campbell reported a traumatic false aneurysm of the ascending aorta 13 months after the debridement of the aortic valve for calcific stenosis. The aneurysm was successfully ligated. Eliot et al. reported a traumatic false aneurysm following needle puncture of the aorta. The patient died of staphylococcal mediastinitis. Hadorn noted a mycotic aneurysm due to extension of aspergillus endocarditis following the removal of calcified subaortic stenosis.

In the majority of cases bacterial aortitis, with or without aneurysm, is caused by gram-positive cocci, particularly pneumococci and streptococci. Parkhurst and Decker found six pneumococcal and two streptococcal infections among 12 cases. Auerbach reviewed 70 cases of aortitis with bacterial studies and found streptococci in 26 cases, pneumococci in 12, and staphylococci in eight cases. Edwards presented several cases of mycotic aneurysms caused by streptococci and Staphylococcus aureus. Gram-negative bacilli are rarely implicated, but Salmonella infect the arteriosclerotic aneurysms of the abdominal aorta. According to Rob and Ng, staphylococci were often the causative agents in the primary mycotic aneurysms of the lower aorta and its major branches. Apparently chronic staphylococcal sepsis was an important factor. In all but one case the staphylococci were of the aureus group.

Coagulase-negative staphylococcal infection, as seen in our case, is rather unusual. The heavy penicillin treatment given for this highly drug-sensitive organism could not clear
the active inflammatory process in the false aneurysm. The chronic inflammatory process within the aortic wall was still in active progress and contributed to the terminal tear of the prosthesis and incompetence of the valve.

Summary

A case of a mycotic false aneurysm of the ascending aorta and aortic sinus caused by coagulase-negative staphylococcal endocarditis following the placement of a Starr-Edwards prosthesis for rheumatic aortic stenosis is reported. The formation of the aneurysm caused aortic insufficiency, and terminally the prosthesis was torn from the aorta. The occurrence of mycotic aneurysms in the aorta and their etiology and pathogenesis are discussed. Cases of postoperative false-aneurysm formation of the ascending aorta are reviewed.

References


As no two faces, so no two cases are alike in all respects, and unfortunately it is not only the disease itself which is so varied, but the subjects themselves have peculiarities which modify its action.—SIR WILLIAM OSLER. Aphorisms From His Bedside Teachings and Writings. Edited by William Bennett Bean, M.D. New York, Henry Schuman, 1950, p. 34.
Mycotic False Aneurysm of the Aorta Following Aortic Valvular Prosthesis: A Case Report
NOBUHISA BABA and TREVOR L. MCKISSICK

Circulation. 1965;31:575-578
doi: 10.1161/01.CIR.31.4.575

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1965 American Heart Association, Inc. All rights reserved.
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/31/4/575

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/