BACTERIAL AORTITIS AND MYCOTIC ANEURYSM OF THE AORTA

A Report of Twelve Cases *

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Because of the resistance of the large elastic arteries to bacterial infection in general, mycotic (bacterial) aneurysms of the aorta are among the more uncommon encountered in that artery. Infection in the arterial wall is rarely due to fungi and the inappropriateness of the term mycotic is generally recognized, but it has the advantage of long and common usage since it was applied to these lesions by Osler.[1] It is the purpose of this communication to report 12 cases in which there was bacterial infection of the aortic wall with formation of an aneurysm in 9 of these cases. The 3 cases without aneurysm formation do not differ fundamentally in pathogenesis and are included for that reason. Three cases included in this study have previously been reported.[2-4]

Incidence

During the 50-year period of 1902 to 1951, 22,792 necropsies were performed at the Boston City Hospital. In these cases, 338 aortic aneurysms were encountered: 143 syphilitic, 92 arteriosclerotic, 78 dissecting, 9 mycotic, and 16 unclassified. The incidence of aortic aneurysm was 1.5 per cent.

During the review of these cases, some observations were made on the incidence of the more common types of aortic aneurysm which are worthy of inclusion. Of the 143 syphilitic aneurysms, 124 occurred in men and 19 in women. The syphilitic lesions were found in the thoracic aorta in all but 2 cases. In 37 cases death was due to internal rupture of the aneurysm; external rupture was not noted. Most of the syphilitic aneurysms (76 per cent) were encountered in the age group 41 to 70 years. Similarly, a majority of the arteriosclerotic aneurysms occurred in men, 60 of the total of 92. In contrast to the predominantly thoracic location of the syphilitic aneurysms, 73 of the arteriosclerotic group were found in the abdominal aorta and but 13 in the thoracic aorta. In 6 cases there were multiple arteriosclerotic aneurysms. Most of these aneurysms (88 per cent) occurred in the age group 61 to 90 years.

* Received for publication, January 6, 1955.
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TABLE I

Data on Twelve Cases of Bacterial Aortitis

Case	Sex	Age	Location	Description	Other aortic disease	Source of infection	Type	Other findings
1	M	30	Descending thoracic	1.5 cm. aneurysm		Tuberculous lymphadenitis	Secondary	Miliary tuberculosis
2	M	34	Sinus of Valsalva	1.2 cm. aneurysm		Aortic endocarditis	Secondary	Lobar pneumonia
3	M	44	Ascending thoracic	Longitudinal slit, rupture with hemopericardium	Atherosclerosis, cystic medionecrosis		Primary	Calcific aortic stenosis
4	F	29	Ascending thoracic	3 cm. aneurysm		Subacute bacterial endocarditis	Secondary	Trivalvular rheumatic heart disease
5	M	39	Sinus of Valsalva	3 cm. aneurysm, rupture and hemopericardium	Atherosclerosis	Subacute bacterial endocarditis	Secondary	Rheumatic heart disease
6	M	53	Aortic valve ring	1 cm. aneurysm	Atherosclerosis	Bacterial endocarditis	Secondary	
7[3]	M	35	Ascending thoracic	Multilocular aneurysm, 6 x 4 x 3 cm.	Congenital hypoplasia	Cellulitis of foot	Primary	Splenic and renal infarcts, focal embolic glomerulonephritis
8[3]	M	39	Ascending thoracic	Fusiform and saccular aneurysms, 4 x 2 x 2 cm.	Syphilitic aortitis	Gonococcal arthritis	Primary	
9[4]	M	75	Ascending thoracic	0.5 cm. slit, rupture with hemopericardium	Atherosclerosis	Bacterial endocarditis	Secondary	
10	M	9 mo.	Descending thoracic	Slit communicating with esophagus		Mediastinitis	Secondary	
11	M	55	Descending thoracic	5 cm. aneurysm, rupture into right pleural cavity	Atherosclerosis	Pneumonia (?)	Primary	
12	M	68	Descending thoracic	10 cm. aneurysm, rupture into posterior mediastinum	Cystic medionecrosis	Pneumonia	Primary	

Observations made on the frequency with which the various types of aortic aneurysms were encountered during the 50-year period indicate an increase in incidence of arteriosclerotic aneurysms, paralleling an increase in the average age of patients necropsied, and a sharp drop in the incidence of syphilitic aneurysms, attesting to the efficacy of improved methods for the treatment of syphilis. In the first 4 decades, 1902–1941, syphilis was responsible for 52 to 70 per cent of all aortic aneurysms. In the last decade, however, there was a drop to 25 per cent. Contrariwise, there was noted a steady rise in the incidence of arteriosclerotic aneurysms until they accounted for 43 per cent of all aortic aneurysms in the last decade included in the study, 1942–1951. These observations are in agreement with those of Maniglia and Gregory.[5] Dissecting aneurysms were not studied in detail.

The 12 cases of bacterial aortitis and mycotic aneurysm of the aorta showed neither a tendency to occur in a particular age group nor a detectable change in incidence over the 50-year period; but the number of cases is very small in relation to the total. Eleven of the 12 cases occurred in men; all of the lesions were found in the thoracic aorta. Pertinent pathologic data are collected in Table I.

Pathologic Findings

In size, the lesions ranged from a 1 mm. erosion to a 10 cm. sac (Figs. 1, 2, and 3). In 3 cases (nos. 3, 9, and 10), infection in the aortic wall had produced an erosion and rupture without aneurysmal dilatation. In 6 cases there was rupture of the lesion as follows: into the pericardial cavity in 3 cases, and into the right pleural cavity, the posterior mediastinum, and the esophagus in one case each. Microscopically, there was noted destruction of the intima and musculoelastic lamellae by an acute inflammatory process (Fig. 4). Heavy infiltrates of neutrophils and abscess formation were noted frequently (Figs. 5 and 6). Bacteria often were demonstrable in tissue sections. In older lesions and at the margins of acute inflammatory zones there often was active fibroblastic proliferation and formation of granulation tissue. It is of particular interest that, in those cases in which aneurysms were unassociated with bacterial endocarditis or infection in adjacent structures and in several other cases as well, study of the aorta showed the presence of additional aortic disease which included atherosclerosis, cystic medionecrosis (Fig. 7), syphilitic aortitis, and congenital hypoplasia.

An intravascular source for infection was apparent in 5 cases. In 2 cases (nos. 1 and 10) the source of infection was found in adjacent structures in the mediastinum. Of the 5 remaining cases there was a

putative source for infection in remote structures in 4: a history of cellulitis of the right foot in case 7, a history strongly suggestive of gonococcal arthritis in case 8, a history suggestive of pneumonia 6 weeks before in case 11, and organizing bronchopneumonia found at necropsy in case 12. In case 3, no source for the infection was apparent. The results of bacteriologic studies are indicated in Table II.

TABLE II

Infecting Organisms in Cases of Bacterial Aortitis

Case	Organism	How demonstrated
1	*Mycobacterium tuberculosis*	Direct smear
2	Pneumococcus, type 1	Ante-mortem blood culture
3		
4	Gram-positive cocci	Sections of aneurysm
5	*Streptococcus viridans* Gram-positive cocci	Culture of vegetations Sections of aneurysm
6	Pneumococcus, type 28 Beta hemolytic streptococci	Culture of vegetations Culture of vegetations
7	Gram-positive cocci	Sections of aneurysm
8	Pneumococcus, type 17 *Neisseria gonorrhoeae*	Post-mortem culture of blood, aneurysm, and spleen Post-mortem culture of aneurysm and spleen
9	Atypical coliform organism	Culture of vegetations, tissue sections
10	Pneumococcus, type 33 Fusospirochetal organisms Gram-positive cocci	Post-mortem culture of lungs Tissue sections Tissue sections
11	Pneumococci, diplococci (Gram-positive)	Ante-mortem throat culture Tissue sections
12	Diplococci (Gram-positive)	Tissue sections

The bacteriologic findings indicate the prominence of Gram-positive cocci, particularly the pneumococcus, as causative organisms in these cases. These cocci, when identified culturally, were found to be pneumococci in 5 cases and streptococci in one case. Both gonococci and pneumococci were isolated in case 8. *Mycobacterium tuberculosis* and an atypical coliform organism were found in one case each (nos. 1 and 9). There was one case of fusospirochetal infection (case 10).

ILLUSTRATIVE CASES

Case 2

A 34-year-old white man was admitted with a history suggestive of lobar pneumonia and physical findings were compatible with that diagnosis. Blood cultures

were positive for a pneumococcus, type 1. On the 39th hospital day, the patient developed high fever with rising pulse and respiratory rates. He suddenly developed extreme dyspnea and died rapidly on the 41st hospital day.

At necropsy there was lobar pneumonia of the right lung. On the left anterior aortic valve cusp, near the edge, there was a vegetation 7 mm. in diameter. Above the right anterior cusp was an aneurysm of the sinus of Valsalva, 1.2 cm. in diameter. On microscopic examination of sections taken through the involved aortic cusp and adjacent aneurysm there was necrosis and destruction of the cusp with a typical bacterial vegetation. The aneurysm contained laminated blood clot and the underlying aortic wall was heavily infiltrated with neutrophils and contained scattered abscesses (Fig. 6). Masses of cocci were present in the sections.

Case 3

A white man, 44 years old, who did not appear acutely ill, was admitted to the hospital because of lassitude, fatigue, and malaise. The heart was enlarged to the left and a rough systolic murmur at the apex entirely replaced the first sound. There was also a short, blowing, mid-diastolic murmur at the apex. Over the second, third, and fourth left intercostal spaces was a loud friction rub. The clinical diagnosis was pericarditis. The patient died quietly in his sleep.

At necropsy there was an acute fibrinous pericarditis and hemopericardium (200 cc.). The aortic valve was thickened, with adherence of two cusps, presenting the appearance of nodular calcific aortic stenosis, but without evidence of fresh endocarditis. There was an irregular, longitudinal slit, 1.5 cm. above the aortic valve. The borders of this slit were raised, irregular, and bore small vegetations. A probe passed through it entered the pericardial cavity. On microscopic examination of the aorta, there was atherosclerosis and cystic medionecrosis (Fig. 7) with dissection. In the region of the perforation there was destruction of the aortic wall by an acute inflammatory process, with deposits of fibrin and neutrophils, formation of abscesses, and marginal fibroblastic proliferation.

Case 10

A white male infant, 9 months of age, was admitted because of anorexia, restlessness, and fever for 1 week. On the day before admission there were two episodes of hematemesis. Fifteen minutes after admission there was another bout of hematemesis and the patient died. There was no history of ingestion of a foreign body.

At necropsy, there was found just beyond the origin of the left subclavian artery a small area, 0.5 cm. in diameter, in which the aorta appeared thinned and slightly discolored. In the center of this area was a small hole, 1 mm. in diameter (Fig. 1), through which a probe could be passed into the esophagus. Between the aorta and the esopha-

gus was a fistulous tract lined by ragged, soft red tissue. In the esophagus was a longitudinal slit, 8 mm. in diameter, which communicated with the sinus tract. The gastrointestinal tract contained large amounts of dark red blood. On microscopic examination the fistulous tract was found to be lined with necrotic débris and fresh granulation tissue. There were many collections of neutrophils and small abscesses. Bacterial stains revealed a variety of organisms, including short fusiform bacilli, large spirochetes, cocci in short chains and clusters, and short bacilli.

Case 11

A 55-year-old man was admitted to the hospital because of right anterior chest pain. He had had chronic cough with expectoration for many years, with occasional attacks of pain in the left knee and ankle. Two years previously he had had several abscesses drained in the right foot and had not worked since that time. Six weeks before admission he had a bout of stabbing right upper abdominal pain aggravated by inspiration and coughing. Temperature was 100.8° F. On physical examination there was dullness over the entire right hemithorax with diminished breath sounds over that area. Serologic test for syphilis was negative. Culture of the sputum yielded a pneumococcus. Roentgenograms of the chest showed an increase of bronchovascular markings on the right and slightly diminished radiolucency in the right middle and lower lung fields. These changes were interpreted as suggestive of pneumonitis. The patient was treated with penicillin. Ten hours after admission he had a generalized convulsion and expired within 40 minutes.

At necropsy the right pleural cavity contained a large blood clot weighing 3,000 gm. Just above the crura of the diaphragm there was an aortic aneurysm, 5 cm. in diameter. In the intimal surface this was apparent as a shallow depression with ragged edges and a slit-like tear in the center (Fig. 2). On microscopic examination all coats of the aorta were infiltrated with neutrophils, often aggregated into small abscesses (Fig. 5). There was partial necrosis and fragmentation of the medial lamellae, particularly in the region of the perforation which communicated with the right pleural space. Paired Gram-positive cocci were identified in appropriately stained sections of the aortic wall.

Case 12

A white man, 68 years old, was admitted because of severe substernal and epigastric pain of 6 hours' duration. During the 7 years before admission, he had had infrequent paroxysms of dyspnea, wheezing, and cough which were not incapacitating. Six months before admission he developed increasing hoarseness, for which a cause was not found on laryngoscopic examination. About 6 hours before admission he complained of excruciating substernal pain associated with severe dyspnea, cyanosis, and sweating. Physical examination was negative except for rhonchi and wheezes in both lungs. The temperature was 101° F.; pulse rate, 120; respirations, 30; leukocyte count, 18,000 per cmm. with 81 per cent neutrophils. Serologic test for syphilis was negative. Slight deviation of the trachea and superior mediastinal contents to the right was noted on roentgenologic examination of the chest. A barium study of the

esophagus showed almost complete extrinsic obstruction in the middle third. Two episodes similar to the admitting complaint occurred during the following 9 days. Shortly after the second such episode, the patient died.

At necropsy, there was a fusiform aneurysm of the descending thoracic aorta, 10 cm. in length and 13 cm. in maximum circumference. It had ruptured anteromedially in the plane between the aorta and esophagus, and had compressed the esophagus in its middle third (Fig. 3). The presence of old blood clot and fibrous tissue deposition in this area suggested previous leakage. The recurrent laryngeal nerve was not identified. In microscopic sections there was destruction of all layers of the aortic wall with fragmentation of elastic fibers (Fig. 4). On the inner surface of the aneurysm there were layers of old laminated blood clot. A neutrophilic and monocytic infiltrate extended through all coats of the aorta and there were abscesses in the adventitia. Small numbers of paired Gram-positive cocci were seen in appropriately stained sections.

Discussion

Bacterial infections of the aortic wall may be of intravascular or extravascular origin. In those of intravascular origin the source of infection is more commonly the vegetations of bacterial endocarditis, as in cases 2, 4, 5, 6, and 9 of the present series. These lesions commonly are found in the sinuses of Valsalva as a result of the extension of aortic endocarditis to involve the proximal portion of the aorta. Some infections of the aortic wall are of extravascular origin and arise by extension from adjacent lesions such as masses of tuberculous lymph nodes or abscesses. Cases 1 and 10 are illustrative of this mode of pathogenesis, the aortic infection having extended from an adjacent focus of tuberculous lymphadenitis in the former case and from mediastinitis, presumably the result of perforation of the esophagus, in the latter.

Bacterial infections of the aortic wall also occur in cases in which there is neither a demonstrable intravascular nidus of infection, as bacterial endocarditis, nor infection in adjacent structures. In such cases infection may originate in distant foci and be disseminated by the blood stream. Aneurysms in which this pathogenesis is postulated have been termed primary mycotic aneurysms by Crane,[2] who defines this entity as "a lesion developing in the wall of an artery which is not associated with any demonstrable intravascular inflammatory focus, as bacterial endocarditis, or with any inflammatory process in the surrounding tissues." To apply the term primary to aneurysms of this

sort is not to deny them causality, since reasonable sources for blood stream infection can be found in most cases. Of the five primary mycotic aneurysms in the present series, plausible sources for infection were available in four. In case 7, the patient had recently been treated for cellulitis of the foot. In case 8, in which the aneurysm was of gonococcal origin, the history was strongly suggestive that the patient had had gonococcal arthritis. The history of case 11 suggested pneumonia 6 weeks before admission to the hospital and, in case 12, an organizing bronchopneumonia was found at necropsy. Of the 24 cases of primary mycotic aneurysm reported prior to 1945,[6] most of the primary infections were in soft tissue, bone and joints, and in the lung.

Any artery may be involved by bacterial infection in the ways described but the aorta is the more frequently affected,[7] favorite sites being the root and ascending thoracic portions. Next most commonly involved are the abdominal arteries, particularly the superior mesenteric, hepatic, and splenic. In these vessels infection is one of the more common causes of aneurysm. The intracranial arteries, particularly the middle cerebral, and the large vessels of the extremities are the next most frequently involved.

The means by which bacteria gain access to the aortic wall in cases in which there is direct extension of the inflammatory process from an infected aortic valve or from diseased mediastinal structures is obvious. The means by which organisms gain access in cases in which there is no continuity between the involved structures have been discussed by Stengel and Wolferth[7] and by Rappaport[8] and may be summarized as follows: first, that bacteria or infected emboli settle or lodge on the intimal surface of the artery; second, that bacteria or infected emboli lodge in the vasa vasorum. Septic emboli may bring infected material directly in contact with vessel walls in such situations as at the bifurcations of the cerebral arteries, but direct infection of the intact, undiseased intima of a large elastic artery, such as the aorta, must be extremely rare or non-existent.

That bacteria may gain access to the aortic wall by way of the vasa vasorum is illustrated by two reported cases. Owens and Bass[9] reported a case of tuberculous aneurysm of the abdominal aorta in which there was no adjacent tuberculous focus. It was their opinion that the organisms entered the aorta via the vasa vasorum. In a case of bacterial aortitis complicating syphilitic aortitis, reported by Rappaport,[8] there were multiple miliary abscesses in the media and intima and it is likely that, in this case also, the infection was transmitted by the vasa vasorum.

It is significant that, in the present series, except in those cases in which there was a demonstrable intravascular or adjacent focus of infection, underlying disease was found in the aorta in all cases. Congenital hypoplasia of the aorta was noted in case 7. In discussing the high incidence of bacterial infection and mycotic aneurysm in the adult type of coarctation of the aorta, Abbott[10] has pointed out that the dilated and atheromatous aorta below the stenosis, and the kinks and deformities of the constriction provide a favorable nidus for the lodgment of bacteria which might otherwise be quite avirulent. Thus the vicinity of a coarctation, like other congenital defects, provides a definite *locus minoris resistentiae.*

Rappaport[8] also has mentioned that the ulcerated, atherosclerotic aortic intima may provide a suitable site for bacterial infection. Syphilitic aortitis was a complicating factor in case 8 of this series. It is of interest to note that, in an early case of mycotic aneurysm described by Osler,[1] the lesion was engrafted on a syphilitic aorta. In a case of mycotic aneurysm of the abdominal aorta with dissection, reported by Lippincott,[11] syphilitic aortitis was believed to have been present also. It is reasonable to suppose that the wrinkled plaques and increased medial vascularization of the syphilitic aorta may increase the hazard of bacterial infection. A case of suppurative aortitis with cystic medionecrosis, dissection, and perforation of the aorta has been reported by Williams,[12] and it is possible that medial degeneration may somehow facilitate the establishment of infection in the aortic wall. Medial dissection has been recorded occasionally in cases of bacterial aortitis or mycotic aneurysm. Dissection was noted in cases 3 and 9 of the present series and also in Lippincott's case. In neither of the last 2 cases was medial degeneration or cystic necrosis noted, but it was present in case 3 of the present series.

Summary

Twelve cases in which there was bacterial infection of the aortic wall have been presented. In 9 cases there was formation of a mycotic aneurysm. In 7 cases the source of infection was bacterial endocarditis or disease in adjacent mediastinal structures. In 5 cases, the source of infection was remote (primary mycotic aneurysms). These aneurysms represent an incidence of 2.6 per cent of 338 aneurysms occurring in 23,000 necropsies. All of the aneurysms were thoracic in location. In no case had the diagnosis been made before death.

Underlying aortic disease was found in many cases. These processes included atherosclerosis, cystic medionecrosis, congenital hypoplasia,

and syphilitic aortitis. This observation suggests that pre-existing disease of the aorta favors the establishment of infection in it.

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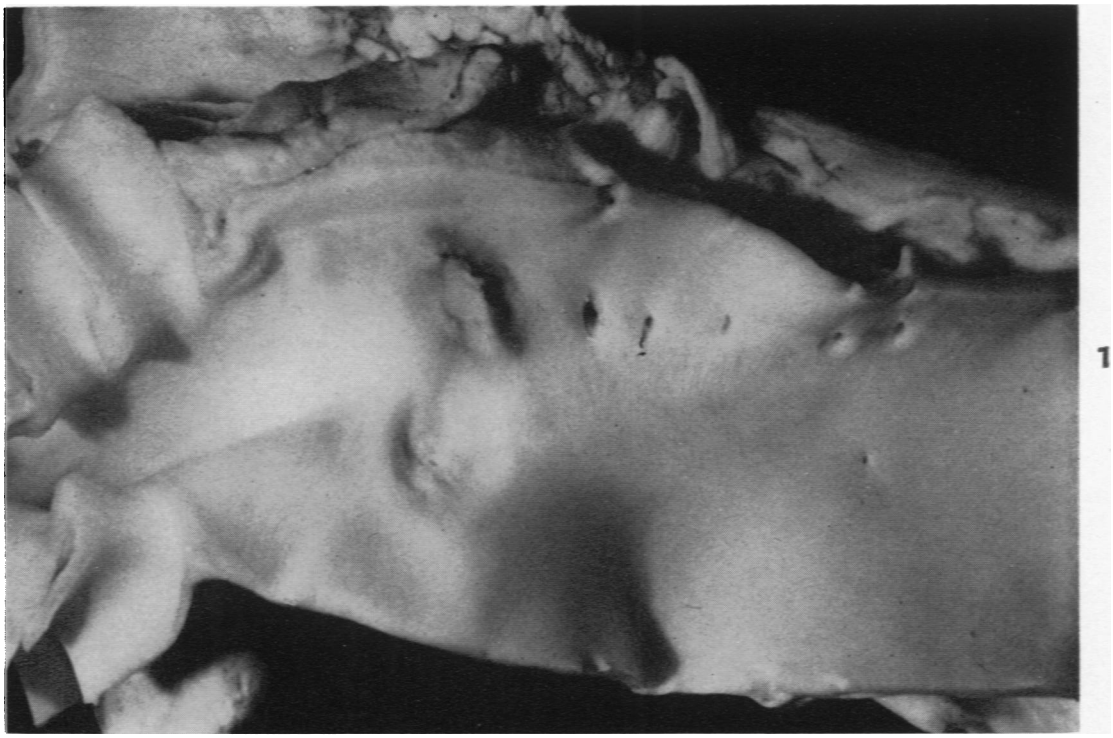
LEGENDS FOR FIGURES

FIG. 1. Case 10. Slit lesion in aorta below origin of left subclavian artery, communicating with esophagus.

FIG. 2. Case 11. Primary mycotic aneurysm of descending thoracic aorta. Rupture into right pleural cavity.



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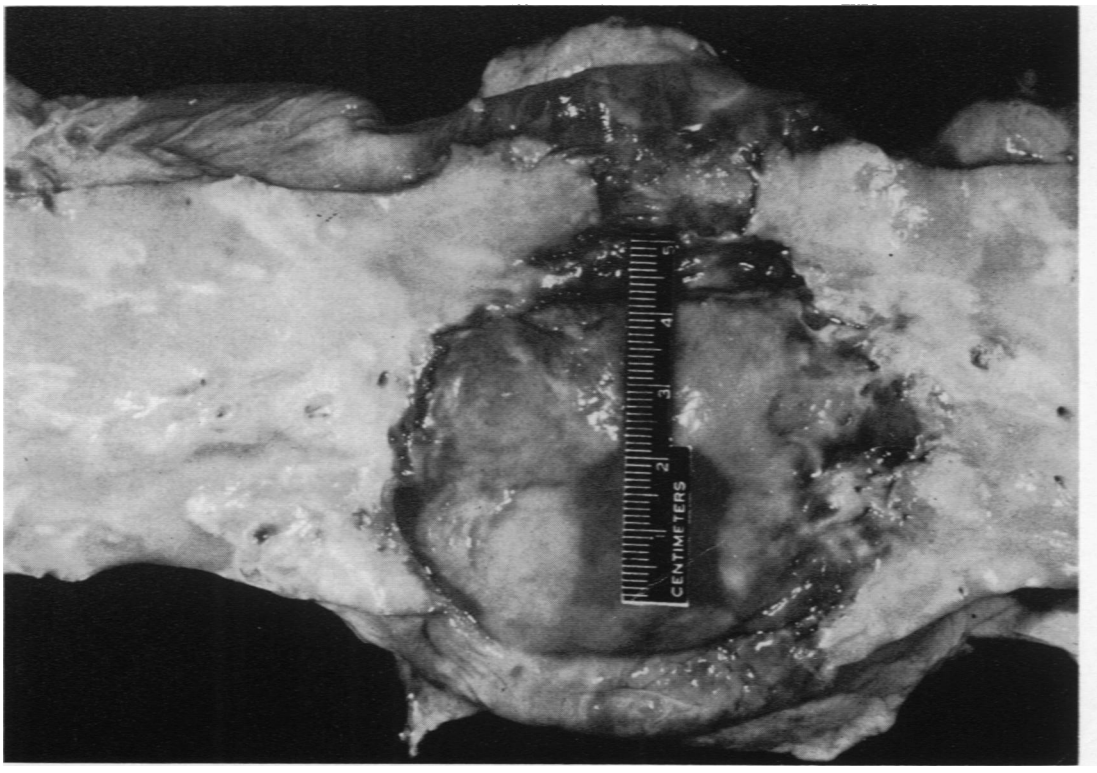

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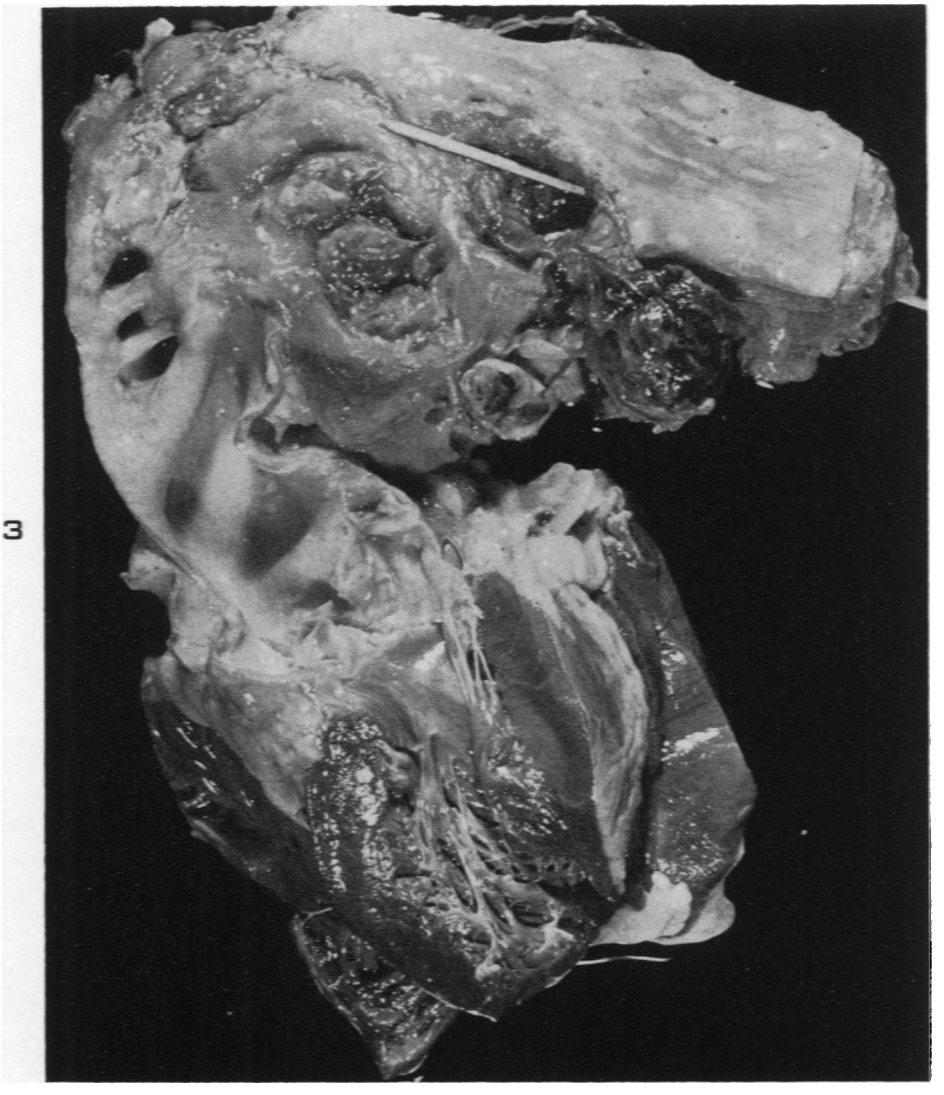
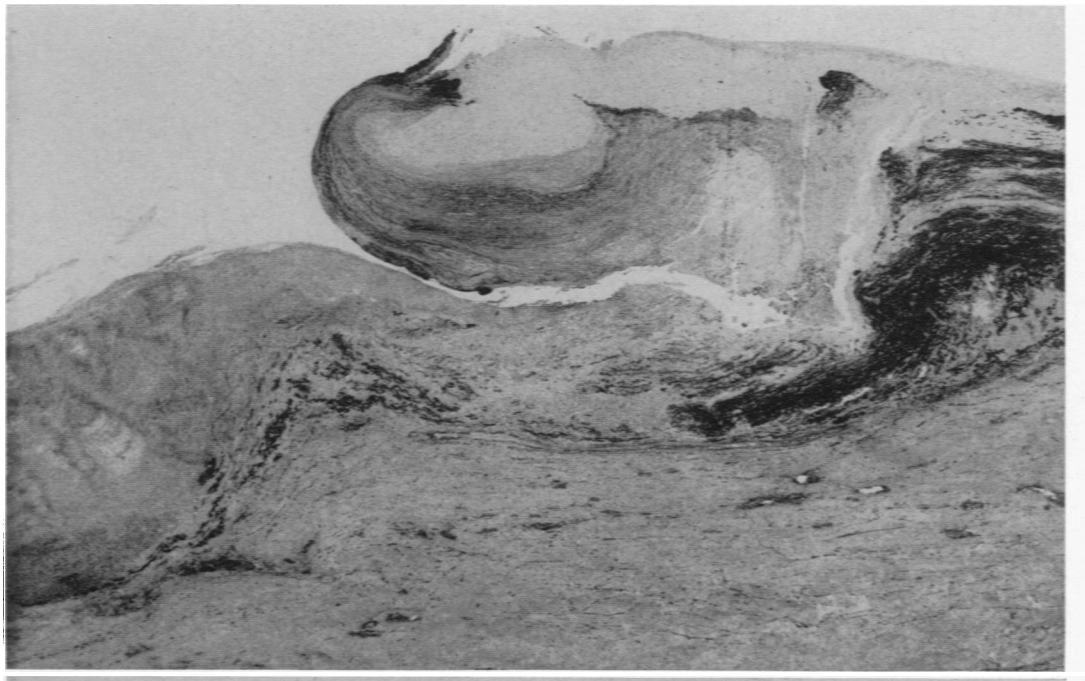


FIG. 3. Case 12. Primary mycotic aneurysm of descending thoracic aorta. Rupture into posterior mediastinum with compression of esophagus.

FIG. 4. Case 12. Edge of aneurysm, showing destruction of elastic fibers. Verhoeff-van Gieson's stain. × 46.

FIG. 5. Case 11. Neutrophilic infiltration with abscess formation in intima. Phloxine-methylene blue stain. × 32.



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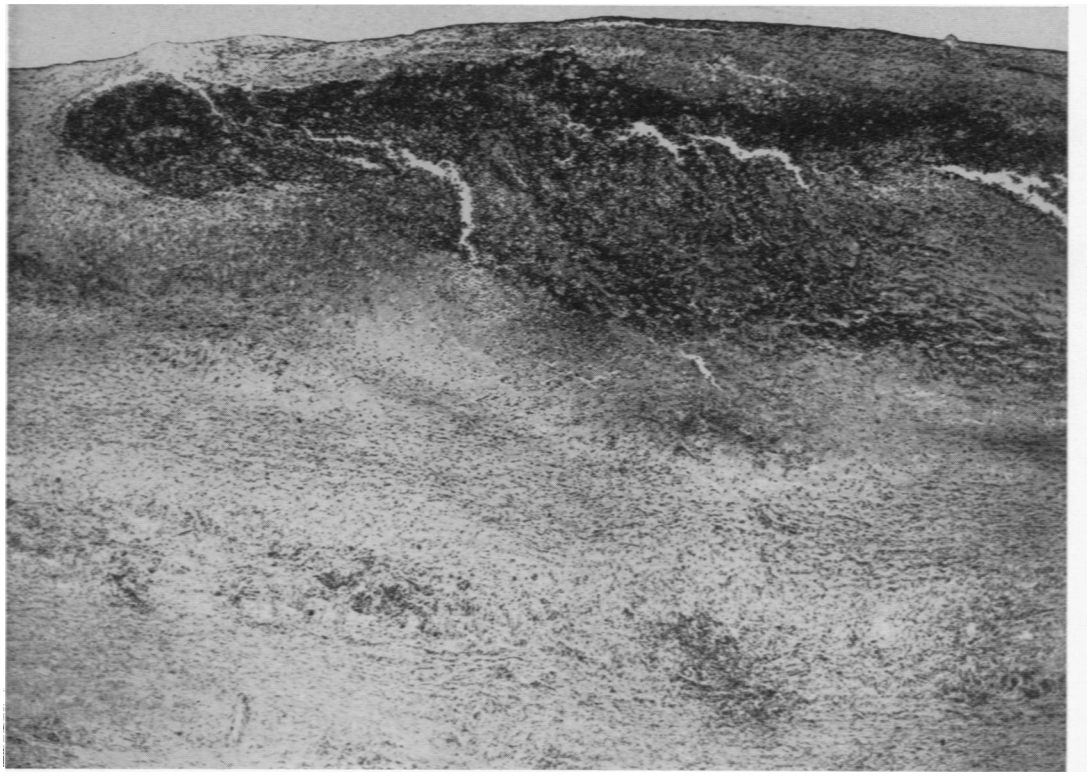

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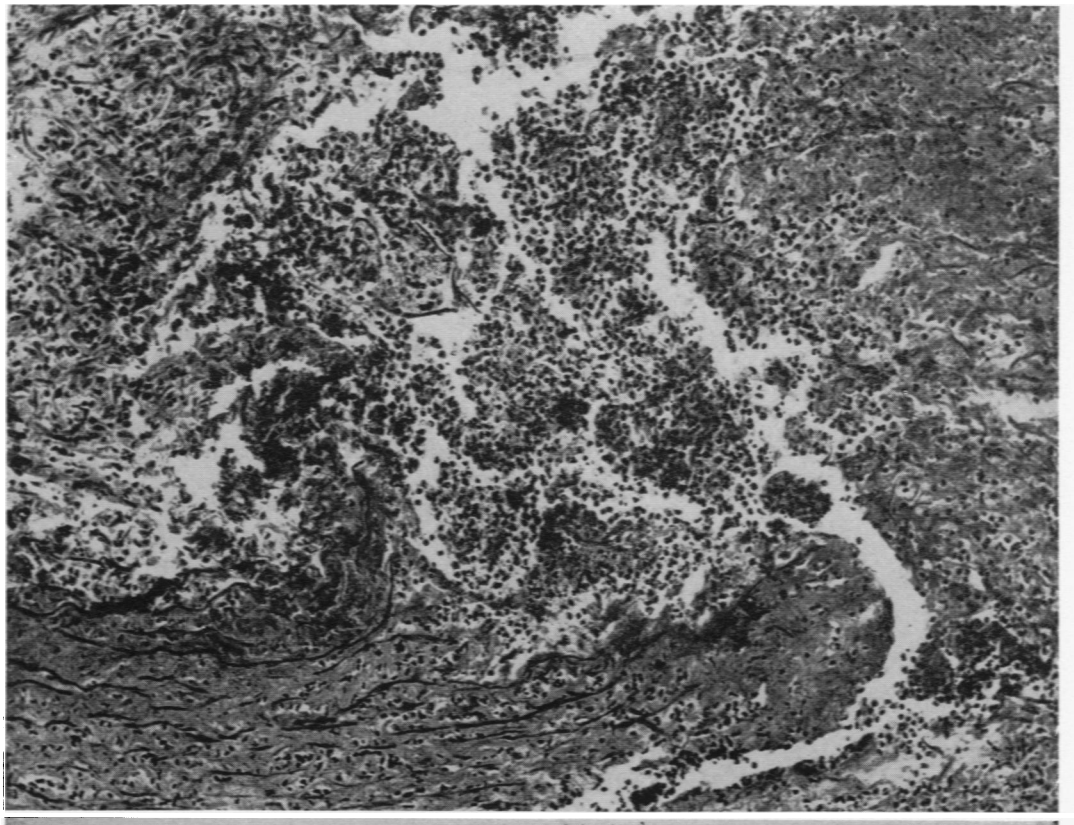
FIG. 6. Case 2. High-powered view of medial abscess, showing fragmentation of elastic fibers. Phloxine-methylene blue stain. × 740.

FIG. 7. Case 3. Portion of uninvolved aorta, showing areas of cystic medionecrosis. Phloxine-methylene blue stain. × 74.



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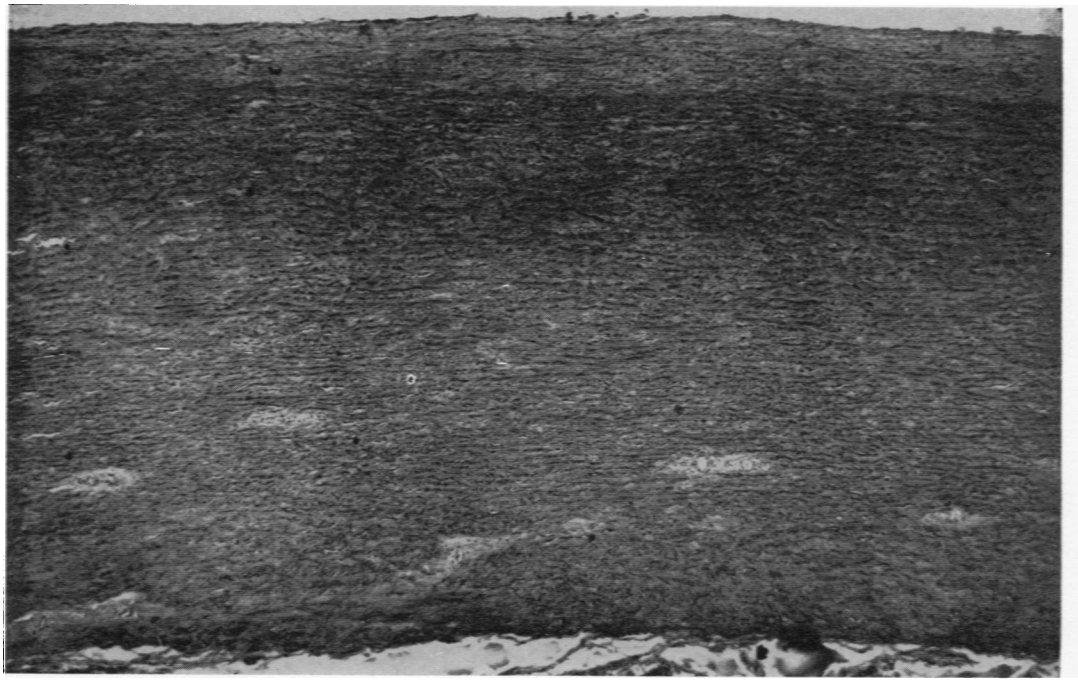

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