Infected Aortic Aneurysm after Intraabdominal Abscess

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Infected abdominal aortic aneurysm is rarely associated with an antecedent history of a localized suppurative process. This report describes a case in which an infected aortic aneurysm occurred after surgical treatment of an appendiceal abscess. Diagnosis and management of infected aneurysms are discussed, together with a review of the literature. ***(Texas Heart Institute Journal 1987; 14:208-214)***

Key words: Aneurysm, infected; aorta, abdominal; abscess, appendiceal

ALTHOUGH THE AORTA and other large arteries are highly resistant to infection,[1-3] there are a number of conditions (e.g., arteriosclerosis, vascular anomalies, and cystic medial necrosis) in which this resistance is impaired. Under these circumstances, infectious aneurysmal dilatation of the aorta may occur.[4-7] Previous aortic grafting, renal transplantation, and intraabdominal abscesses have also been implicated in the development of infected abdominal aortic aneurysms.[8-11] Before use of antibiotics became widespread, these lesions were most often associated with bacterial endocarditis[12,13]; recently, however, their epidemiology has changed: Most cases involve either a preexistent arteriosclerotic aneurysm onto which infection is superimposed or a *de novo* infected aortic aneurysm produced by bacteremia from an extravascular septic focus.[3,14,15] Rarely do aneurysms of the abdominal aorta result from a coexistent or an antecedent localized suppurative process.[11] The contiguous spread of a septic process is often cited as one of the pathogenic mechanisms that produce an infected abdominal aortic aneurysm,[3,14] but there has been only one previous report of such a lesion arising in this manner.[11] In the following case, an infected abdominal aortic aneurysm resulted from the contiguous spread of an intraabdominal abscess. The literature is reviewed, and the pathogenesis of infected abdominal aortic aneurysms is discussed.

CASE REPORT

A 50-year-old woman presented with a 1-week history of nausea, vomiting, and abdominal pain associated with multiple febrile episodes. On admission, her temperature was 39° C and abdominal examination elicited diffuse abdominal tenderness and guarding. A hematologic profile revealed leukocytosis of 23,000/cu mm, with a leftward shift. The patient was taken to the operating room, where, after the induction of general anesthesia, a large mobile mass was easily palpable in the right iliac fossa. At abdominal exploration, a perforated appendiceal stump with an associated abscess was found. A right hemicolectomy, with an end-ileostomy and creation of a colocutaneous mucous fistula, was performed without incident. No abnormality of the abdominal aorta was

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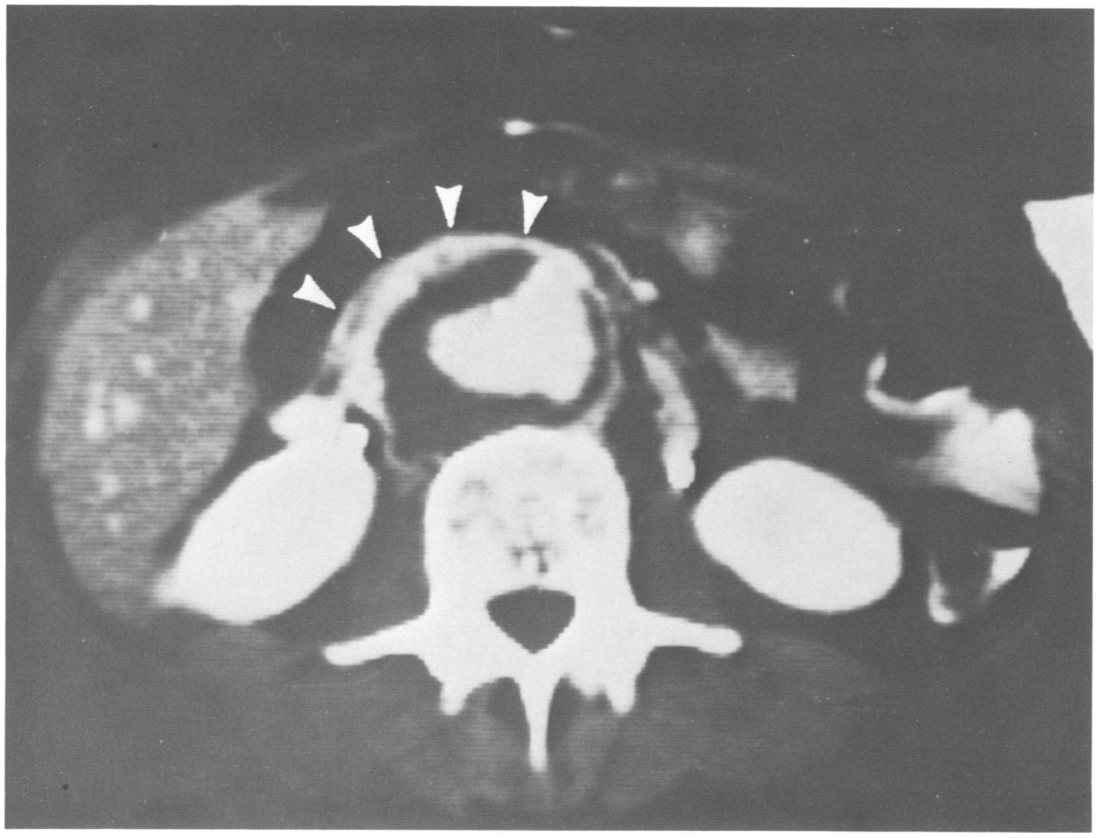


Fig. 1 Computerized axial tomographic scan showing an infrarenal abdominal aortic aneurysm, with discontinuity of the posterior aspect of the aneurysmal wall indicating rupture and leakage.

noted. Preoperative blood cultures and intraoperative cultures of the abscess site yielded *Escherichia coli*. After receiving a 10-day course of intravenous antibiotics (tobramycin and clindamycin), the patient was discharged from the hospital in good condition.

Approximately 4 weeks later, she was readmitted for elective ileotransverse reanastomosis, and bowel preparation was commenced. Her only complaint at this time was persistent low back pain, but her temperature was 38.6° C, and she had a leukocytosis of 21,000/cu mm. Computerized axial tomography of the abdomen revealed localized dilatation of the infrarenal abdominal aorta (Fig. 1). Digital subtraction arteriography confirmed the presence of a localized saccular aortic aneurysm but showed no evidence of arteriosclerosis of the aortoiliac system (Fig. 2). Antibiotic therapy was commenced, and the patient was prepared for surgery. Extraanatomic left axillobifemoral bypass grafting was performed according to the method described by Louw and others.[16-18] After arterial reconstruction had been completed, the abdomen was reexplored. Multiple adhesions of the small intestine were lysed, and the small bowel and mesentery were fully mobilized to expose an infrarenal abdominal aortic aneurysm that measured 10 cm in diameter. There was no purulent collection or active hemorrhage associated with the lesion, but a small hematoma on its posterior aspect indicated adventitial disruption. The aneurysm extended from just below the level of the renal arteries to the bifurcation of the aorta. Proximal control of the aorta was obtained at the diaphragmatic hiatus, and distal control was established by isolating the common iliac arteries. The aneurysm was resected without compromising the renal arterial inflow. The proximal aortic stump was then closed in two layers with interrupted polypropylene sutures. The common iliac arteries were closed in a similar fashion, and ileotransverse anastomosis was performed

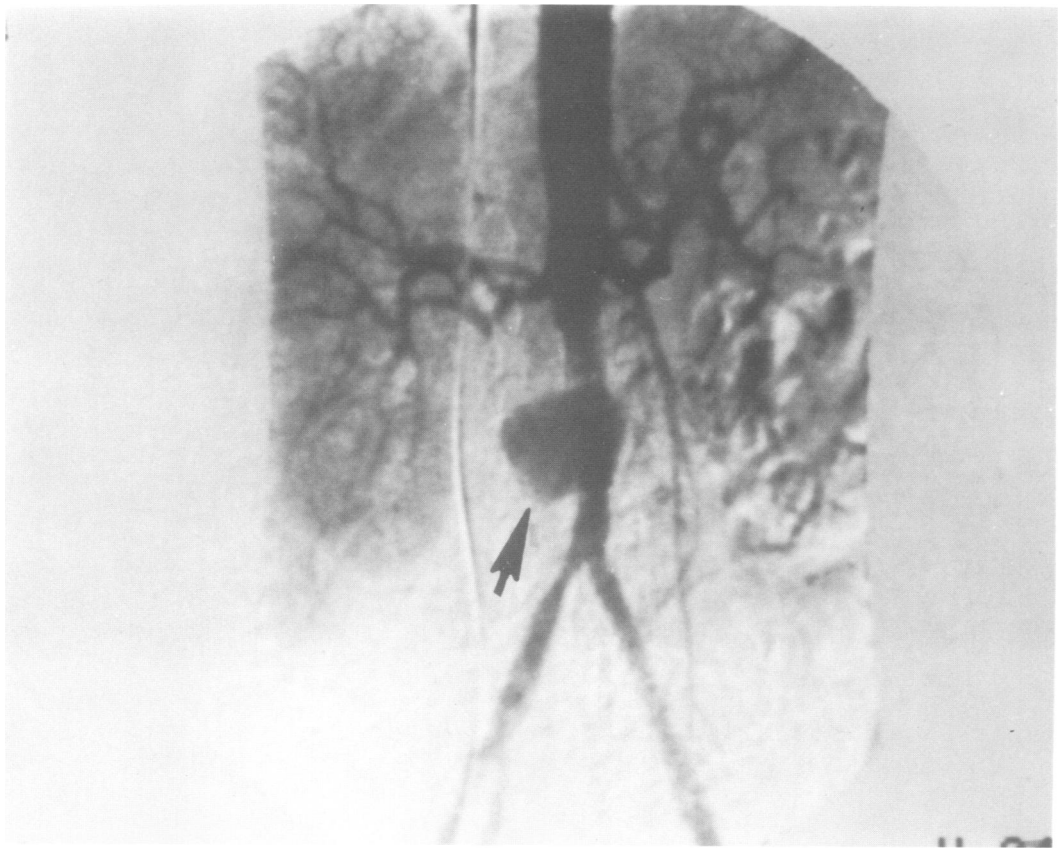


Fig. 2 Digital subtraction arteriogram, clearly showing an infrarenal abdominal aortic aneurysm with a noticeable absence of arteriosclerosis in the aortoiliac system.

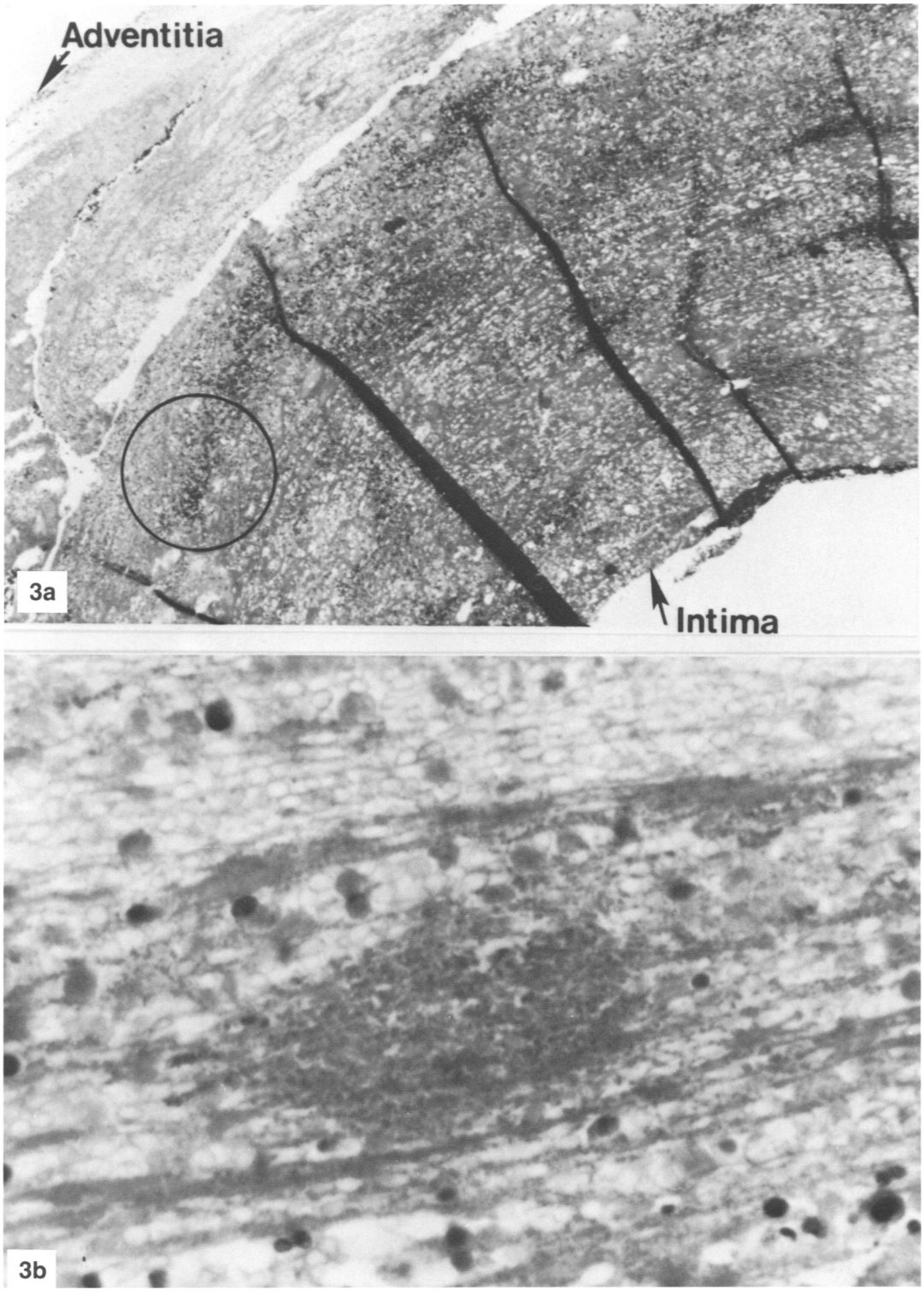
in two layers. A closed drainage system was used to prevent the formation of an infected hematoma in the aortic bed.

An intraoperative gram stain revealed gram-negative rods, and subsequent cultures of the aneurysmal wall grew *E. coli*. Pathologic examination showed microabscess formation in the periaortic tissues, as well as in the adventitia and the media of the aortic wall, and diffuse inflammation of all layers of the aneurysmal wall (Fig. 3). Postoperatively, the patient was treated with intravenous antibiotics for 6 weeks and remained afebrile. Follow-up digital subtraction arteriography showed patent arterial grafts and an intact suture line in the proximal aortic stump (Fig. 4).

The patient did well for approximately 12 weeks postoperatively but then experienced sudden abdominal pain and distension; shortly thereafter, she died. No postmortem examination was performed, but we assume that suture-line dehiscence led to exsangination.

DISCUSSION

Infected abdominal aortic aneurysms are far less common than arteriosclerotic aneurysms but are among the earliest arterial lesions described in the medical literature.[19] Ambrose Paré is credited with the first description of a syphilitic aortic aneurysm, in the sixteenth century. In 1885, Sir William Osler[12] coined the term "mycotic aneurysm" to describe arterial lesions caused by septic emboli from a condition originally described as "malignant endocarditis." Stengel and Wolforth[13] reported 217 "mycotic" aneurysms and showed that the aorta, which was involved in 30% of the cases, was the most frequently affected structure. They also noted that the word "mycotic" was imprecise, because such lesions included bacterially infected aneurysms as well as those produced by ". . . a special group of infections that have come to be called the mycoses. . . ." The term "primary mycotic aneurysm" was previously



Figs. 3a and **3b** Photomicrograph of the aortic wall after resection. Diffuse, acute inflammation is present in the adventitia and media, and clusters of bacterial organisms extend from the adventitia into the media. (3b is an enlargement of the circled area in 3a.)

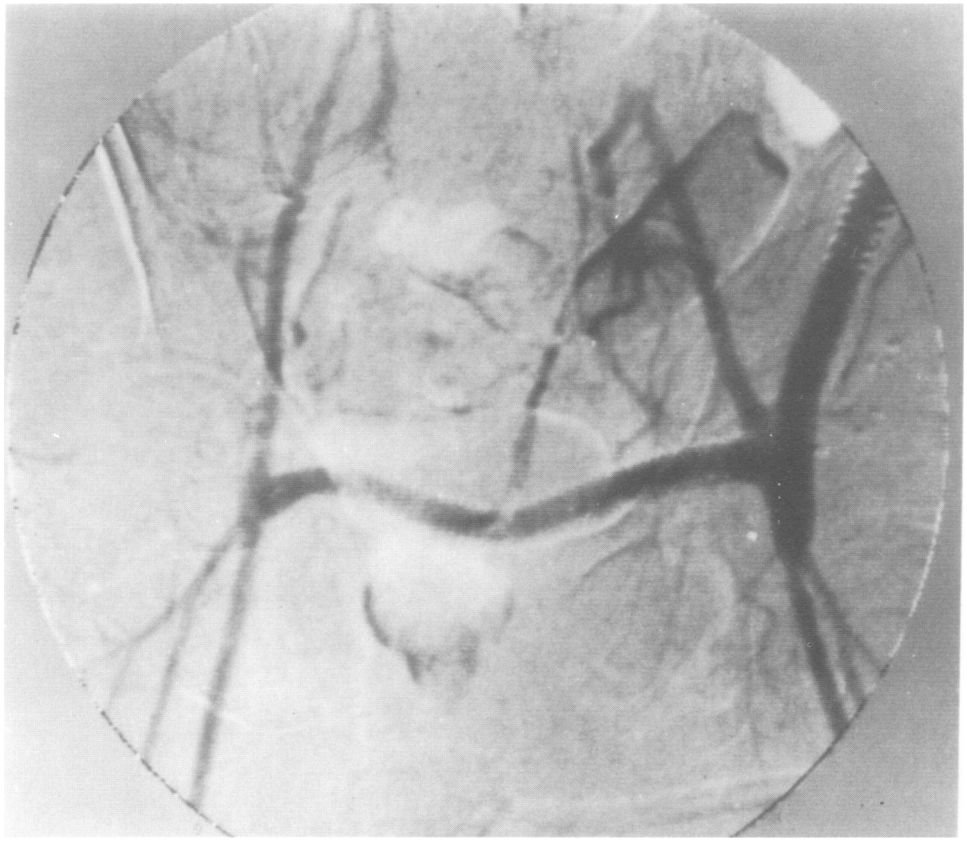


Fig. 4 Postoperative digital subtraction arteriogram showing patency of the extraanatomic arterial grafts and integrity of the proximal aortic stump, with excellent blood flow to both lower extremities.

used to described lesions without an intravascular focus of infection or any inflammatory process in the surrounding tissues.[19] The term "mycotic aneurysm" is now reserved for infections caused by fungi, and "infected aneurysm" is used to denote bacterial seeding of an arteriosclerotic aneurysm. Some authors, however, still consider aneurysms caused by septic emboli from bacterial endocarditis to be "mycotic" aneurysms, as originally described by Osler.

Although the nomenclature is somewhat misleading, the pathogenesis of infected abdominal aortic aneurysms has been understood for several years.[3,13,14] The most frequent cause is seeding of preexisting arteriosclerotic abdominal aortic aneurysms arising from bacteremia associated with an extravascular septic focus.[15] In many cases, however, the source of the bacteremia is never determined.[3] In a recent series, the *Salmonella* genus accounted for the largest percentage (66%) of infected abdominal aortic aneurysms; staphylococci and other gram-positive organisms were less common.[3,20] Fungi actually account for very few mycotic aneurysms.[14,21-23]

An infected aneurysm can arise via direct extension of an adjacent suppurative process into the aorta or via extension through the lymphatic system. Although any artery can be involved, infections adjacent to the thoracic and abdominal aorta are most frequently reported.[21,24,25] The contiguous infectious processes that most commonly yield aneurysmal dilatation of the aorta are tuberculosis and actinomycosis.[14,26] A review of the literature reveals only one previous case[11] in which a suppurative process produced an infected abdominal aortic aneurysm. In our case, there was both bacteriologic and pathologic documentation of a suppurative process that invaded the aortic adventitia, as well as arteriographic evidence of aneurysmal dilatation in the absence of arteriosclerosis of the

aortoiliac system.

Classically, diagnosis of an infected aneurysm is based on clinical symptoms and a high index of suspicion.[14] Arteriography is most often used to confirm the diagnosis, but computerized axial tomography and ultrasonography have recently been shown to be effective in detecting the presence of an aneurysm. In this case, digital subtraction arteriography was used. This is a relatively new diagnostic modality in which small amounts of contrast media are injected into the central venous circulation; by means of computer detection, augmentation, and imaging, virtually all the major arterial and venous structures can be visualized. Moreover, arterial puncture and catheterization can be avoided, thus eliminating the risk of disrupting an infected or false abdominal aortic aneurysm.[20]

Mendelowitz[27] described seven patients with *Salmonella* infections of abdominal aortic aneurysms. All underwent resection of the aneurysm and local reconstruction with prosthetic grafts, but there were no long-term survivors. Bennett and Lewis reported a series of 17 patients with infected abdominal aortic aneurysms.[20] Six of these, who did not undergo surgery, died within 12 months. Of the remaining eleven patients, five died within the first 10 postoperative days, and the other six died during the first 2 months, of hemorrhage from anastomotic disruption or graft sepsis. Although no studies are available comparing anatomic to extraanatomic aortic reconstruction for infected aneurysms, a recent review of five cases has shown a long-term survival rate of 60% after axillobifemoral bypass grafting.[11,14,28,29]

The prognosis may be related to the microbiology of the infection. Jarrett[14] described ten abdominal aortic aneurysms that were infected with gram-positive organisms; only one of these lesions ruptured. In contrast, five of six aneurysms infected with gram-negative organisms ruptured. The mortality rate for gram-positive infections was 50%, compared to 84% for gram-negative infections.

Current treatment of infected abdominal aortic aneurysms has evolved from experience with infections complicating vascular reconstructive surgery.[8] The main goals are complete excision of the infected aneurysm, restoration of blood flow to the extremities through uninfected tissue planes, and long-term organism-specific antibiotic therapy.[18,28,29] Surgical treatment may consist of a two-stage procedure including (1) excision of the aneurysm and placement of a temporary tube or bifurcation graft and (2) definitive extraanatomic grafting 3 to 6 months later[30,31]; alternatively, a one-stage procedure may be carried out, as in our case.[11,18,29]

Despite the development of extraanatomic bypass grafting, the prognosis for patients with an infected abdominal aortic aneurysm is extremely poor. The inability to sterilize the aortic bed and its surrounding tissues with surgical drainage and antibiotic therapy is the most important factor in the low survival rate. Quick, accurate diagnosis before rupture, resection of all infected material, extraanatomic bypass, and appropriate antibiotic therapy are the cornerstones of successful management.

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