Infected Aortic Aneurysms

A Changing Entity

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Infected (mycotic) aortic aneurysms are infrequent and, without surgical intervention, usually lead to uncontrolled sepsis or catastrophic hemorrhage. Symptoms are frequently absent or nonspecific during the early stages, and a high index of suspicion is essential to make the diagnosis. Surgery performed after rupture carries high morbidity and mortality rates. Bacterial endocarditis with streptococcus pyogenes was the most common cause of infected aortic aneurysm in the pre-antibiotic era. Today, arterial trauma due to iatrogenic manipulation and depressed immunocompetence have become more common risk factors. *Staphylococcus aureus* and *Salmonella* are the most frequent bacteria identified. The authors' recent experience in six patients with infected aortic aneurysms who underwent arteriography and computed tomography was reviewed and these diagnostic methods compared. Computed tomography was found to be more sensitive in the diagnosis of the early stages of the disease, allowing for follow-up by serial scans in a noninvasive and less costly manner. Successful treatment, in four of these patients, was accomplished by aneurysmal resection and extra-anatomic bypass or *in situ* prosthetic reconstruction. A higher clinical awareness of this disease, leading to early computed tomography evaluation and prompt surgical intervention under appropriate and intensive antibiotic therapy, appears to offer the best chance of survival in patients with this difficult condition.

A CENTURY HAS passed since the classic report of Sir William Osler[1] to the Royal College of Physicians in the landmark Gulstonian Lecture in London in 1885 on "malignant endocarditis" in which he described the pathophysiology and introduced the term of mycotic aneurysms. He evaluated the relationship between the presence of aortic valve vegetations in a single patient and four aneurysms of the aortic arch that had the "appearance of fresh fungus vegetations" on their internal surface. The patient was a 30-year old man admitted with chills, fever, and pneumonia who developed bacterial endocarditis and eventually died of aneurysmal rupture. Osler deduced that embolization of these organisms to a peripheral vessel led to septic degeneration of the arterial wall, "mycotic aneurysm" formation and rupture. The term "mycotic aneurysm" has since been applied to arterial infections of all types, although today it is generally used more restrictively for fungal infections and, hence, the designation of infected aneurysms (IAA) is the most appropriate, as suggested by Jarrett et al.[2] Earlier reports[3] in 1844 had described the presence of abscesses in arterial walls caused by infected emboli, and Koch in 1851[4] and Tufnell in 1853[5] recorded single case reports of peripheral aneurysms occurring in young patients, in their early 20s, during treatment for endocarditis.

Most IAA in the earlier reports were associated with valvular infectious disease. This was found in 187 of the 217 cases (86%) assembled by Stengel and Wolferth in 1923.[6] The aorta was the most frequently involved artery, being affected in 66 of 217 cases. In the pre-antibiotic era, bacterial endocarditis and its complications were uniformly fatal. The development of vascular grafts and antibiotic therapy lead to the first reported survival of patients with an IAA in 1962 by Sower and Whelan[7] and Morris et al.,[8] but even as late as the 1960s and early 1970s most of the patients with this disease process died.

Newer microbiologic techniques for identifying the infecting organism and appropriate antibiotic therapy combined with successful replacement of infected valves have markedly reduced the incidence of embolomycotic aneurysms of the aorta in the United States. Occasional reports of septic emboli from bacterial endocarditis still appear in the literature, particularly in children.[9,10] This is in sharp

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contrast with the reports from the 1930s,[11] when an IAA "in the absence of valvular disease was very unusual." The term "primary mycotic aneurysm" was introduced by Crane,[11] applying it to those aneurysms occurring in the absence of overt sepsis.

In the postantibiotic era, about 80% of the infections of the aorta appear to result from microbial arteritis.[12] An increasing number of IAAs appearing because of the implantation of circulating bacteria in a diseased, atherosclerotic, or traumatized aortic intima, with wall separation and aneurysm formation have been reported in recent years. These vessels are usually already involved by acquired diseases or congenital anomalies. Another mechanism of infection is due to direct extension or through lymphatic vessels from an adjacent infectious process. Infection in a pre-existing aneurysm also can occur and is estimated to occur in 3% of aneurysms.

Several risk factors are more common now than in the past. These include diagnostic or therapeutic arterial catheterization, illicit intravascular drug use, the higher prevalence of patients with depressed immune competence secondary to either chronic or neoplastic diseases, and, since 1981, the rising number of patients with acquired immune deficiency syndrome.

Changes in the diagnosis, causes, risk factors, natural history, and treatment of IAA prompted us to review our recent surgical experience.

Case Reports

During the period between 1986 and 1991, six patients were evaluated and underwent resection of IAAs. The clinical and diagnostic findings as well as the surgical procedures and outcome are summarized in Table 1. The three men and three women were 61 to 75 years of age (mean, 68.7). Two of the aneurysms involved the descending thoracic aorta, and both patients complained of prominent chest pain. The remaining four involved the abdominal aorta, three located in the infrarenal segment and one supraceliac. Abdominal, flank, or back pain were present in all instances.

Predisposing conditions included diabetes mellitus in three patients, atherosclerosis in four patients, and rheumatoid arthritis and carcinoma of the bladder in individual cases.

All patients had evidence of sepsis with fever, leukocytosis, and, in three of them, positive blood cultures (two salmonella species and one *Staphylococcus aureus*). Salmonella was also identified by a computed tomography (CT)-guided needle biopsy of a para-aortic mass in another patient. These organisms were confirmed also by cultures of the aneurysm wall at the time of the resection; in the remaining two patients, *Listeria monocytogenes* and a Staphylococcus species were the infecting organisms isolated from the wall of the aneurysm. Both of these patients had undergone prior antibiotic therapy. Pre-existing sepsis was documented in one patient with urinary tract infection, another with colonic diverticulitis, and in two with Salmonella enterocolitis.

In no instance was the aneurysm diagnosed on a clinical basis alone. A plain lateral chest radiograph suggested the diagnosis of a descending thoracic aneurysm that was confirmed by CT scanning and angiography in one patient. The other thoracic aneurysm was known to be present for the past 5 years from the time of an exclusion procedure performed in another hospital for an acute dissecting aneurysm with ligation and division of the aorta proximal to the origin of the left subclavian artery

TABLE 1. *Summary of Findings*

Age	Sex	Location of Aneurysm	Associated Conditions	Symptoms/Signs	Organism	Angiograph	Computed Tomography	Surgery	Outcome
72 (1986)	M	Descending thoracic	Atherosclerosis, rheumatoid arthritis	Fever, leukocytosis, recurrent chest pain	*S. pneumonia*	Saccular TAA (3 cm)	Periaortic mass saccular TAA	*In situ* graft	Alive at 5 yr
75 (1986)	M	Supraceliac	Atherosclerosis, diverticulosis	Fever, leukocytosis, abdominal pain, diarrhea	*L. monocytogenes*	Saccular AAA (11 × 8 cm)	Saccular AAA (4.5 cm) 10 days, saccular AAA (8 cm)	*In situ* graft	Alive 2.5 yr
66 (1988)	M	Infrarenal	Atherosclerosis, diabetes, cancer of the bladder	Fever, leukocytosis, flank pain, urinary infection	*S. aureus*	Not done	Periaortic mass, saccular AAA	Drainage abscess, Ax-fem, fem-fem	Died (6 wk), sepsis
70 (1989)	M	Infrarenal	Atherosclerosis, salmonellosis	Fever, leukocytosis, back pain	Salmonella	Refused	Saccular AAA (2 cm) 3 mo, saccular AAA (5.5 cm)	Ax-fem, fem-fem	Died (4 mo), sepsis
68 (1991)	F	Distal abdominal	Diabetes	Fever, leukocytosis, lower abdominal pain	Salmonella	Bilobar saccular AAA	Periaortic mass	Ax-fem, fem-fem	Alive
61 (1991)	F	Descending thoracic	Diabetes, salmonellosis	Fever, leukocytosis, chest pain	Salmonella	Excluded TAA, patent subclavian artery	Perianeurysmal mass	Resection TAA	Alive

and at the distal thoracic level with an ascending aorta to abdominal aorta bypass. The plain film showed a large mediastinal mass that appeared unchanged over the period of observation.

Saccular aneurysms were visualized in three of the patients angiographically (Figs. 1 and 2); in another the left subclavian artery and its origin were seen arising from the excluded thoracic aneurysm. Angiography was refused by one patient and was not performed in the remaining patient because he was seriously ill and underwent surgery based on the CT findings alone.

The CT findings were diagnostic in all six cases. A periaortic soft tissue mass with rim enhancement was seen in three patients; one around a calcified aorta of normal size (Fig. 3A), another around a 3-cm saccular aneurysm, and the last around a large thrombosed, descending aortic aneurysm. Expansion of the soft tissue mass 2 weeks later in the first patient prompted a CT-guided needle aspiration (Fig. 3B). Saccular aneurysms were visualized in the three remaining patients, two of which increased significantly in diameter during the period of observation, from 4.5 to 8 cm over a 10-day interval in one patient and from 2 to 5.5 cm in a 3-month period in another (Figs. 4A and B). Aortic calcification was clearly visible on CTs in four patients in this series.

All patients were treated with the appropriate antibiotics and underwent aneurysmal resection with debridement of the periaortic infected soft tissue. A large defect in the aortic wall was present in two cases, and one of these patients had a periaortic abscess that was drained posteriorly. Extra-anatomic bypasses (axillary–bifemoral) were performed in three patients and interposition grafts in two. The left subclavian artery was ligated at its origin with the resection of the descending thoracic aneurysm in the remaining patient; a graft from the left carotid to the left subclavian artery had been constructed at the time of the original aorto-aorto bypass and aneurysmal exclusion and was patent.

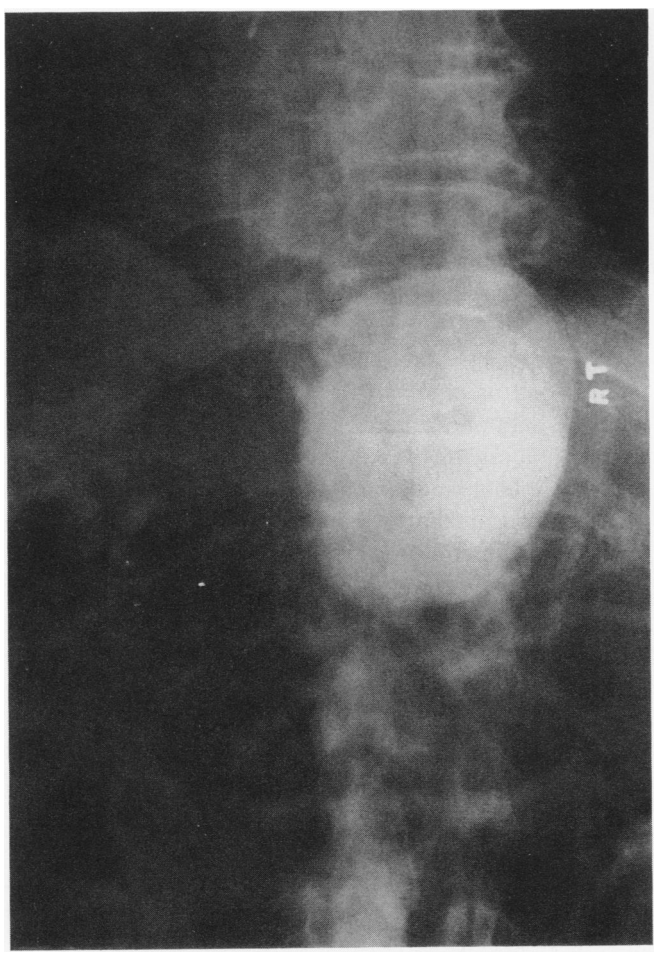

FIG. 1. Anteroposterior projection of retrograde brachial aortogram showing a large, saccular, 8-cm, supraceliac aortic aneurysm (case 2).

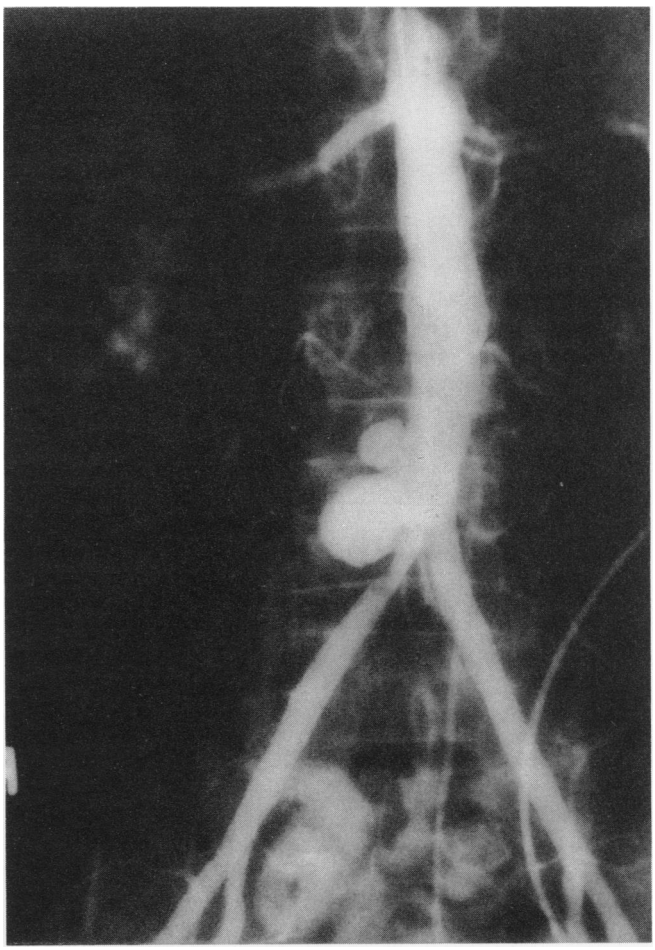

FIG. 2. Transfemoral arteriogram shows atypical aortic aneurysms originating at the distal abdominal aorta (case 5).

The overall mortality rate was 33.3% (two of six patients), one 6 weeks and the other 4 months after surgery because of generalized sepsis. Both of these patients had extensive periaortic infection, with necrosis of the aortic wall and preoperative septicemia with positive cultures of the urine and stool. Extra-anatomic bypass grafts were the operative procedures in these two patients: the inability to debride all infected tissue also may have contributed to the late postoperative deaths in spite of the initial successful surgical results.

Discussion

Infected aortic aneurysms are uncommon but not rare as our experience of six cases in a 5-year period in one institution suggests. Other authors[13] have also identified an increasing number of these aneurysms in recent years, suggesting that its incidence may be increasing.

The nature of IAAs is changing. Bacterial endocarditis was not the cause of the infection in any of our six patients.

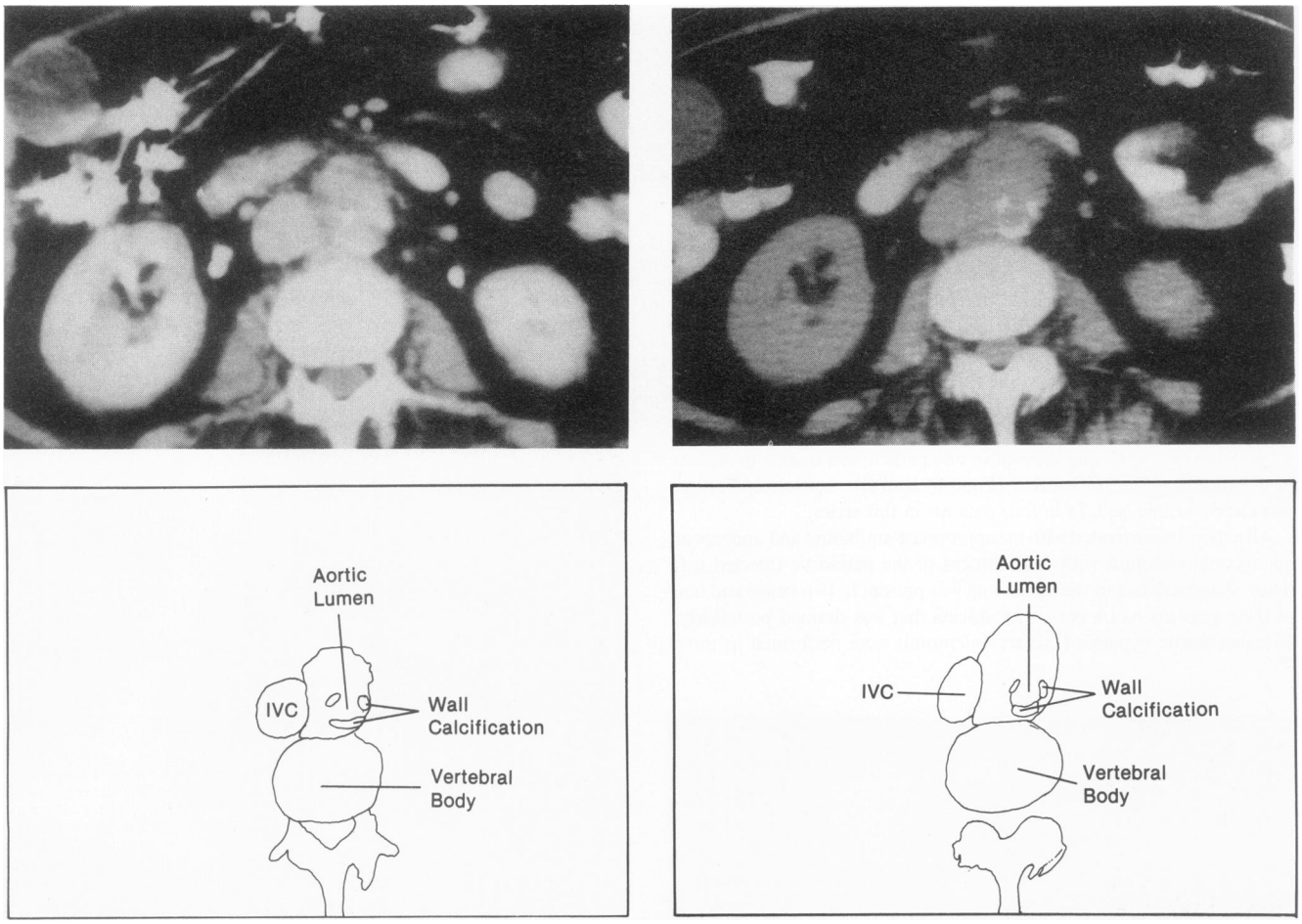

FIG. 3. (A, left) CT scan shows a calcified infrarenal aorta of normal size with an irregular, anterior, soft tissue mass inseparable from the aorta. (B, right) Repeat CT scan 1 week later shows significant enlargement of the soft tissue mass (case 5).

In our review of 19 reports in the English literature involving 28 patients with IAAs (from 1983 to 1991), the findings were similar except for three pediatric patients, in whom embolomycotic aneurysm of the abdominal aorta developed.[9,14] With few exceptions, all of the reviewed reports were of single cases or of small series. In a larger 15-year cumulative review (1968 through 1982), Johansen and Devin[15] found bacterial endocarditis to be a risk factor in only 17% of 220 patients, although they emphasized that most of this subgroup of patients appeared in reports from the British Commonwealth.

The mean age of our patients (68.7 years) is slightly higher than in our reviewed group (mean, 61.2 years), if the three pediatric cases are excluded. This trend toward older age is apparent when earlier series of IAAs are reviewed such as in a collected series by Wilson et al.[16] from the 1960s, in which the mean age for aneurysm secondary to bacterial endocarditis was 26 years.

The *bacteriology* of these infections appears also to have changed. In the pre-antibiotic era, the predominant organisms were *Streptococcus pyogenes, S. pneumoniae,* and staphylococci,[6] whereas in the postantibiotic era the dominant infecting organisms are the *Staphylococcus aureus* and the Salmonella species,[17] accounting for about 40% of the total organisms cultured. Our experience confirms this, these bacteria being identified in four of the six patients. It appears that the presence of atherosclerosis, with or without aneurysmal changes, makes the aorta more susceptible to the development of salmonella endovascular infection, but even the normal arterial intima, usually resistant to bacterial entrance, may be invaded by Salmonella.[18] Given the high prevalence of untreated salmonella gastroenteritis preceding endothelial infection, some authors[19] advise antibiotic therapy for its treatment in patients older than 50 years of age. A tendency toward an early rupture has been noted in the cases due to Salmonella infection.[20]

Reports of other less common organisms are starting to appear in the literature and may reflect the presence of immunocompromised hosts. *Listeria monocytogenes,* for instance, has been incriminated twice as the cause of IAAs[21] and was seen in one of our six patients. A poten-

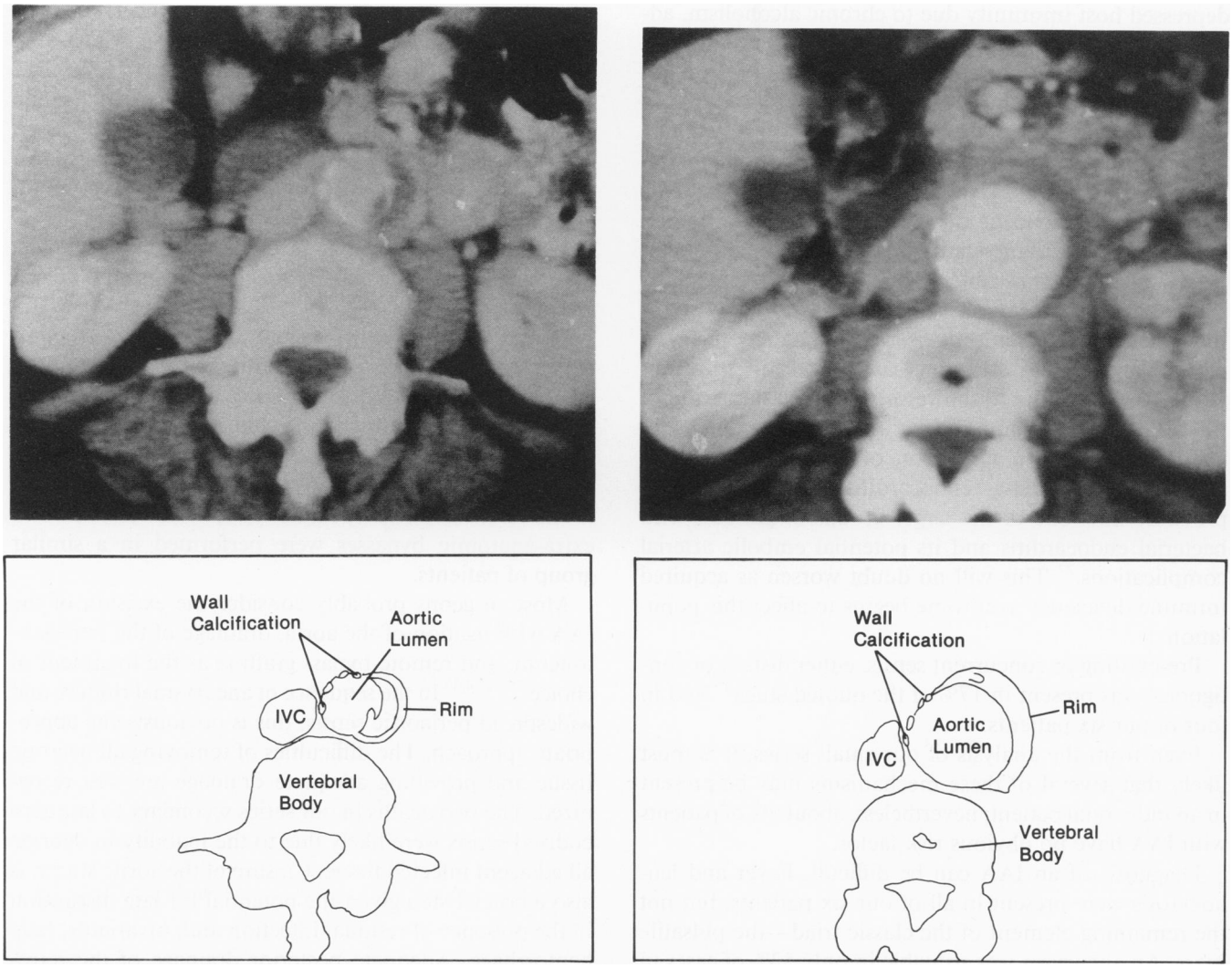

FIG. 4. (A, left) Contrast-enhanced CT scan shows an irregular periaortic soft tissue mass with peripheral enhancement consistent with inflammation. (B, right) CT scan 3 months later shows a sharp increase in the diameter of the aortic lumen (case 4).

tially large and increasing number of immunocompromised patients due to acquired immune deficiency syndrome is another at risk population for IAA. In the past year, three cases of an IAA have been reported in male patients with a positive test for the human immunodeficiency virus. No organisms were cultured from the aneurysmal contents in one of the patients, but *Salmonella* and *Mycobacterium tuberculosis* grew in the two others. These numbers could dramatically change in the near future.

Bacteroides fragilis, an anaerobic organism, has been reported[22,23] as the causative agent of IAA. Although these cases appear to be rare, it is possible that the incidence is higher than reported because negative cultures from IAAs have been stated to be found in as many as 25% of cases,[12] and this may reflect the fact that the anaerobic cultures may not be routinely obtained. The frequent use of antibiotics in general medical and surgical practice is another likely reason for the high percentage of failures in identifying a specific pathogen.

Campylobacter fetus has a particular predilection for the vascular endothelium in immunocompromised patients and, since 1971, nine documented cases of *C. fetus* sepsis associated with abdominal aortic aneurysms have been reported.[24] Rapid increase in the size of the aneurysm is found with this entity, and all the reported deaths in this series followed aneurysmal rupture.

Overall, gram-negative infections have shown a greater virulence with a significantly higher incidence of aneurysmal rupture and death (72% *versus* 25%) than aneurysms with gram-positive infections.[20]

Analysis of the associated conditions present in our group of patients showed that diabetes mellitus, malignant neoplasm, and rheumatoid arthritis were present, alone or in combination, in four of them, indicating compromised immunocompetence. Johansen and Devin[15] found

depressed host immunity due to chronic alcoholism, administration of corticosteroids or cytotoxic agents, and chronic renal failure present in 24% of the patients reviewed. Arterial trauma was the greatest risk factor and was present in 29%. The development of a "mycotic" aortic aneurysm after arteriography or cardiac catheterization is rare,[25,26] but given the large numbers of patients that undergo diagnostic or therapeutic (intra-aortic balloon pump, percutaneous transluminal coronary angioplasty, percutaneous balloon angioplasty, hemodialysis shunts) arterial catheterizations, one has to be aware of this possible complication. Arterial trauma secondary to parenteral drug abuse has increased markedly in recent years. Direct trauma to the vessel with contamination of the resulting hematoma during initial or subsequent injections will lead to formation of an infected pseudoaneurysm or bacterial endocarditis. Drug addicts are probably now one of the larger groups of patients with bacterial endocarditis and its potential embolic arterial complications.[27] This will no doubt worsen as acquired immune deficiency syndrome begins to affect this population.

Pre-existing or concurrent sepsis, either distant or contiguous, was present in 17% of the quoted study[15] and in four of our six patients.

Even from the analysis of our small series, it is most likely that several of these mechanisms may be present in an individual patient; nevertheless, about 3% of patients with IAA have no obvious risk factor.

Diagnosis of an IAA can be difficult. Fever and leukocytosis were present in all of our six patients, but not the remaining element of the classic triad—the pulsatile mass. An aneurysm was palpable in only 53% of cases in one series.[20] A number of cases present as fever of unknown origin with some nonspecific abdominal or chest pain and no distinctive clinical presentation. The systemic manifestations of sepsis may be discrete, and the IAA may go unrecognized, particularly in the early stages.

Positive blood cultures were found in only three of our cases, but a higher percentage (70%) has been reported.[28] Downstream arterial blood cultures from the femoral artery can be particularly helpful in the diagnosis of IAA. Traditionally, aortography is considered crucial to the diagnosis of this entity by demonstrating the presence of lobular, saccular, and eccentric aneurysms in an otherwise normal appearing aorta. Arteriography was performed in four of our patients but more for preoperative planning and confirmation of the CT findings than for diagnosis. Computed tomography scans suggested the diagnosis in all six patients. The CT features have been reported previously,[29] but early findings include the presence of a periaortic soft tissue density with rim enhancement, and intimal calcifications. Days to weeks later, this eccentric, thickened wall may increase in size, and the intimal calcifications may become disrupted or disappear. Enlargement of the aortic lumen eventually develop, which took place between 10 days and 3 months after the original scan in two of our patients; an unusual luminal shape also may be seen. Gas in the perianeurysmal soft tissue has been described.[30] The progression of a normal vessel to a markedly dilated and thinned one may be very rapid, as we and other authors[30–32] have observed.

The *surgical therapy* aims not only to eradicate the IAA and surrounding tissues, but also to re-establish distal arterial flow.

Dismal results of resection and vascular reconstruction for IAAs were described as recently as 1967.[33] All of the patients in that series died of persistent sepsis after *in situ* insertion of a graft in the infected field. Brown et al.,[12] among others, further pointed out the disadvantages of *in situ* reconstruction, where a mortality rate of 32% was found *versus* a mortality rate of 13%, when resection and extra-anatomic bypasses were performed in a similar group of patients.

Most surgeons probably consider the excision of the IAA with ligation of the aorta, drainage of the retroperitoneum, and remote bypass grafting as the treatment of choice.[22,25,34,35] In the sequence of aneurysmal rupture and widespread periaortic sepsis, this is obviously the appropriate approach. The difficulties of removing all necrotic tissue and providing adequate drainage are well recognized. The two deaths in our series secondary to late generalized sepsis were likely due to the inability to debride all adjacent infected tissue. Closure of the aortic stump is also a crucial step given the potential for late disruption in the presence of residual infection and, invariably, fatal hemorrhage. Adequate posterior drainage of the retroperitoneum is required because frequently, even after careful debridement, purulent material with the same organisms as found in the aneurysmal tissues continues to drain for variable periods.

The lower patency rates of the axillary-femoral grafts and the possibility of graft infection due to persistent or recurrent septicemia has led to the re-evaluation of the *in situ* graft interposition. Successful aneurysmal resection and graft replacement has been reported several times[15,21,24,25,36,37] in cases of IAAs after the first initial success by this method in 1962.[7] Two patients in our series were treated in this manner and were alive 2.5 and 5 years after operation. There is no uniformly accepted consensus regarding which patients should be treated by this approach. Reddy[13] describes an approach based on the results of a Gram stain obtained intraoperatively of suspicious areas of the aneurysmal wall and contents as well as the presence of gross infection. Aneurysmal resection and *in situ* graft replacement is advised for cases in which the Gram stain is negative and there is no evidence of macroscopic purulence. In the face of a positive Gram

stain but no gross infection, the same surgical procedure can be cautiously done if one can be fairly certain that debridement can fully clear the retroperitoneal infection. *In situ* reconstruction is also desirable in aneurysms of the suprarenal aorta involving the visceral arteries, given the lack of any practical alternative. In a large series of patients reported by Chan et al.[38] and others,[39] the authors conclude that this is the method of choice for central mycotic aneurysms but advise lifetime oral antibiotic suppression.

A persistent fever, positive blood cultures, evidence of anastomotic breakdown, or signs of retroperitoneal sepsis should indicate the need for the placement of an extra-anatomic bypass and prompt removal of the interposed aortic prosthesis. The rate of failure of interposition grafts has been reported to be 23% with infection caused by gram-positive organisms and up to 63% with gram-negative organisms.[40] Replacement grafting is also an expedient operation when the patient is in extremis. When there is evidence of gross infection, an extra-anatomic bypass should follow the resection of the IAA and the debridement of the retroperitoneal infected tissue. Concern about prolonged leg ischemia after excision of the aneurysm followed by the extra-anatomic bypass can be allayed by reversing the order of the procedures.[35] The possible bacterial seeding of the fresh graft in the process of the resection and debridement of the IAA and retroperitoneal tissues does not appear to have practical significance.[41]

The treatment of IAA begins preoperatively with the appropriate antibiotics adjusted to the cultured organism and with effective serum antibiotic levels. Surgery should, if possible, be performed after the establishment of effective antibiotic therapy but should not be delayed beyond that time (1 to 4 days). Aggressive and appropriate antibiotic treatment must be continued after operation to accomplish sterilization of the blood stream and the operative site. There are no definite recommendations for the duration of the postoperative antibiotic therapy, but a minimum of 6 weeks has been suggested.[13] In certain cases, and depending on the method of arterial reconstruction, oral antibiotics may be required for a lifetime.[42] Late reinfections have been reported in patients who did not continue the therapy, particularly caused by Salmonella organisms.[12]

If the infection is detected early, this approach should provide a favorable outcome in most of these patients (Table 2).

Much remains unknown about the clinical course of IAAs, but experience indicates that, although the rate of the disease progression varies widely between patients, the natural history of these processes is highly unfavorable and usually fatal because of sepsis or hemorrhage.

From our experience and the literature review, it appears that several aspects of this disease have changed over the years. These lesions appear more frequently now in an older population, although the acquired immune deficiency syndrome and drug epidemics may change the demographics of this disease in future years. The main risk factor is no longer the presence of bacterial endocarditis, and IAA will be secondary to various states of depressed immunity, infected sites of arterial trauma, and concurrent sepsis elsewhere. Computed tomography appears to provide earlier diagnosis and a good technique to follow the progress of this disease if the diagnosis is not initially made, but clinical suspicion of this entity is fundamental for its early diagnosis. Angiography provides the anatomic details of the lesion and adjacent vessels, but will not show the degree of periaortic extension that CT can demonstrate.

An early diagnosis permits the *en bloc* excision of the aneurysm and the perianeurysmal infection and allows an interposition graft, a shorter, one-stage procedure, with good long-term results. Delayed diagnosis with extensive periaortic sepsis will require ligation of the aorta and extra-anatomic bypasses. Aortic rupture continues to carry a high mortality rate.

TABLE 2. *Infected Aortic Aneurysms: Patient Survival*

Source	Year	No. of Cases	Survivors No. (%)
Jarrett et al.	1975	12	7 (59)
Davies et al.	1978	3	3 (100)
Scher et al.	1980	2	1 (50)
Johansen and Devin	1983	4	4 (100)
Bitseff et al.	1985	5	3 (60)
Taylor et al.	1987	4	4 (100)
Current series	1991	6	4 (66.7)

References

1. Osler W. The Gulstonian lectures on malignant endocarditis. Br Med J 1885; 1:467–469.
2. Jarrett F, Darling RC, Mundth ED, Austen W. Experience with infected aneurysm of the abdominal aorta. Arch Surg 1975; 110: 1281–1285.
3. Rokitansky. Handbuch der Paathologischen Anatomic, 2nd Edition, 1844, p 553; quoted in Lewis D, Schrager J. Embolomycotic aneurysms. JAMA 1909; 53:1808–1814.
4. Koch L. Ueber Aneurysma der Arteriae Mesenterichae Superioris. Inaug Dis Erlangen 1851; 5–23.
5. Tufnell J. The influence of vegetations on the valves of the heart in the production of secondary arterial disease. Dublin Q J Med Sci 1853; 15:371.
6. Stengel A, Wolferth CC. Mycotic (bacterial) aneurysms of intravascular origin. Arch Intern Med 1923; 31:527–554.
7. Sower ND, Whelan TJ. Suppurative arteritis due to Salmonella. Surgery 1962; 52:851–859.
8. Morris GC, Crawford ES, Cooley DA, DeBakey ME. Revascularization of the celiac and superior mesenteric arteries. Arch Surg 1962; 84:95–107.
9. Nakayama DK, O'Neill JA, Wagner H, Cooper A, Dean RH. Management of vascular complications of bacterial endocarditis. J Pediatr Surg 1886; 21:636–639.

10. Bergsland J, Kawaguch A, Roland JM, et al. Mycotic aortic aneurysms in children. Ann Thorac Surg 1984; 37:314–318.
11. Crane AR. Primary multilocular mycotic aneurysm of the aorta. Arch Pathol 1937; 24:634–641.
12. Brown SL, Busutill RW, Baker JD, et al. Bacteriologic and surgical determinants of survival in patients with mycotic aneurysms. J Vasc Surg 1984; 1:541–547.
13. Reddy DJ. Surgical treatment of infected abdominal aortic aneurysm. *In* Ernst C, Stanley J, eds. Current Therapy in Vascular Surgery, 2nd Edition. Philadelphia: BC Decker, 1989, pp 272–276.
14. Mohyuddin Z, Karjoo M. Successful management of ruptured mycotic abdominal aortic aneurysm in a child. Int Surg 1989; 74: 61–63.
15. Johansen K, Devin J. Mycotic aneurysms. A reappraisal. Arch Surg 1983; 118:583–588.
16. Wilson SE, Wagenen PV, Passaro E. Arterial infection. Curr Probl Surg 1978; 15:21.
17. Davies OG, Thornburn JD, Powell P. Cryptic mycotic abdominal aortic aneurysms: Diagnosis and management. Am J Surg 1978; 136:96–101.
18. Cohen OS, O'Brien TF, Schoenbaum SC, Medeiros AA. The risk of endothelial infection in adults with salmonella bacteremia. Ann Intern Med 1978; 89:931–932.
19. Lane GP, Cochrane AD, Fone DR. Salmonellal mycotic abdominal aortic aneurysm. Med J Aust 1988; 149:95–97.
20. Jarrett F, Darling RC, Mundth ED, Austen WG. The management of infected arterial aneurysms. J Cardiovasc Surg 1977; 17:361–366.
21. Harvey MH, Strachnan CJL, Thom BT. Listeria monocytogenes: a rare cause of mycotic aortic aneurysm. Br J Surg 1984; 71:166–167.
22. Reddy DJ, Lee RE, Oh HK. Suprarenal mycotic aortic aneurysm: surgical management and follow up. J Vasc Surg 1986; 3:917–920.
23. Jewkes AJ, Black J. Infection of an abdominal aortic aneurysm from an appendix abscess. J Cardiovasc Surg 1989; 30:870–872.
24. Rutherford EJ, Eakins JW, Maxwell JG, Tackett AD. Abdominal aortic aneurysm infected with Campylobacter fetus, subspecies fetus. J Vasc Surg 1989; 10:193–197.
25. Baker WH, Moran JM, Dorner DB. Infected aortic aneurysm following arteriographyy. J Cardiovasc Surg 1979; 20:373–377.
26. McCready RA, Siderys H, Pittman JN, et al. Septic complications after cardiac catheterization and percutaneous transluminal coronary angioplasty. J Vasc Surg 1991; 14:170–174.
27. Yellin AE. Ruptured mycotic aneurysm. A complication of parenteral drug abuse. Arch Surg 1977; 112:981–986.
28. Asthana S, Walker D, Iyengar S, Shafran S, Conly J. Mycotic aneurysm presenting as fever of unknown origin. West J Med 1989; 150:694–696.
29. Gomes MN, Choyke PL. Infected aortic aneurysms: CT diagnosis. J Cardiovasc Surg 1991; 32:4.
30. Vogelzang RL, Sohaey R. Infected aortic aneurysms: CT appearance. J Comput Assist Tomogr 1988; 12:109–112.
31. Byard RW, Leduc JR, Chambers RJ, Walley VM. The rapid evolution of a large mycotic aneurysm of the abdominal aorta. J Can Assoc Radiol 1988; 39:62–64.
32. Markus J, Tuttle RJ. Evolution of a mycotic aneurysm of the thoracoabdominal aorta. J Can Assoc Radiol 1989; 40:45–46.
33. Bennett DE, Cherry JK. Bacterial infection of aortic aneurysms—a clinicopathologic study. Am J Surg 1967; 113:321–326.
34. Scher LA, Brener BJ, Goldenkranz RJ, et al. Infected aneurysms of the abdominal aorta. Arch Surg 1980; 115:975–978.
35. Taylor LM, Deitz DM, McConnell DB, Porter JM. Treatment of infected abdominal aneurysms by extra-anatomical bypass, aneurysm excision and drainage. Am J Surg 1988; 155:655–658.
36. Parsons R, Gregory J, Palmer DL: Salmonella infections of the abdominal aorta. Rev Infect Dis 1983; 5:227–231.
37. Meade RH, Moran JM. Salmonella arteritis: pre-operative diagnosis and cure of Salmonella aortic aneurysm. N Engl J Med 1969; 281:310–312.
38. Chan FY, Crawford ES, Coselli JS, et al. In-situ prosthetic graft replacement for mycotic aneurysm of the aorta. Ann Thorac Surg 1989; 47:193–203.
39. Atnip RG. Mycotic aneurysms of the suprarenal abdominal aorta: prolonged survival after in situ aortic and visceral reconstruction. J Vasc Surg 1989; 10:635–641.
40. Ewart JM, Burke ML, Bunt TJ. Spontaneous abdominal aortic infections: essentials of diagnosis and management. Ann Surg 1983; 49:37–50.
41. Trout HH III, Kozloff L, Giordano JW. Priority of revascularization in patients with graft enteric fistulas, infected arteries or infected arterial prosthesis. Ann Surg 1984; 199:669–683.
42. Crawford ED, Crawford JL. Aortic infection. *In* Diseases of the Aorta. Baltimore: Williams & Wilkins, 1984, pp 340–376.

Discussion

DR. HUGH H. TROUT III (Bethesda, Maryland): President Ochsner, Dr. Jones, Members and Guests: In situations in which contamination is likely, it now seems that it is reasonably safe to proceed with *in situ* prosthetic graft insertion when no previous prosthetic graft has been placed. Such settings include a primary aortoenteric fistula with a communication between an abdominal aorta aneurysm and the duodenum. Another instance would be infected aortic aneurysms as presented by Dr. Gomes. One other instance in which *in situ* replacement seems possible is the setting of a prosthetic graft infected by bacterial biofilms such as coagulase-negative staphylococcus, as has been recently reported. For all other vascular contamination situations in my view, however, *in situ* grafting should be avoided if at all possible. Appropriate therapy in these instances should be a remote bypass such as an axillobifemoral bypass, followed by removal of the contaminated graft.

I have one question. Now that polytetrafluoroethylene (PTFE) is available in bifurcation form, do you believe because of reduced quantitative bacterial adherence that PTFE has an advantage over Dacron as a prosthetic replacement graft? I want to thank Dr. Gomes for providing me with a copy of his excellent manuscript and to congratulate him on a thorough presentation helping to define further those instances in which *in situ* grafting may be safely employed. Thank you for the privilege of the floor.

DR. MARIO N. GOMES (Closing discussion): I would like to thank Dr. Trout for the kind comments. I do believe that the use of a PTFE graft in this setting may further decrease the possibility of graft infection, but it is more important to make the diagnosis of IAA in early phase and be fairly certain that the periaortic involvement is not present. In these circumstances, the potential problem of graft infection should not be significant, but the use of a PTFE graft may be advantageous. I am fairly confident that an early diagnosis of IAAA will lead to a safe graft interposition, which is not the case when extensive periaortic infection is present.