Tuberculous Mycotic Aneurysm of the Aorta*

Review of Published Medical and Surgical Experience

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To define the epidemiology, pathogenesis, pathology, presentation, and management of tuberculous mycotic aneurysm of the aorta (TBAA) in the therapeutic era, we reviewed all of the cases reported in the English language literature from 1945 to the present. To the 39 cases in the published literature, we add two cases of our own. Although it is exceedingly rare, the prevalence of this lesion has remained relatively constant. In 75% of the cases, TBAA appeared to result from erosion of the aortic wall by a contiguous focus; 25% from direct seeding of the aortic intima or of the adventitia or media (via the vasa vasorum). Most of the aneurysms were saccular (90%) and false (88%). The thoracic and abdominal aortas were affected with equal frequency. The mean (± SD) age of the patients was 50 ± 16 years. Twenty-two were men, and 19 were women. In 63% of the cases, tuberculosis (TB) was diagnosed at presentation. Disseminated TB was present in 46% of the cases. One or more of three clinical scenarios suggested TBAA: persistent pain, major bleeding, and a palpable or radiographically visible para-aortic mass, especially if it is expanding or pulsatile. In turn, each of these findings suggested a complication of TBAA that may be an indication for surgical intervention. Among the patients who were offered both medical and surgical treatment, 20 of 23 (87%) survived. Among those who were offered only one form of treatment or were offered no treatment at all there were no survivors. Both in situ reconstruction with a prosthetic graft, and extra-anatomic bypass appeared to offer excellent results, provided that an effective regimen of antituberculous drugs was delivered postoperatively. We offer our conclusions: (1) symptomatic TBAA is a rare but uniformly fatal lesion if not diagnosed promptly, (2) in the context of active TB, and especially miliary TB, TBAA should be suspected whenever one or more of the three clinical scenarios are present, and (3) combined medical and surgical therapy appears to offer the best chance of a cure.

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Key words: extrapulmonary tuberculosis; tuberculous mycotic aneurysm of the aorta

Abbreviations: TB = tuberculosis; TBAA = tuberculous mycotic aneurysm of the aorta

The term mycotic aneurysm is a misnomer that has nevertheless been generally adopted to describe aneurysms that occur secondary to the infectious destruction of an arterial wall. Osler¹ was the first to describe a patient with multiple beadlike aneurysms resulting from suppuration in the vessel wall. Because the appearance of the lesion resembled a fungal growth, he used the term mycotic aneurysm.

In the preantibiotic era, mycotic aneurysms of intravascular origin (infection from “within”) were, for the most part, limited to patients with infective endocarditis. In a series of 217 cases reported in 1923,² 86% of the mycotic aneurysms were associated with infective endocarditis. In no patient was the aneurysm tuberculous. Although extension of infection to the walls of the small pulmonary and meningeal arteries from neighboring or contiguous inflammatory foci (infection from “without”) often caused aneurysms in tuberculous cavities and the meninges, tuberculous aneurysms of larger vessels, particularly the aorta, whether from within or without, were very rare. A review by Haythorn³ in 1913 identified only 4 cases, a review by Gellerstedt and Säfwenberg⁴ in 1933 identified only 21 cases, and a...
search of the world literature by Silbergleit et al in 1965 identified a total of 51 cases. Even when allowing for the limited imaging capability of the past (most cases were diagnosed postmortem), these are remarkably small numbers, especially when one considers the following: both infection with, and disease resulting from tubercle bacilli were very common in the preantibiotic era; systemic bacillemia is a constant feature of primary infection with tubercle bacilli; and involvement of the nonsystemic circulation is a regular feature of pulmonary tuberculosis (TB).6

Although the first case of tuberculous involvement of the aorta (aortitis) was reported in 1882 by Weigert,7 the first case of tuberculous mycotic aneurysm of the aorta (TBAA) was not reported until 1895.8 No patients were known to have survived TBAA until the combined technologies of modern imaging capability, antituberculous drugs, and vascular grafts became available. In 1952, Herndon et al9 attempted the first surgical repair of TBAA, but the patient died 6 days after surgery. In 1955, Rob and Eastcott,10 using an orlon cloth graft, reported the first successful reconstruction of TBAA. The present review describes the epidemiology, pathogenesis, pathology, presentation, and management of these lesions in the therapeutic era. All of the cases of TBAA reported in the English language literature from 1945 to the present were researched. To the 39 cases in the published literature, we add two cases of our own.

**Case 1**

A 67-year-old male immigrant to Canada from Afghanistan was admitted to the hospital with a 6-month history of fever, night sweats, abdominal pain, and constipation. Ten days previously, a technically adequate ultrasound of the abdomen had been unremarkable; the infrarenal aorta measured 1.93 cm in diameter. The patient was febrile and underweight. His abdomen was diffusely tender, but without guarding or rebound. A chest radiograph, initially interpreted as demonstrating increased interstitial markings, evolved over 2 weeks to a miliary pattern. Airway secretions were negative for acid-fast bacilli on smear, but were positive on culture for *Mycobacterium tuberculosis* that was susceptible to all first-line antituberculous drugs. An infused CT scan of the abdomen, performed on day 20 of hospitalization and 8 days before the introduction of antituberculous drug therapy (isoniazid, 300 mg/d; rifampin, 600 mg/d; pyrazinamide, 1,500 mg/d; and ethambutol, 800 mg/d), demonstrated enhancement and swelling of the omentum on the right, and no other abnormalities suggestive of peritonitis. Some months later, a careful review of the same CT scan revealed a thickened inflamed wall of the cecum, an inflammation of the mesentery, a thickened greater omentum, and an enhancing peritoneum. No ascites or lymphadenopathy was evident, as shown in Figure 1 (top, A). The same CT scan also demonstrated the lower abdominal aorta to be displaced anteriorly off of the vertebral bodies by a 3 × 2-cm enhancing mass (Fig 1).

On day 78 of hospitalization (day 51 of antituberculous drug treatment), the patient’s continuing abdominal pain prompted another infused CT scan of the abdomen (Fig 2 [top, A, and middle, B]). The omental thickening and peritoneal enhancement noted previously had then resolved. However, there

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21906/ on 06/22/2017)
was an interval progression of the para-aortic inflammatory reaction with what appeared to be a false lumen opacified in the retroaortic mass, as shown in Figure 2 (bottom, C). Because a mycotic aneurysm with a contained rupture was suspected, a laparotomy was performed later that day. An inflammatory mass appeared to involve the infrarenal aorta above its bifurcation. Proximal and distal control of the aorta was secured, and the intervening aorta was incised. A 4- to 5-cm mass emanated from a hole measuring 2 cm in diameter in the posterior wall of the aorta. The contents of the mass posterior to the hole included a clot and fibrinous white material that was later demonstrated to be necrotic tissue teeming with acid-fast bacilli. Ultimately, this tissue tested culture-positive for \(M\) \textit{tuberculosis}. The area was debrided, and an omental patch was inserted over the aorta and iliac vessels for added protection. The abdomen was closed, and an axillofemoral graft was performed. The patient’s postoperative course was uneventful, and he completed a full course of antituberculous drug therapy.

**Case 2**

Just 4 months after the first case, a 77-year-old white woman presented with a 4- to 6-week history of general malaise, anorexia, weight loss, night sweats, and intermittent fever. For 3 weeks, she had had back pain severe enough to interfere with her ability to walk. On examination, she was febrile and overweight, with percussion tenderness of the back at the thoracolumbar level. No neurologic deficit was present. A chest radiograph demonstrated a miliary pattern, and a spinal CT scan suggested spondylitis at the T11–12 level. A vertebral disk aspirate was smear- and culture-positive for \(M\) \textit{tuberculosis} that was susceptible to all first-line antituberculous drugs. On day 18 of hospitalization, 14 days after the introduction of antituberculous drugs (isoniazid, 300 mg/d; rifampin, 600 mg/d; pyrazinamide, 1,500 mg/d; and streptomycin, 800 mg/d), a complaint of vague abdominal pain prompted a CT scan of the abdomen. The images suggested tuberculous involvement of the right crus of the diaphragm, and a mycotic aneurysm of the aorta at the level of the celiac axis. The presence of a suprarenal saccular aneurysm was confirmed on the coronal section of an MRI. A sagittal section and a gadolinium-enhanced axial section, as shown in Figure 3, suggested that the aneurysm was secondary to the spondylitis and involvement of the right crus. Angiography that was done in preparation of the patient for surgery demonstrated a 3.5 × 5.0-cm false aneurysm arising from the lateral wall of the aorta just below the celiac axis (Fig 4). On the 25th day of hospitalization, the patient was taken to the operating room. Through a thoracoabdominal incision, a dacron aortic interposition graft was placed, and the celiac and superior mesenteric arteries were revascularized. The false aneurysm was formed through a 2 × 1.5-cm hole in the aorta. Its wall consisted of fibrin, granulation tissue, and necrotic debris that tested positive for
acid-fast bacilli on stain and that was culture-positive for *M tuberculosis*. The aorta itself was irregular at the site of the aneurysm and was minimally atherosclerotic elsewhere. Two months postoperatively, the patient was still recovering in the hospital.

**Discussion**

**Epidemiology**

A tuberculous aneurysm of the aorta is exceedingly rare. Over the past 53 years, since the introduction of antituberculous drug therapy, only 41 cases have been reported in the English language literature. An additional case may have occurred, but the documentation was considered inadequate for inclusion. Reviews by Haythorn in 1913, by Gellerstedt and Säfwenberg in 1933, from the preantibiotic era, and by Silbergleit et al in 1965, spanning the pre- and postantibiotic era, suggest that this lesion has always been rare, a conclusion that is further supported by independent autopsy and clinical data. During the 50-year period from 1902 to 1951, when 22,792 necropsies were performed at Boston City Hospital, 338 aortic aneurysms were encountered and only one was tuberculous. In another review of 178 abdominal aortic aneurysms found among 20,201 necropsies at the Mayo Clinic from 1925 to 1954, only 6 were mycotic and none were tuberculous. In a recent population-based series of patients with disseminated TB, a group that is at increased risk of TBAA (see below), not a single case of TBAA was identified over a 15-year period. TBAA case reports have, for the most part, come from the industrialized world, where the prevalence of TB has declined over the past half-century. Nevertheless, the 5-year rate of reporting of TBAA cases has not changed significantly, probably because advances in diagnostic and treatment capability have offset the decrease in prevalence.
Pathogenesis

Of the 41 TBAA cases reported in the English language literature in the therapeutic era, 5–9, 10, 12, 15–38, 40–45, 47–49 20 patients (49%) died. 9, 12, 15–26, 30, 33, 35, 37, 40, 41 The case of Harris and Hougen was also reported by Sung et al. The case of Patra et al. was also reported by Sandron et al. It is considered probable that one case was later included in two case series, and another case was later included in a single case series. Because all of the deaths were followed by a postmortem, and because most of the survivors were from recent years when imaging capability was more sophisticated, some conclusions may be drawn about pathogenesis.

Tubercle bacilli may reach the aortic wall in one of three ways:

1. The bacilli may implant directly on the internal surface of the vessel wall. Normally, the aortic intima is very resistant to infection. However, when this lining is altered by atherosclerosis (plaques and/or ulcers), the resistance to infection is lowered, and the surface may become colonized by blood-borne bacilli. With TB case rates in industrialized countries being highest among the elderly, (who have most of the atherosclerosis), one might anticipate that seeding of the aorta would be a common finding. Schmorl, reporting on 123 autopsies of patients with acute miliary TB, found 5 cases showing TB of the inner wall of blood vessels localized on atheromatous ulcers. In contrast, Slavin et al., reporting on 100 autopsies of “late generalized tuberculosis,” did not describe any cases of tuberculous seeding of atheromas. Unless intimal seeding is complicated by endarteritis (inflammation of the arterial wall) and aneurysm/perforation, it may go unrecognized during life. In the present series, atheromatous plaques were described in 14 of 24 patients (58%) whose nontuberculous aortas were described.

2. The bacilli may be carried to the adventitia or media by the vasa vasorum.

3. Involvement of the vessel wall may occur by direct extension (or indirectly via the lymphatics) from a contiguous focus such as a lymph node or paraspinal abscess.

In the majority of the reported cases of TBAA (30 of 40, 75%), a contiguous focus of TB was described: lymph nodes in 19 of 30 cases (63%), and other sites—including paraspinal abscesses, lung, pericardium, vertebrae, and prostate—in 11 of 30 cases (37%). Deciding which came first, however—the aortitis or the contiguous focus—can be difficult. In some cases, the authors were quite convinced that the erosion of the aorta, with the subsequent aneurysm formation, was secondary to the adjacent focus. In other cases, especially in those with adjacent lymph node disease, the authors were uncertain whether aortitis and aneurysm formation proceeded from tuberculous lymphadenitis or vice versa. It seems probable, though, based on the number of cases with a credible contiguous focus, that the dominant mechanism of aneurysm formation was erosion through the aorta of a contiguous focus of disease. This conclusion is consistent with events in the pulmonary circulation, where virtually all endarteritis is secondary to contiguous parenchymal disease.

In the remaining 10 reported TBAA cases (25%), including our first case study, no contiguous focus of disease was described. 18, 24, 27, 32, 37, 38, 43, 44, 48 Thus implicating either mechanism 1 or 2 (above). Such aneurysms have been called “primary mycotic aneurysms,” a term coined by Crane in 1937 to describe “a lesion developing in the wall of an artery which is not associated with any demonstrable intravascular inflammatory focus as with bacterial endocarditis, or with any inflammatory process in the surrounding tissues.” In our first case study, a temporally spaced series of radiologic studies provided convincing evidence for a hematogenous pathogenesis. An ultrasound of the abdomen performed 10 days before hospital admission revealed a normal infrarenal aorta. On the day of admission, however, a chest radiograph demonstrated an interstitial pattern that evolved to a classical miliary pattern over 2 weeks. Since it takes an estimated 2.5 to 6 weeks for radiographic changes to develop after dissemination, it is very unlikely that the aorta was the source of the bacillemia. Rather, CT images (Fig 1, 2) strongly suggest that aortitis and aneurysm formation followed the direct seeding of the aortic intima (atherosclerosis was present) or, alternatively, the adventitia or media (via the vasa vasorum). No para-aortic focus was present. The perirenal space that contains the aorta was isolated from other intra-abdominal disease (peritonitis and possible pericecal abscess) by Gerota’s fascia. In our second case, CT and MRI scans strongly suggested that aortitis and aneurysm formation followed from tuberculous spondylitis and paraspinal abscess.

In contrast to nontuberculous species that have been reported to cause embolomycotic (embolization of infected material from the heart to either the vasa vasorum or a small arterial branch) aneurysms in patients with endocarditis, no case of TBAA was reported to be associated with endocarditis. M tuberculosis was considered to be the etiologic agent in all TBAA cases but one caused by bacillus Calmette-Guérin vaccine.
Pathology

In 1913, Haythorn\(^3\) described four types of tuberculous arterial disease: (1) miliary TB of the intima, (2) polypi of tuberculous tissue attached to the intima, (3) TB involving several layers of the wall, and (4) tuberculous aneurysm.

In the pre- and early postantibiotic era, when necropsies were more frequently performed, aortitis (endarteritis of the aorta) without aneurysm (type 3 of Haythorn) was reported to be as common as aortitis with aneurysm (type 4 of Haythorn).\(^5\) The perforation of the infected vessel wall might be contained (an aneurysm) or uncontained (a perforation without aneurysm formation). The difference between the two was often subtle.\(^59,60\) Recent reports from India suggest the possibility of additional pathology, heretofore unrecognized in Western experience. These reports describe irregularities of the aortic lumen, stenotic lesions, and occlusion in young patients with TB elsewhere in the body.\(^59–63\) The authors conjectured that some of the lesions may have resulted from a hypersensitivity reaction to tuberculous antigens.

In the present series, aneurysms of the thoracic and abdominal aorta occurred with equal frequency. There were a total of 19 aneurysms of the thoracic aorta (3 ascending,\(^20,28,38\) 2 arch,\(^36,37\) and 14 descending\(^12,16,19,21,24,26,30,31,33,35,37,40,42,45\)). There were a total of 21 aneurysms of the abdominal aorta (4 suprarenal,\(^15,23,34\) [including our case 2 study] and 17 infrarenal,\(^5,9,10,17,18,22,25,27,29,30,32,41,44,47–49\) [including our case 1 study]). There was also one thoracoabdominal aneurysm.\(^43\) The rarity of thoracoabdominal localizations is perhaps a result of the low incidence of atheroma at this level, the small number of lymph nodes around this segment of the aorta, or the separation of the aorta from its neighboring structures by the diaphragm.\(^43\) Most of the aneurysms were saccular (98%) and false (88%), respectively: 35 of 39 and 28 of 32. Three aneurysms were true,\(^30,36\) and one was dissecting.\(^24\) The type of aneurysm (true, false, or dissecting) was not reported in nine of the cases.\(^10,12,18,29,30,32,33,47,49\) Four patients (10%) had more than one tuberculous mycotic aneurysm.\(^26,41,44,48\) In one case, the infection may have resulted from a preexisting nonmycotic aneurysm. Calcification was very uncommon. Rupture with massive bleeding occurred in 14 of the 20 deaths (70%),\(^15–17,19–23,25,30,33,37,40,41\) Three patients died perioperatively,\(^9,26,35\) one patient each died of miliary TB\(^18\) and congestive heart failure,\(^24\) and in one patient the cause of death was not reported.\(^12\)

Presentation

The first 20 cases of TBAA in this series\(^8,10,12,15–30\) were significantly more likely to be diagnosed at postmortem than the second 21 cases,\(^5,31–38,40–45,47–49\) (5%; including both of our case studies); 11 of 20 vs 1 of 21 (p < 0.05). This may reflect the advent of increased awareness and improved imaging capability, as well as the gradual decrease in the number of necropsies that were performed. The mean (± SD) age was 50 ± 16 years old (range, 6 to 86 years), and the gender distribution was almost equal, 22 men and 19 women.

TB of any type was diagnosed on presentation in 25 of 40 cases (63%; including both of our case studies).\(^17–20,22,24,28,29,30,32,33,34,35,37,38,40–45,48\) Disseminated TB was present in 19 of 41 patients (46%). Seven patients had disseminated TB at postmortem.\(^9,16,18,19,24,25,26\) Twelve patients had disseminated TB at premortem, including both of our case studies,\(^12,27,28,33,35,37,38,40,43,45\) This is a remarkably high number, given that disseminated TB accounts for only 3% of all TB cases in populations with a low seroprevalence of HIV.\(^14\)

One or more of three clinical scenarios suggested TBAA in 39 of 40 patients (98%; Table 1). Each scenario suggested a complication of TBAA that may be an indication for surgical intervention: (1) persistent chest, abdominal, or back pain (n = 25; including both of our case studies),\(^5,9,10,15–17,19,20,23–30,41–44,47–49\) (2) hypovolemic shock or other evidence of major bleeding, particularly into the lung or GI tract, but also into the pleural space, peritoneal cavity, retroperitoneum, or pericardial space (n = 15),\(^15–17,19,21–23,25,26,30,33,34,37,40,45\) or (3) palpable or radiographically visible para-aortic mass, especially if expanding or pulsatile (n = 25),\(^3,10,20,21,23,26,28–30,31–38,40–42,44,45,47,48\) Of the

### Table 1—Clinical Presentation of Tuberculous Aneurysms of the Aorta

<table>
<thead>
<tr>
<th>Clinical Scenario*</th>
<th>Abdominal TBA</th>
<th>Thoracic TBA</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent chest, abdominal, or back pain, No. of patients (%)</td>
<td>17 (77)</td>
<td>8 (44)</td>
<td>25 (64)</td>
</tr>
<tr>
<td>Hypovolemic shock or other evidence of major bleeding, No. of patients (%)</td>
<td>6 (29)</td>
<td>9 (50)</td>
<td>15 (38)</td>
</tr>
<tr>
<td>Palpable or radiographically visible para-aortic mass, especially if expanding or pulsatile, No. of patients (%)</td>
<td>11 (52)</td>
<td>14 (77)</td>
<td>25 (64)</td>
</tr>
</tbody>
</table>

*Clinical information was not available on one case.\(^12\)
patients with abdominal TBAA, scenarios 1 and 3 were the most common. Of the patients with a thoracic aneurysm, scenario 3 was most common (Table 1). Massive bleeding was a more common presentation among the first 20 reported cases (50%) than it was among the last 21 reported cases (24%), respectively: 10 of 20 vs 5 of 21. Short periods of “herald” bleeding often warned of subsequent exsanguinating hemorrhage. Scenario 3 was more common among the recent thoracic TBAA cases (90%) than among the earlier reported group (50%), respectively: 9 of 10 vs 4 of 8.

When these scenarios are present in the context of TB and, particularly, disseminated TB, TBAA should be suspected. Fever was reported in 14 of 40 patients (35%; including both of our case studies).10,16,19,20,23,30,32,35,36,38,49 Hypertension was very uncommon. Of the four patients who were reported to have an elevated BP, three (including our case 2 study) had a false aneurysm of the abdominal aorta,5,23 and one patient had a dissecting aneurysm of the thoracic aorta.24 This last patient was severely hypertensive. A few patients had an underlying condition that was known to increase the risk of TB: one was HIV-infected,49 one had rheumatoid arthritis,10 and another was on cytotoxic drugs.47

In 15 of 40 patients (38%), TB was not diagnosed at presentation. Mycotic aneurysms in these patients have been termed “cryptogenic,” ie, arising in the absence of an obvious inflammatory lesion elsewhere.64 In patients diagnosed with an aortic aneurysm who may be febrile but have no diagnosis of TB, infective endocarditis, or other source of pyogenic infection, TBAA should be considered if the aneurysm is false, if it does not involve the ascending aorta, and if it is associated with a contiguous focus of disease on CT scan.38

Fluoroscopy and aortography were very useful investigative tools with one caveat: a tuberculous aneurysm need not pulsate31 or fill with contrast.44,48 This is important to remember considering that the failure to do so may lead to the performance of a biopsy as the next diagnostic test. Since the first report of its use by Harris and Hougen in 1978,38 an infused CT scan has proved to be the most reliable means of demonstrating the aneurysm, and it often provides useful information on the status of the para-aortic tissue, as evidenced in both of our case studies.42,43,45,47,49 In our first case, it was the saccular appearance on CT in a patient with disseminated TB that suggested the TBAA diagnosis. In our second case, an MRI scan provided additional information. The MRI scan promises to be a useful investigative procedure in the future.95

Management

It is clear that treatment for symptomatic TBAA is both medical and surgical (Table 2). No patients survived without medical therapy. Both of the patients who underwent surgery but did not receive antituberculous drugs died,9,26 and no patient survived without surgery. Fifteen patients,12,15–19,21–25,30,37,40,41 including five who had been on antituberculous drugs for 1 or more months,17,19,22,30,40 did not have surgery and died. Three patients underwent surgery and died intraoperatively,20,33 or immediately postoperatively.36 Each of these three patients had been taking antituberculous drugs for a month or more before undergoing surgery. All the remaining patients received both surgery and antituberculous drugs, and all survived. In 19 of 21 patients, including both of our case studies, the duration of follow-up was reported, averaging (± SD) 10 ± 6 months (range, 2 to 24 months)5,10,28–30,31,32,34,36–38,42–45,47,49 Among these 21 patients, 9 (including our case 1 study) had taken antituberculous drugs for 1 or more months before undergoing surgery.27,29,33,34,37,38,42,45 Thus, despite 1 or more months of anti-TB drugs, progression of the lesion resulting in death or surgery occurred in 17 of the total of 41 cases. It is conceivable that access to the aneurysm by what is otherwise an effective antituberculous drug regimen is limited to the aortic blood (the small vessels within the vicinity of the aneurysm were invariably thrombosed) which may not easily penetrate the laminated thrombus that forms the wall of the aneurysm. Mechanical forces no doubt further contribute to the propagation of the lesion.

In this regard, our first case was quite illustrative. Between the first and second CT scan of the abdomen, during which time the patient received 51 days of antituberculous drug therapy, there was, as demonstrated by CT, a complete resolution of the intra-

<table>
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<tr>
<th>Table 2—Treatment and Outcome of Patients With Tuberculous Mycotic Aneurysm of the Aorta</th>
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<tbody>
<tr>
<td><strong>Treatment</strong></td>
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<tr>
<td></td>
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<tr>
<td>Neither medical nor surgical</td>
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<tr>
<td>Surgical alone</td>
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<tr>
<td>Medical alone</td>
</tr>
<tr>
<td>Both medical and surgical</td>
</tr>
<tr>
<td>Including preoperative medical therapy*</td>
</tr>
<tr>
<td>Not including preoperative medical therapy</td>
</tr>
<tr>
<td><strong>Total</strong></td>
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</tbody>
</table>

*Refers to preoperative medical therapy of 1 month or more.
abdominal inflammatory reaction. Nevertheless, there was obvious progression of the aneurysm, and *M tuberculosis* could still be isolated from the arterial wall.

In deciding how to manage these lesions, it is important to note that almost all of the cases of TBAA reported in this series were symptomatic with persistent pain or bleeding, complications indicating a possible need for surgery. Only six patients presented with scenario 3 alone,31,32,35–38 and they were all operated on. It is not known whether all asymptomatic TBAAs require surgery. It would certainly seem to be appropriate to operate on an expanding lesion, as well as an asymptomatic false TBAA, given the pathology of these lesions and the uniformly fatal outcome should the rupture become uncontained. In the setting of active TB, a small asymptomatic fusiform aneurysm, especially one whose mycotic nature is questioned (for example, when no para-aortic focus is present), might reasonably be observed.

Once symptomatic TBAA is identified, surgery must not be delayed. The size of the aneurysm does not appear to influence the need for surgery; however, aneurysms as small as 1.0-cm in diameter may rupture,26 and the destruction of the wall may occur with unpredictable rapidity. Antituberculous drugs must be instituted the moment that TB is suspected. However, even when these drugs are begun at the time of, or shortly after, successful surgical repair (as in the cases of 13 of 20 successfully treated patients), good outcomes may be anticipated.

Surgical experience suggests that both in situ reconstruction (assuming that the anastomotic sites are free of disease) and extra-anatomic bypass (when technically feasible)66 are appropriate procedures, provided that the repair is followed by an effective regimen of antituberculous drugs (Table 3). It must be recognized, however, that although the presence of an aneurysm and its mycotic nature might be predicted preoperatively, the likelihood of it being tuberculous might not be anticipated. In over one third of the patients in this series, TB was not diagnosed at presentation. Even at surgery, determining the tuberculous nature of the lesion is problematic. The gross appearance may not be distinctive, and acid-fast stains are unlikely to be performed. Thus, it would be entirely appropriate for the surgeon to consider the lesion to be one of the more common nonmycobacterial mycotic aneurysms, or perhaps an “inflammatory” aortic aneurysm,67 and to proceed accordingly. Since such an approach is more likely to lead to the performance of extra-anatomic bypass68–72 or, more recently, the in situ insertion of an aortic conduit homograft,73,74 the surgical intervention should nevertheless be successful, provided that specimens are sent for histopathology and/or acid-fast stain and culture, and that surgery is followed by appropriate medical therapy. Because specimens are not always handled as such, it is conceivable that some cases of undiagnosed TBAA have been cured with surgery alone.

### Table 3—Surgical Experience With Tuberculous Aneurysm of the Aorta*

<table>
<thead>
<tr>
<th>Type of Repair</th>
<th>No. of Patients</th>
<th>Outcome</th>
<th>Survived†</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aneurysmorrhaphy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic26,28</td>
<td>2</td>
<td></td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Abdominal9</td>
<td>1</td>
<td></td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>In Situ Reconstruction</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic31,35,37,38,42,43,45</td>
<td>7†</td>
<td></td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Abdominal6,10,27,30,32,47,49,49</td>
<td>10</td>
<td></td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Extra-anatomic Bypass</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Thoracic</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Abdominal24, Case 1</td>
<td>2</td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
<td></td>
<td>19</td>
<td>3</td>
</tr>
</tbody>
</table>

*The two patients in whom the type of repair was not reported and the two patients in whom fatal rupture occurred during dissection are not included in the table. †“Survived” indicates patient survival for 1 or more months after surgery. ‡The number includes one patient who underwent aneurysm resection and replacement with a patch graft,35 and one patient who had a thoracoabdominal aneurysm repair.43

### Conclusions

This retrospective case series of TBAA in the therapeutic era was compiled from all of the reports of this lesion in the English literature. Accordingly, the study has some obvious limitations. Almost all of the cases were from English-speaking developed countries, thus compromising the broader applicability of the results. Furthermore, the cases reported in the literature may not be representative of all of the TBAA cases in developed countries during the study years. Better therapy and fewer necropsies may have skewed the probability of reporting toward those cases having successful outcomes. Few reports deal with asymptomatic TBAA. Recognition of these limitations, however, must be tempered by the understanding that no population-based series or controlled clinical trial is likely to be performed.

1. Symptomatic TBAA is an extremely rare but uniformly fatal lesion if not diagnosed promptly.

2. In the context of active TB, especially miliary TB, TBAA should be suspected whenever one or more of three clinical scenarios is present (Table 1).

3. There is no evidence that medical (or surgical) therapy alone will cure these lesions.
4. Combined medical and surgical therapies would appear to offer the best chance for a cure.
5. Surgery for symptomatic TBAA must be performed promptly.
6. Both in situ reconstruction with a prosthetic graft, and extra-anatomic bypass appear to offer excellent results, provided an effective regimen of antituberculous drugs is delivered postoperatively.

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