Mycotic aortitis occurring after aortic dissection is rare. This article describes the case of a 26-year-old woman with a complicated type B aortic dissection who became deathly ill from a mycotic aortic aneurysm 4 months after her initial dissection. Prior to her dissection, the patient was healthy with no family history of Marfan syndrome, Ehlers-Danlos syndrome, Loeys-Dietz syndrome, or other aortopathy. She was involved in a minor automobile accident 2 weeks prior to her dissection but suffered no major injuries.

PRESENTATION
This patient’s story started on November 1, 2004, during a morning walk when she felt a sudden 10/10 pain in her chest and back that was followed within minutes by paralysis of her legs. After some delay, she was transported to her community hospital, where she was diagnosed with a type B aortic dissection with aortic occlusion at the renal artery level and occlusion of the right renal artery. There was significant delay before she was transferred to a tertiary medical center, where she underwent emergent axillofemoral bypass for lower extremity and pelvic revascularization.

Because of the delay in treatment, the patient had extensive myonecrosis of her legs requiring fasciotomies of the legs, including the left thigh with eventual debridement of a significant portion of the left quadriceps. This myonecrosis with the ischemic insult to the right kidney resulted in dialysis-dependent renal failure for 3 weeks. The patient did not have spinal cord ischemia. Revascularization of her legs recovered leg function, and eventually with intensive physical therapy, she became ambulatory. She had vancomycin-resistant enterococci (VRE) and Clostridium difficile colitis during this hospitalization.

She was transferred to rehab on December 1, with open granulating thigh and leg wounds, which were colonized with MRSA. Her eGFR was 19, she had lost 25% of her body weight, and her stool was VRE positive. On January 10, 2005, 3 months after her initial dissection, she was discharged home. She was hospitalized from January 25 to February 10 for final skin grafting without complications.

On March 10, the patient was readmitted with a 3-week history of fevers, progressive fatigue, weight loss, and a 1-week history of back and abdominal pain radiating to the left groin. Her ESR (erythrocyte sedimentation rate) was 122, C-reactive protein test was 22, white blood count (WBC) was 22,000, and blood cultures were positive for MRSA. There was no bacterial endocarditis by echo, colonoscopy was positive for a few superficial ulcerations, and indium WBC scan was nondiagnostic. The skin grafts were healed without cellulitis, and the urine was clear. Computed tomography (CT) showed no pneumonia, abscesses, or infection in the axillofemoral
graft, and the thrombosed abdominal aorta measured 3.5 cm in diameter (Figure 1). She was treated with intravenous vancomycin and rifampin for sepsis from MRSA, the source of which remained obscure. The patient failed to improve over the next 2 weeks, remaining febrile with a WBC > 20,000 with progressive weakness and cachexia. By March 28, her abdominal aorta had increased to 7.5 cm in diameter, and she was referred and transferred to the University of Wisconsin Hospitals and clinics (UWHC) with the diagnosis of mycotic aortic aneurysm (Figure 2).

**ASSESSMENT**

On arrival at UWHC, it was evident how critically ill the patient was. She was lethargic and malnourished, with an albumin of 1.7 and prealbumin of 11. She had lost 40% of her body weight since November. Her serum creatinine level was 2.5, and her WBC was 25,000 despite weeks of antibiotics. She was febrile to 101°F with a sinus tachycardia of 110 to 120 and hypotensive with systolic blood pressures of 70 to 80 mm Hg. Her abdomen was diffusely tender with an easily palpable tender pulsatile mass. From her CT scan, it was obvious she had mycotic aortitis with a rapidly expanding mycotic aneurysm, which had increased from 3.5 cm on March 10 to 5.5 cm on March 25 to 7.5 cm on March 28, the day of admission. The visceral aorta had also dilated significantly since March 10.

It was obvious that the patient needed emergent surgical treatment, but what repair could cure someone with so much MRSA-infected phlegmon? Her type B aortic dissection further complicated decision making because of unknown aortic integrity, with the infection making the use of felt pledges unwise. With MRSA and the amount of infected tissue, we believed the likelihood of successful treatment with a synthetic material was low. The use of cryopreserved aorta was problematic because of the size differential, suturing to a dissected dilated aorta, and the risk of continued infection with anastomotic or graft failure. The most likely cure was the use of live viable tissue to reconstruct the aorta after radical debridement so antibiotic could reach vascularized tissue. This was the fundamental principle guiding the treatment decisions, with a fallback plan of reconstruction with a Gore-Tex graft (Gore & Associates, Flagstaff, AZ) if in situ reconstruction using the patient’s own aortic tissue was not possible. It was also apparent that, at a minimum, the supr-
Surgery

The patient was taken to surgery on the day of admission. The presence of a functioning axillofemoral bypass was helpful for leg and pelvic perfusion. The aneurysm was approached through a thoracoabdominal extraperitoneal incision at the eighth intercostal space, with incision of the diaphragm through the aortic hiatus. This allowed control of the supraceliac aorta, which had a much smaller diameter than the visceral and renal aorta and total exposure of the visceral and renal arteries. As a part of our spinal cord protection protocol, the patient was passively cooled to 34°C before cross-clamping the aorta and was given 30 mg/kg of methylprednisolone and low-dose naloxone intravenously. We also use thiopental to achieve EEG-monitored burst suppression and keep the mean arterial pressure at 85 mm Hg or greater.

The aorta was cross-clamped above the celiac artery, and the aneurysmal and visceral aorta was opened. The aortic debris, which consisted of infected old and new thrombus, was radically debrided to vascularized adventitia or peri-aortic tissue. This infected material was sent for culture and gram stain. Fogarty balloon catheters (Edwards Lifesiences, Irvine, CA) were then used to occlude the celiac and superior mesenteric artery, and the left renal artery was perfused with 500 mL of cold (4°C) renal perfusion solution (lactated Ringer’s with 1,000 units of heparin and 25 gm of mannitol/L). The right renal artery was also perfused with 200 mL after excising the dissecting septum. This renal perfusion further cooled the patient to 31°C for additional hypothermic end-organ protection (renal, liver, and spinal cord).

The visceral and renal aorta was then reconstructed by imbricating the wall to decrease the overall aortic diameter (and thus reduce wall tension) as shown in Figure 3A and 3B. The aorta below the renal level was unsuitable for this reconstruction.

When closing, a 10-flat Jackson-Pratt drain attached to a Reliavac suction was left in the area of aortic debridement.

Recovery

The first 36 hours after surgery required pressor support with vasopressin and epinephrine, but the patient did not bleed, and her renal function remained stable, not requiring dialysis. She was extubated 48 hours after surgery and was maintained on intravenous hyperalimentation for 10 days and then enteral tube feedings for 3 weeks to supplement oral intake because of her severe malnutrition. Her intraoperative cultures grew MRSA, and she was maintained on intravenous antibiotics for 6 weeks. This lengthy antibiotic course was administered partially because of how long it took her inflammatory markers to decline (Figure 4), which correlated with improvement in her CT scan (Figure 5). CT scans also showed the return of body fat, indicating recovery from her severe malnutrition.

This patient returned to her job after 4 months and has lived a normal life for the last 7 years on metoprolol and losartan for blood pressure and occasional monitoring of her aortic diameter and axillofemoral bypass. Her aortic diameter has slowly increased and is now almost 5 cm in diameter just distal to the subclavian, but the visceral aorta remains < 3 cm. Her renal function has continued...
to improve with a creatinine level now of 1.1 mg/dL and eGFR of 51.

**DISCUSSION**

Clearly, this case is unique and demonstrates a solution that is unusual. It does show that the aortic wall, even in relatively recent dissection, is strong enough for reconstruction. This may not be the case, however, in patients with Ehlers-Danlos syndrome or Loeys-Dietz syndrome,1,2 when aortic wall integrity can be questionable even without dissection. In patients with Marfan syndrome, this fragility is less worrisome in our experience.

We have treated 21 patients with mycotic aneurysms of the thoracic or thoracoabdominal aorta, and (except in this case) in situ replacement was performed with Gore-Tex grafts (Table 1). None of the clostridia patients survived, and *Staphylococcus aureus* recurred in one patient who died. The salmonella patients were left on life-long suppressive therapy, and there were no recurrences.

The primary principle we followed in this case, as in all cases, was radical local debridement of all devitalized and infected tissue and inert plaque.3 We do not use assisted circulation in thoracoabdominal replacement; we do use moderate hypothermia, spinal fluid drainage, naloxone, and steroids for spinal cord protection, as well as cold renal perfusion for renal protection and to lower core temperatures to 32°C.4,5 The protective effect of profoundly cooling the kidney is dramatically demonstrated in this patient with impaired renal function, which was completely preserved.

Because of her age and relative good health, this patient will eventually need to have her aorta replaced as it becomes more aneurysmal, making follow-up essential at reasonable time intervals depending on the growth rate.

Charles Acher, MD, is with the Division of Vascular Surgery at the University of Wisconsin in Madison, Wisconsin. He has disclosed that he has no financial interests related to this article. Dr. Acher may be reached at (608) 265-4420.

Martha Wynn, MD, is with the Department of Anesthesiology at the University of Wisconsin in Madison, Wisconsin. She has disclosed that she has no financial interests related to this article.


**TABLE 1. OVERVIEW OF PATIENTS WITH THORACIC OR THORACOABDOMINAL MYCOTIC ANEURYSMS**

<table>
<thead>
<tr>
<th>21 Mycotic TAAs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, 66 y</td>
</tr>
<tr>
<td>12 men</td>
</tr>
<tr>
<td>Symptoms</td>
</tr>
<tr>
<td>Back pain: 76%</td>
</tr>
<tr>
<td>Fever: 88%</td>
</tr>
<tr>
<td>Malaise: 70%</td>
</tr>
<tr>
<td>Leukocytosis &gt; 10,000: 76%</td>
</tr>
<tr>
<td>Average symptom time: 3 weeks</td>
</tr>
<tr>
<td>Blood cultures positive: 54%</td>
</tr>
<tr>
<td>Arterial wall cultures positive: 88%</td>
</tr>
<tr>
<td>Gram stain of aortic wall positive: 82%</td>
</tr>
<tr>
<td>CT and angio diagnosis</td>
</tr>
<tr>
<td>Multiple aneurysms: 48%</td>
</tr>
</tbody>
</table>

**Bacteriology**

- 82% gram + organisms
  - 10 *Streptococcus*
  - Group B, pneumonia, viridans
    - 1 *Enterococcus*
    - 2 salmonella
    - 3 *Staphylococcus aureus*
  - 1 MRSA
  - 1 *Bacteroides fragilis*
  - 1 *Clostridium septicum*
  - 1 *Clostridium perfringens*
  - 1 no growth but + gram stain

**Treatment**

- Aggressive aortic debridement and in situ replacement with Gore-Tex
- 6 to 12 weeks of intravenous antibiotics depending on organism
- Postoperative imaging surveillance and sedimentation rate, C-reactive protein, and WBC for 2 years

**Results**

- 3 (15%) died in hospital
- 1 (5%) died at 4 months from recurrent infection
- 1 died at 31 months of myocardial infarction
- 85% alive at 1 year
- 65% alive at 5 years