CASE REPORT

Rapidly Progressing Aneurysm of Infected Thoracic Aorta with Pseudoaneurysm Formation

Akio Iimori 1, Yumiko Kanzaki 1, Shinpei Ito 2, Takuya Kotani 3, Suzue Hirano-Kuwata 4, Masahiro Daimon 1, Takahiro Katsumata 1, Hiroyuki Akagi 1, Tsuyoshi Komori 5, Fumio Terasaki 1, Nobukazu Ishizaka 1 and Akira Ukimura 2

Abstract

A 60-year-old man presented with chest discomfort with fever and high C-reactive protein (CRP). Chest computed tomography (CT) disclosed a mediastinal soft tissue swelling originating from the aortic arch, and gallium-67 single-photon emission CT revealed intense uptake in the same region. We initially suspected mediastinitis and/or a thoracic aortic infection. Antibiotics improved his symptoms and CRP levels. However, a follow-up CT scan 33 days later, revealed an aortic arch aneurysm and the patient was diagnosed with infective aortic aneurysm. Here, we report a rare case of a rapidly progressing aneurysm of infected aorta aortic infection with pseudoaneurysm formation.

Key words: infected aneurysm, gallium scintigraphy, computed tomography, SPECT/CT


DOI: 10.2169/internalmedicine.49.4068)

Introduction

Infection of the aorta is rare and difficult to treat, and it is associated with significant mortality. The key to survival is early diagnosis followed by intensive treatment with appropriate antibiotics and prompt surgical intervention. An immediate diagnosis should be pursued based on computed tomography (CT). Fusion images obtained from CT and gallium-67 citrate (Ga-67) single-photon emission CT (SPECT) are also useful in confirming the location of an infected aorta. Here, we present a rare case of a rapidly progressing aneurysm of infected thoracic aorta associated with pseudoaneurysm formation.

Case Report

A 60-year-old man with diabetes mellitus, hypertension, and a history of radiation therapy for laryngeal cancer was admitted initially to another hospital because of chest pain, high fever of unknown origin, and high C-reactive protein (CRP) concentration. Despite a few days of intravenous antibiotic treatment for the bacterial infection with unknown foci, the fever and CRP concentration remained unchanged. Owing to persistent fever, the patient was referred to our institution for further evaluation and treatment. On admission, he complained of having had chest discomfort during the preceding week. On initial examination, his body temperature was 38.3 °C, blood pressure was 166/87 mmHg, his heart rate was 110 beats/min, and respiratory rate was 28 breaths/min. The oxygen saturation was 98%. Physical examinations revealed no remarkable signs. A chest radiograph revealed a mild widening of the mediastinum (Fig. 1) and an electrocardiogram showed sinus tachycardia and left ventricular hypertrophy. Laboratory studies, summarized in Table 1, revealed leukocytosis with a white blood cell (WBC) count of 13,390 cells/μL (91% neutrophils, 3% lymphocytes, and 4% monocytes) and a CRP concentration of 30.7 mg/L. Chest CT disclosed a mediastinal soft tissue swelling originating from the aortic arch with upper mediastinitis.

1Department of Internal Medicine III, Osaka Medical College, Takatsuki. 2Department of General Medicine, Osaka Medical College, Takatsuki. 3The First Department of Internal Medicine, Osaka Medical College, Takatsuki. 4Department of Cardiovascular Surgery, Osaka Medical College, Takatsuki and 5Department of Radiology, Osaka Medical College, Takatsuki

Received for publication June 11, 2010; Accepted for publication August 20, 2010
Correspondence to Dr. Yumiko Kanzaki, in3089@poh.osaka-med.ac.jp
(Fig. 2A). We suspected mediastinitis and/or thoracic aortic infection as an inflammatory cause. A transthoracic echocardiogram revealed no evidence of infective endocarditis and a gastroscopy revealed no particular finding of mediastinitis. Empirical treatment with ceftriaxone 2 g/day and vancomycin (VCM) 1 g/day and strict control of serum blood sugar level were initiated parenterally. Ga-67 scintigraphy showed abnormal accumulation at the same site. We also performed a Ga-67 SPECT and fused the images with his chest CT images (Fig. 3), which supported the notion that this region was the source of the inflammation. The infectious parameters gradually improved after a few days, and the patient’s symptoms also showed improvement. On day 14 of hospitalization, a follow-up chest CT showed that the swelling of the aortic arch had reduced (Fig. 2B). We continued the antibiotics and on day 30, the patient complained of slight back pain. On day 33, follow-up chest CT showed an aortic arch aneurysm that had developed rapidly (Fig. 2C). In addition, magnetic resonance imaging (MRI) showed a saccular aneurysm at the aortic arch (Fig. 4), therefore the patient was diagnosed to have infective aortic arch aneurysm. We tried to conduct strict blood pressure control and continued the antibiotic therapy. We also changed VCM to linezolid as it is thought to have better tissue distribution and is more suitable for patients with renal dysfunction. However, on day 40, another follow-up chest CT revealed the progressive enlargement of the aortic arch aneurysm compared with that observed one week previously (Fig. 2D). We decided to perform immediate surgery when the signs of impending rupture were proven on CT. A preoperative aorta-coronary angiogram showed normal coronary vessels. Thereafter, on day 50, partial aortic arch replacement was performed (Fig. 5). The aneurysm was saccular in shape and had a punched-out orifice, a very thin wall, and a thick mural thrombus, therefore we diagnosed this saccular aneurysm was pseudoaneurysm. During surgery, the infected aortic tissue was debrided, and the infected area was found to be more widespread than that indicated by CT imaging. In addition, a mycotic pseudoaneurysm was also noted in the arch region, because a descending aorta-to-aortic arch prosthetic interposition graft was implanted and connected to the innominate artery smoothly. The patient’s blood cultures and resected tissue cultures tested thereafter were negative. A pathological exam revealed an atheroma that had been deposited by cholesterin on an aortic wall. The adventitial site exhibited fibrosis, hyperplasty of small vessels, and neutrophil infiltration, but no bacterial colony was apparent.

The postoperative course was uneventful. After surgery, antibiotics were administered with ampicillin sodium/sulbactam sodium 3 g/day and linezolid 1,200 mg/day for 3 weeks intravenously, and then linezolid 600 mg/day for 3 weeks orally. The patient’s overall condition remained good and he was discharged 44 days after the surgery. He has remained in a stable condition for a year after surgery.

**Discussion**

Here, we present a case of rapidly progressing aneurysm of infected thoracic aorta with pseudoaneurysm formation, which resolved after intensive antibiotic therapy and surgical therapy. Infected aorta is an uncommon though potentially devastating condition with only 0.7-2.7% of all aortic aneu-

---

**Table 1.** Laboratory Findings on Admission

<table>
<thead>
<tr>
<th>Hematology</th>
<th>Biochemistry</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC 13390 cells/μL</td>
<td>AST 65 IU/L</td>
</tr>
<tr>
<td>RBC 459 ×10⁶ cells/μL</td>
<td>ALT 70 IU/L</td>
</tr>
<tr>
<td>Hb 13.5 g/dL</td>
<td>LDH 213 IU/L</td>
</tr>
<tr>
<td>Ht 38 %</td>
<td>CK 28 IU/L</td>
</tr>
<tr>
<td>Plat 34 ×10⁶ platelets/μL</td>
<td>BS 335 mg/dL</td>
</tr>
<tr>
<td>Blood culture (-)</td>
<td>CRP 30.7 mg/dL</td>
</tr>
<tr>
<td></td>
<td>Cm 0.9 mg/dL</td>
</tr>
<tr>
<td></td>
<td>BUN 14 mg/dL</td>
</tr>
<tr>
<td></td>
<td>UA 14 mg/dL</td>
</tr>
<tr>
<td></td>
<td>T-Chol 211 mg/dL</td>
</tr>
<tr>
<td></td>
<td>Na 132 mEq/L</td>
</tr>
<tr>
<td></td>
<td>K 4.2 mEq/L</td>
</tr>
<tr>
<td></td>
<td>Cl 94 mEq/L</td>
</tr>
<tr>
<td></td>
<td>HbA1C 11.3 %</td>
</tr>
</tbody>
</table>
The course of disease as shown through changes in chest axial computed tomography (CT) scans. A: On admission, the CT image showed visible mediastinal soft tissue swelling originating from the aortic arch with mediastinitis (white arrow). B: Day 14, the swelling of the aortic arch appears to have improved. C: Day 33, a pseudoaneurysm with periaortic infiltration (white arrow) visible in the upper mediastinum. D: Day 40, the aortic arch aneurysm was more dilated than the previous day (white arrow).

Ga-67 and CT fusion imaging. A. Ga-67 image shows abnormal accumulation at the upper mediastinum. (black arrow) B. Ga-67 SPECT and CT (Fig. 2A) fusion image of the infected aorta (red region).

Chest longitudinal magnetic resonance imaging on day 34. The image clearly shows a saccular aneurysm in the aortic arch.

Aneurysms being of the mycotic type (1-4); the incidence of mycotic aneurysms in the aortic arch is even lower (5). Infected aorta is also notoriously difficult to diagnose, and its detection requires strict attention to the patient’s symptoms. It can be caused by the following: 1) formation of mycotic aneurysms secondary to septic microemboli in the vasa vasorum; 2) extension from a contiguous infected focus; 3) hematogenous seeding of the intima during bacteremia originating from a distant infection; and 4) direct contamination following trauma to the arterial wall (6, 7). Most infected atherosclerotic aneurysms occur in elderly men. A large percentage of patients have underlying diseases or
other relevant factors that increase their risk of the development of arteriosclerosis. These disease or factors include diabetes mellitus, hypertension, heavy smoking, or long-term corticosteroid therapy (2, 4, 7, 8). In general, the arterial intima is very resistant to infection; however, if this intimal lining is altered by atherosclerotic plaques or ulcers, resistance of arterial intima to infection is lowered, and its surface may become colonized by blood-borne organisms. Secondary infection of a pre-existing aneurysm occurs most commonly in the abdominal aorta, the site of 70% of such cases, because this portion of the aorta is most frequently and severely damaged by atherosclerosis. Both ascending and descending aortic aneurysms individually account for about 15% of secondary infection cases (9). When primary bacteremia is the cause, it most commonly originates from a distal infection in soft tissue or an infection in the lung, bone, or joint tissue. At the site of a preexisting aneurysm, the wall of the aorta is thinned and can be further weakened by focal acute and chronic inflammation; therefore, this site is as likely to develop into an infected pseudoaneurysm, as is its propensity to rupture (2-7, 10).

Approximately 60%, of infected aortic aneurysms are caused when a previously atherosclerotic vessel is colonized by gram-positive organisms, while 35% are caused by gram-negative bacilli colonization, especially salmonellae. Staphylococci are implicated in 40% of cases overall; more than two-thirds of which involve Staphylococcus aureus (1-4, 6, 9). In the present patient, we suspected colonization by methicillin-resistant Staphylococcus aureus, because VCM and linezolid were effective but CTRX was not. Patients with infected aortic aneurysms usually also have leukocytosis and increased CRP, but these associated conditions are nonspecific. Occult-infected aneurysms have also been identified in patients with fever of unknown origin. CT, MRI, and Ga-67 scintigraphy are modalities that have been used to localize intra-arterial infections (11-14). CT conveys anatomical information and SPECT reveals functional changes; these two modalities may be used complementarily. Thus, a Ga-67 SPECT/CT fusion image was quite useful in establishing the diagnosis for this patient. Our diagnosis of infected thoracic aorta was based on the patient’s fever and elevated inflammatory signs along with the Ga-67 SPECT/CT fusion image. As infected aortic aneurysms usually develop rapidly even while the patients’ inflammatory signs are improving, follow-up CTs are very important to determine whether an aneurysm is forming (2, 4). The possible routes of pathogenesis and the speed of growth in mycotic aneurysm remain unclear (1, 2). In the present case, a severe atherosclerotic plaque was seen on the inner surface of the aorta during the operation, which was much more than that was extensive than that revealed by CT imaging. We conjecture that sclerotic plaque became infected when the bacteremia developed and the aortic wall was subsequently ruptured by the infection (1), and also that the pseudoneurysm had developed rapidly even though infection was controlled. It thus follows that, one should not rely on the results of imaging alone for the understanding and treatment of infected aortic aneurysms.

Infected aortic aneurysms usually require treatment with both intravenous antibiotics, and surgical excision, because antibiotic therapy alone is usually insufficient. The mortality rate among patients with infected aortic aneurysms treated with antibiotics alone has been reported at 16% - 44% (5, 15). The critical points in the management of infected aortic aneurysm are the optimal timing of surgical procedure and the antibiotic strategy. The incidence of vascular complications was shown to be clearly associated with ruptured aneurysm and extensive periaortic infection at the

Figure 5. Clinical course of the patient. CTRX: ceftriaxone, VCM: vancomycin, LDN: linezolid, WBC: white blood cell count, CRP: C-reactive protein, Crn: Creatinine, BUN: Blood urea nitrogen.
time of surgery (1, 2). Thus, complete excision of the affected aorta is the key to curative treatment (5, 16). Cases of successful endoluminal treatment of mycotic aortic aneurysms have also been reported (17, 18). In general, surgical procedures are associated with substantial mortality rates as well as a risk of recurrent infection although there are several exceptions (16, 19). After surgery, survival is primarily influenced not by the type of reconstruction but by the status of aneurysmal rupture; about 30-50% of aneurysms are reported to rupture (1, 2, 5, 16). The outcome is worse when the aneurysm has already ruptured or penetrated, and when it penetrated leading to formation of pseudoaneurysm. The required length of the follow-up period is not well established; recommendations range from 6-12 weeks to lifelong (1, 2, 4, 6).

In conclusion, increased clinical awareness of this condition among practitioners, immediate and follow-up CT evaluation, and prompt surgical intervention under appropriate and intensive antibiotic therapy appears to offer the best chance of survival for patients with infected aortic aneurysm.

References