Purulent Pericarditis Due to Group B Streptococcus and Mycotic Aneurysm of the Ascending Aorta

— Case Report —

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A 61-year-old female, with a history of uterine and cervical cancer treated with radical hysterectomy and 2 years of postoperative chemotherapy, presented to the emergency department with dyspnea on exertion. Computed tomography of the chest revealed a large pericardial effusion and a sacciform aneurysm of the ascending aorta. The patient subsequently underwent emergency pericardiocentesis with drainage of approximately 330 ml of a bloody and turbid effusion. Cultures from the effusion yielded group B streptococcus. Multiple organ failure and disseminated intravascular coagulation syndrome occurred in the acute phase, but gradually improved with continuous antibiotic therapy. On the 194th hospital day, in situ reconstruction of the ascending aorta was successfully performed using a synthetic graft. Although rarely reported, both purulent bacterial pericarditis and mycotic aneurysm can be life-threatening. (*Jpn Circ J* 2000; 64: 83–86)

Key Words: Cardiac tamponade; Group B streptococcus; Mycotic aneurysm; Purulent pericarditis

We report a case of purulent pericarditis due to group B streptococcus (GBS) along with a mycotic aneurysm of the ascending aorta. Both purulent pericarditis caused by GBS and mycotic aneurysm of the ascending aorta are extremely rare, with a thorough survey of the literature revealing only one other case of the former and 43 cases of the latter. We discuss here the pathogenesis of both entities, and suggest that in the present case both diseases occurred simultaneously as a result of hematogenous infection, even though the source of the bacteremia remains unclear.

Case Report

A 61-year-old female presented to the emergency department with dyspnea on exertion. She had undergone radical hysterectomy for uterine and cervical cancer 3 years earlier, with 2 years of postoperative chemotherapy with mitomycin C, etoposide, and cisplatin. One month prior to presentation, she experienced flu-like symptoms and developed progressive dyspnea. On admission she had a clear sensorium and cold extremities. Physical examination revealed a blood pressure of 100/80 mmHg, a temperature of 36.1°C, and tachypnea at 30 breaths/min. Cardiac exami-

Fig 1. Electrocardiogram showing tachycardia and low voltage in the limb leads.

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nation revealed a regular tachycardia at 180 beats/min, and extra heart sounds were not auscultated.

Laboratory tests revealed the following values: blood gas analysis breathing room air, pH 7.170, PaCO₂ 7.5 mmHg,

Fig 2. Chest radiograph showing mild cardiomegaly and plate-like atelectasis of both lower lung fields.

PaO₂ 110.1 mmHg, base excess -25.5 mmol/L; leukocyte count $14.7 \times 10^3/\mu\text{l}$ with significant shift to the left; hemoglobin 13.2 g/dl; platelets $22.5 \times 10^4/\mu\text{l}$; fibrinogen 674 mg/dl; C-reactive protein 26.7 mg/dl; and erythrocyte sedimentation rate 41 mm at the first hour. Electrocardiography revealed sinus tachycardia at 180 beats/min and low voltage in the limb leads (Fig 1). Chest radiography showed mild cardiomegaly and plate-like atelectasis in both lower lung fields (Fig 2). Echocardiography revealed a large pericardial effusion, and excluded the presence of valvular dysfunction or vegetation. Computed tomography of the chest revealed a thickened pericardial membrane, the large pericardial effusion, bilateral pleural effusions, and an irregularly enhanced sacciform aneurysm of the ascending aorta (Figs 3, 4).

Clinical Course

Cardiac tamponade developed soon after chest imaging, requiring emergency pericardiocentesis with ultrasound guidance. Open drainage of the pericardium was performed to remove the large, bloody and turbid effusion. Approximately 330 ml drained within 2 days. However, persistent hypotension was followed by multiple organ failure and disseminated intravascular coagulation syndrome. The patient underwent continuous hemodialysis and ultra filtration with anticoagulant therapy.

After the pericardial drainage, an intravenous drip of vancomycin hydrochloride (VCM) and aztreonam (AZT)

Fig 3. Computed tomography of the chest revealed a thickened pericardial membrane, a large amount of pericardial effusion, and bilateral pleural effusion.

Fig 4. Computed tomography of the chest performed on the first hospital day shows an irregularly enhanced sacciform aneurysm of the ascending aorta.

Fig 5. Follow-up computed tomography (CT) of the chest performed on (A) hospital day 42 and (B) hospital day 105, showing gradual development of the mycotic aneurysm of the ascending aorta when compared with the CT performed on the first hospital day (Fig 4).

was administered together with an intramuscular injection of streptomycin sulfate (SM). Within 5 days, group B streptococcus (GBS) was isolated from the pericardial culture, and the antimicrobial therapy was changed to penicillin G (24 million units/day) and clindamycin (1200 mg/day) in accordance with the culture sensitivities. Antibiotic therapy was continued for 3 weeks, during which time the patient was afebrile with a temperature of 36.8°C, and the leukocyte count and the C-reactive protein had decreased to $3.9 \times 10^3/\mu\text{l}$ and 4.9 mg/dl, respectively. The effusion cultures did not yield any malignant cells or organisms other than GBS. Local recurrence and systemic metastasis were excluded after examination for serum tumor marker, vaginal cytology, computed tomography of the abdomen, and gallium scintigraphy.

The mycotic aneurysm of the ascending aorta seemed to develop gradually on review of serial computed tomography of the chest (Fig 5). Enhancement of the aneurysm was increased due to a filling defect in the aneurysm, most likely representing a thrombus or bacterial plaque, which slowly disappeared after therapy with antibiotics and anti-coagulants.

The patient's condition slowly improved, and on hospital day 194, she underwent in situ reconstruction of the ascending aorta using a synthetic graft. We found mild aortic atherosclerosis and a multilocular, sacciform aneurysm of the aorta arising anteriorly near the brachiocephalic trunk compressing the pulmonary artery. The aneurysmal neck was 3 cm in diameter, and the maximal diameter of the aneurysm was 5×4 cm. The portion of aorta containing the aneurysm was resected, and a synthetic aortic graft was placed. The interior wall of the aneurysm was free of colonized bacteria and covered with a thin thrombotic layer. Microscopic examination of the aneurysmal sac revealed that the intima was completely destroyed, with only slight remnants of the elastic fibers. Bacterial cultures collected during the operation were negative. The ascending aorta reconstruction was a great success, and the patient recovered quickly without any complications.

Discussion

We reviewed the literature on cases of purulent pericarditis and mycotic aneurysm reported from 1989 to 1998. One case only of purulent pericarditis due to GBS¹ and 43 cases of mycotic aneurysm of the ascending aorta² were found. We did find 1 other case of concomitant purulent pericarditis and mycotic aneurysm; however, the causative organism was *Staphylococcus aureus*.³

Purulent pericarditis was associated with a high fatality rate prior to the antibiotic era⁴ but now, with the widespread use of antibiotics, the mortality has decreased markedly, although the pathogenic bacteria have diversified, and the routes of infection have changed.^{5,6} Previously, Gram-positive cocci, especially *Streptococcus pneumoniae* and *S. aureus*, were common⁷ and direct transmission from intrathoracic disease, such as pneumonia, and empyema was often seen in infants and children.⁸ Today, purulent pericarditis due to Gram-negative bacteria, such as *Escherichia coli*, *Klebsiella pneumoniae*, *Proteus mirabilis*, and *Pseudomonas aeruginosa*, has been increasing, whereas *Salmonella* and *Listeria meningitis* and fungal infections are found less often.^{5,6} The route of infection has changed from infectious endocarditis, thoracic trauma or the infection of organs adjacent of the diaphragm to hematogenous

transmission. Purulent pericarditis is now more prevalent in adults, because modern medicine is able to maintain patients who are immunodeficient for a variety of reasons.^{9,10}

GBS is a rare cause of purulent pericarditis and is more commonly associated with skin infection, endocarditis, puerperal infection, neonatal septicemia, and meningitis.¹ In the present case GBS was definitively identified from cultures of the pericardial effusion, but not from other cultures obtained from the vagina and blood. The patient had had flu-like symptoms with a refractory cough and fever quite some time before her condition changed greatly, so it is most likely that the infection originated from the upper respiratory tract and eventually extended to the pericardium through bacteremia.

A mycotic aneurysm is a localized, abnormal dilatation of the infected arterial wall, and the progressive expansion eventually destroys the aneurysmal wall.^{11,12} Depending on the location, it often progresses undiagnosed, resulting in recurrent bacteremia from an unidentified source. The aneurysm eventually ruptures, causing cardiovascular collapse for which subsequent emergency surgery is often unsuccessful.¹³⁻¹⁵ The pathogenesis of mycotic aneurysm has changed significantly since it was first described by Sir William Osler in 1885. Accordingly, the clinical spectrum of the disease has expanded over the past century,¹⁶ and today all aneurysms with an infectious component are called mycotic aneurysms.

The pathogenesis of mycotic aneurysms can be divided into 4 mechanisms.¹⁷

- (1) Septic microemboli to the bifurcation points of smaller vessels in the vasa vasorum of larger vessels (the mechanism of mycotic aneurysms associated with endocarditis).
- (2) Local extension of an infected focus.
- (3) Direct trauma with contamination (ie, accidental, self-induced, or iatrogenic).
- (4) Hematogenous seeding from a remote focus.

Those arising from the latter mechanism are also called 'primary mycotic aneurysms' and appear in the absence of local infection or an intravascular focus such as infectious endocarditis.¹⁸ They may occur when circulating bacteria from a remote source lodge on abnormal intima in an atherosclerotic or aneurysmal vessel. The requirement for atherosclerosis supports the hypothesis that thoracic and abdominal aorta are the most frequent targets.¹⁷ In many cases of primary mycotic aneurysm, the source of bacteremia remains unknown. Blackett et al reported a case of mycotic aneurysm of the aorta due to group B streptococcus in which the source of the bacteremia was not identified.¹⁹ In the present case, the aneurysm of the ascending aorta showed a solitary trace of septic embolism and the aneurysm most likely resulted from hematogenous seeding of the aortic wall with pre-existing intimal damage during a bacteremia from a distant focus.

Regarding the successful in situ reconstruction of the ascending aorta with a synthetic graft, we found several reports of operations in which allograft material was used for emergent surgery.²⁰⁻²² We elected to operate in the chronic phase for 3 reasons: (1) the patient's condition in the acute phase was tenuous due to the uncontrolled infection, (2) we could not obtain allograft material within 2 weeks, and (3) we expected that infection to an urgently reconstructed vessel would almost certainly occur and would be even worse with a synthetic vessel.

The patient was discharged from hospital 1 month after

surgery. She remains alive and without any complications 16 months later.

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