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A Case of Recurrent Aortic Rupture Associated with *Klebsiella pneumoniae* Pericarditis Treated by Two Separate Aortic Operations

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A 49-year-old female presented with severe dyspnea. She was diagnosed with cardiac tamponade combined with ascending aortic pseudoaneurysm and rupture, which was caused by *Klebsiella pneumoniae* infection. This extremely rare condition was managed by an emergency pericardiostomy and two separate aortic operations. Antibiotics active for the *K. pneumoniae* isolate were used throughout. The patient was well for nine months after discharge and continues to be followed up for signs of possible reinfection.

Key words: 1. *Klebsiella pneumoniae*
2. Aortic aneurysm
3. Infection

CASE REPORT

A 49-year-old female was transferred to emergency department of Dankook University Hospital due to severe dyspnea. On initial physical examination, hypotension (84/57 mmHg), tachycardia (130 beats/min), and tachypnea (32 breaths/min) were observed. The patient had a history of severe alcoholism and was a heavy smoker, and had poorly controlled diabetes and hypertension. Computed tomography (CT) scan of the chest performed at another facility just before transfer to our hospital revealed massive pericardial fluid and air bubbles in the pericardial space (Fig. 1). Laboratory data showed leukocytosis (24,900/ μL) and elevated C-reactive protein (11.10 mg/dL). Cardiac tamponade caused by infectious pericarditis was presumed. Emergent pericardiostomy was performed through a subxiphoid incision under general anesthesia. Yellowish and turbid fluid (500 mL) was drained initially. Empirical antibiotic therapy with vancomycin+piperacillin and tazobactam was maintained postoperatively. After the surgery, dyspnea was relieved but the mediastinal widening persisted on postoperative chest X-ray. Chest CT was performed to clarify the cause. Surprisingly, a 2-cm pseudoaneurysm was revealed at the distal ascending aorta, with a surrounding hematoma (Fig. 2A). This lesion was regarded as an impending rupture, so re-exploration of the pericardial space and aortic repair were performed on the fifth hospital day. The left femoral artery was exposed and cannulated for cardiopulmonary bypass (CPB), and this was followed by a median sternotomy. Upon opening, the pericardium was found to be thickened due to chronic inflammation, with the pericardial space filled with a mixture of heavily organized necrotizing materials and hematoma (Fig. 2B). Aggressive decortication of the pericardium was performed. A 2-cm-sized aortic perforation was found at the distal ascending aorta near

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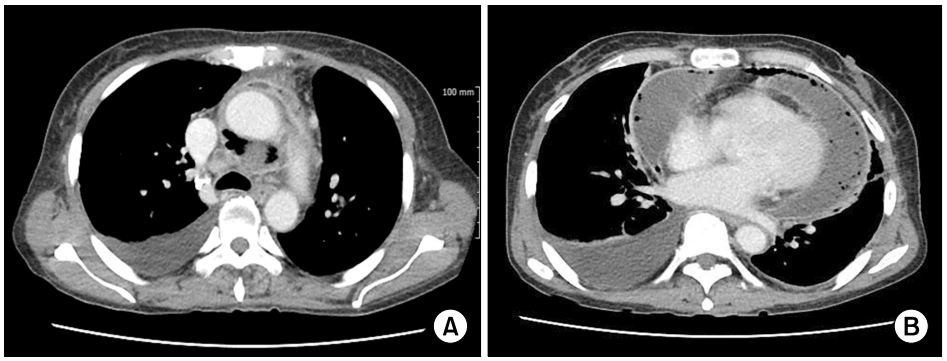

Fig. 1. (A, B) Preoperative computed tomography images show a large pericardial effusion contained with air bubbles and an intact ascending aorta. This image was taken at another facility just before transfer to Dankook University Hospital.

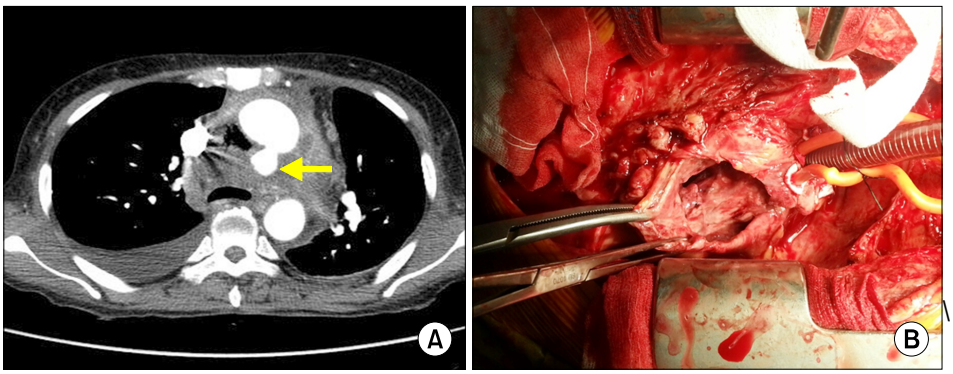

Fig. 2. (A) A computed tomography image on the fifth hospital day shows a 2-cm pseudoaneurysmal sac at the distal ascending aorta (arrow). (B) A photograph taken during the second operation shows thick and purulent pericardium containing heavily organized necrotizing materials and hematomas.

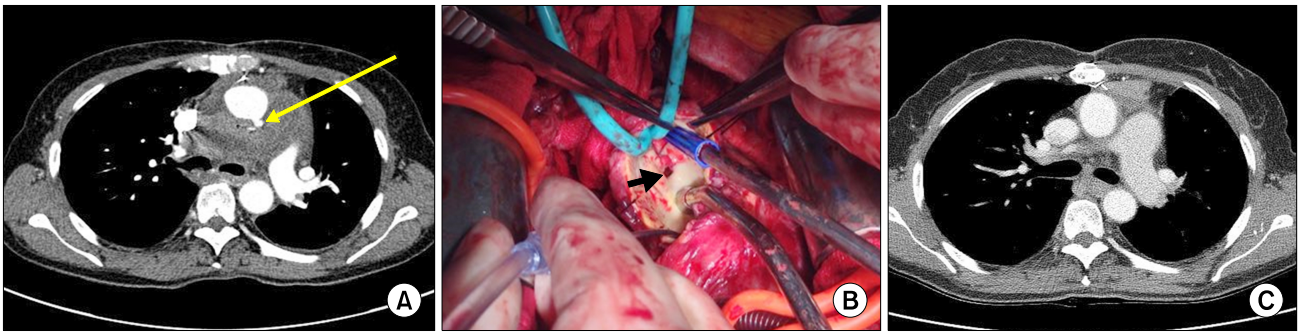

Fig. 3. (A) A CT image two months after the second operation shows a recurrent pseudoaneurysmal sac at the repaired site (arrow). (B) A photograph taken during the last operation shows an opening of the pseudoaneurysmal sac (arrow). (C) A postoperative CT image six months after the last operation shows the repaired aorta. CT, computed tomography.

the lesser curvature. The hole was occluded using a Foley catheter. Venous cannulation was performed through the right atrial appendage and CPB was started. Deep hypothermia was induced to a body temperature of 21°C and CPB was then stopped. The aortic defect was repaired by a horizontal mattress suture technique with two-pledget reinforced 4-0 polypropylene sutures, and CPB was restarted. CPB weaning was done uneventfully. The postoperative course was smooth with stable vital signs. *Klebsiella pneumoniae* was isolated on culture of pericardial tissues obtained during the pericardiostomy. Antibiotic therapy was changed to ceftriaxone, to which the isolate was more sensitive, and maintained for three weeks. The patient was discharged with a prescription of oral antibiotics.

Two months later, the patient was readmitted with fever and chest pain. Chest CT revealed an extravasation from the

previous aortic repair site (Fig. 3A). A second aortic operation was performed. The left femoral artery and vein were exposed and cannulated for partial CPB during the resternotomy. The aortic defect was identified at the previous repair site. The lesion was unable to be repaired as it was in the first surgery, and instead required replacement of the ascending aorta. An additional venous cannula was inserted through the right atrial appendage, and deep hypothermia to 20°C was induced. CPB was stopped, and the ascending aorta was replaced by a 26 mm Hemashield Platinum (Maquet Cardiovascular, Fairfield, NJ, USA) after all infected aortic tissues were resected. During the stopped CPB, bilateral antegrade cerebral perfusion was maintained. An aortic perforation was observed when the aorta was opened (Fig. 3B), and the previous suture materials were detached from the aortic wall. The lesion was considered to be a result of the *Klebsiella* infection. CPB weaning was uneventful. Postoperative ventilator support was needed for four days due to sepsis despite ceftriaxone prophylaxis. Bacteremia was confirmed, with *K. pneumoniae* as the source. The antibiotic regimen was changed to ciprofloxacin and imipenem, to which the latest isolate was most sensitive. Subsequently, the septic condition was markedly controlled. The intravenous regimen was prolonged for six weeks after the last operation. The patient was discharged without complications and was well, without evidence of reinfection, nine months later. The most recent CT evaluation occurred six months after the last operation (Fig. 3C).

DISCUSSION

Aortic aneurysms caused by bacterial infections are rare. They comprise only 0.7%–1.3% of all aortic aneurysms [1]. *Klebsiella*-associated infections are extremely rare [2]. The risk of mortality and morbidity in infectious aortic aneurysm is very high, with reported rates of 10%–36% and 60%–70%, respectively [1]. These aneurysms result from a weakened aortic wall, which can easily rupture with pulse pressure and septic conditions caused by bacteremia. Thus, early diagnosis and appropriate medical and surgical treatment are essential to patient survival.

Despite the strong resistance of the aortic intima to infection, intimal infections can progress when they occur. Several mechanisms of the seeding infection into the aortic wall have been proposed. In patients with bacteremia, the organisms can seed to pre-existing atherosclerotic plaques, or septic emboli can enter into the vasa vasorum. If one or more layers of the aortic wall are damaged, the weakened wall is exposed to localized high pressure that can cause pseudoaneurysmal change. Another mechanism is direct invasion into the aortic wall from the surrounding infected tissue adjacent to the aorta; infected aneurysm combined with spondylitis or pancreatitis is a good example of this [3].

In general, the most common bacterial organisms that cause infectious aortic aneurysms are *Staphylococcus aureus* and *Salmonella* species. *Streptococcus, Pseudomonas, Candida, Cryptococcus, Aspergillus,* and *Mycobacterium tuberculosis* are other sources of infection [4]. Aortic infections from *K. pneumoniae* are rare; the few cases of infectious aortic aneurysm caused by *K. pneumoniae* are mostly reported from eastern Asia, including Japan and Taiwan [5]. *K. pneumoniae* is a gram-negative encapsulated rod. Gram-negative bacteria, including *K. pneumoniae*, have a higher tendency to expand and rupture the aortic wall than gram-positive bacteria [4]. *K. pneumoniae* is generally seen in patients with pneumonia, skin or soft tissue infection, or urinary tract infection. *Klebsiella* infection is strongly related to diabetic or immunosuppressed patients [2].

Broad-spectrum antibiotics should be initiated immediately after the diagnosis of infectious aortic disease. The antibiotics should be changed according to the results of susceptibility testing. The period of antibiotic therapy is debatable; generally, 6–8 weeks or longer of intravenous antibiotics is recommended [6]. Antibiotics should be discontinued only after careful examination for remaining infection.

In situ replacement, including patch angioplasty or graft replacement, is recommended for the treatment of ascending aorta or aortic arch infectious aneurysms. However, the decision to insert foreign materials in the infected bed is complicated by the possibility of reinfection. *In situ* replacement with an artificial graft has been reported to be successful, without recurrence or reinfection, when all possibly infected tissues were completely removed and postoperative antibiotic therapy was administered [7]. Efforts to reduce postoperative

graft infection have involved artificial grafts like rifampicin-bonded or gentamicin-soaked grafts; these efforts can be particularly useful for staphylococcal infections. Cryopreserved homograft is another alternative, as is omental flap coverage on the graft. Artificial grafts suffer from the disadvantage that antibiotics and cytokines are unable to reach surrounding tissues because of their avascular structure. Well-vascularized omental tissues can be useful for supplementation [6,8].

In our case, we chose a primary repair technique for managing the aortic perforation, because the size of the aortic perforation seemed to be controlled and the minimal use of foreign material was beneficial in an actively infectious environment. However, our initial strategy failed. While the homograft appealed as a good substitute, it was not available in our insitutition. Instead, we performed an ascending aortic replacement with Hemashield Platinum (Maquet Cardiovascular). Meticulous decortication of the pericardial space and massive irrigation with antibiotic (vancomycin) solution followed the aortic replacement. Postoperatively, adequate antibiotic coverage and effective drainage using multiple holed chest tubes were maintained.

In summary, we report an extremely rare case of aortic rupture with *Klebsiella*-related pericarditis. To our knowledge, this is the first case of aortic rupture associated with *Klebsiella* infection in Korea. The origin of the infection (whether infected aorta or pericarditis) is unknown. The latter might be more probable because chronic inflammatory changes and markedly thickened pericardium were observed in the operative field. The change of antibiotic therapy and protracted use of the more effective antibiotics were helpful in controlling the recurrent infection. The patient was covered with intravenous ciprofloxacin and imipenem for six weeks and then discharged with oral ciprofloxacin for four weeks. She was well for nine months without any further antibiotics but will need careful monitoring for possible reinfection.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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