Fulminant mediastinitis due to extended-spectrum beta-lactamase-producing *Klebsiella pneumoniae*: atypical presentation and spreading following cardiac surgery†

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Abstract

Mediastinitis due to *Klebsiella pneumoniae*, related to thoracic wall contamination after cardiac surgery, has rarely been described. We aim to report a case of fulminant mediastinitis due to extended-spectrum beta-lactamase-producing *K. pneumoniae*, secondary to a disseminated concomitant pulmonary infection. The patient remained pauci-symptomatic until clinical manifestations of sepsis acutely appeared.

Keywords: Fulminant mediastinitis • *Klebsiella pneumoniae* • Cardiac surgery • Nosocomial infection

CASE REPORT

The subject of this report was a 53-year old female with smoking and obesity as vascular risk factors. After a non-ST segment elevation Myocardial infarction acute coronary syndrome and iatrogenic right coronary artery dissection, an urgent isolated off-pump coronary artery bypass grafting with a saphenous vein to the posterior descending artery was performed. The patient was extubated without complications 12 h after surgery. The right pleura was accidentally opened during surgery, and a mediastinal right pleural drain was inserted. During the early postoperative period, she suffered a left pneumothorax requiring a pleural drainage. Intensive care unit (ICU) discharge was delayed until the seventh postoperative day due to hypoxemic respiratory failure and symptomatic atrial fibrillation, which were treated with intravenous amiodarone and vasoactive drugs. The echocardiography showed a mild pericardial effusion without haemodynamic compromise. During this period, the patient remained afebrile, and no symptoms or radiological or analytical signs of infection developed. She was readmitted in ICU 24 h later, due to a severe respiratory failure requiring orotracheal intubation and ventilatory support. The chest X-ray showed right basal parenchymatous condensation. The echocardiography showed a mild pericardial effusion. The echocardiography showed a mild pericardial effusion without haemodynamic compromise. During this period, the patient remained afebrile, and no symptoms or radiological or analytical signs of infection developed. She was readmitted in ICU 24 h later, due to a severe respiratory failure requiring orotracheal intubation and ventilatory support. The chest X-ray showed right basal parenchymatous condensation. The echocardiography revealed a moderate pericardial effusion and a haematoma in the free wall of the right ventricle with signs of cardiac tamponade. At this moment, the blood test showed a 27 740 leucocytes/mm³ count. Neither wound drainage, nor sternal instability, was identified. Therefore, the patient underwent redo surgery. Intrapericardial haematoma and pericardial effusion with purulent aspect were removed, and vacuum-assisted closure drainage was placed. Then, an empirical antibiotic treatment with meropenem, amikacin and linezolid was started. During the early post redo surgery, the patient required high doses of intravenous inotropics due to severe sepsis and haemodynamic instability. Fulminant sepsis and multiorgan failure led to death 48 h after redo surgery. Extended-spectrum beta-lactamase-producing *Klebsiella pneumoniae* was isolated in pericardial effusion cultures.

DISCUSSION

Mediastinitis remains one of the most serious complications of median sternotomy after open-heart surgery. Its incidence varies from 0.4 to 5% and, in most centers, is described as between 1 and 2% [1, 2], with an in-hospital mortality rate of 8.6–47% [2]. Diabetes, obesity, vascular disease, previous smoking, previous cardiac surgery, bilateral internal mammary arteries mobilization, chronic obstructive pulmonary disease and prolonged surgical procedures are described as risk factors for mediastinitis [1]. However, *K. pneumoniae* mediastinitis is infrequent (3–7%) [1, 3] and usually associated with secondary thoracic wall contamination.

Post-sternotomy mediastinitis is still a significant cause of morbidity and mortality after cardiac surgery. At our institution, it occurs in 1.08% of all patients, with a mortality rate of 28.3%. The overall percentage of beta-lactamase-producing *K. pneumoniae* in Spain is 5.05%, and among these cases, 68% is associated with nosocomial infections, 18% to health care and only 10% is community-acquired [4]. In our population, the most commonly isolated pathogens in mediastinitis were *Staphylococcus* (79.1%), followed by gram-negative bacteria (56.71%) and *K. pneumoniae*.

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(16.4%). From this last group, the prevalence of extended-spectrum beta-lactamase-producing *K. pneumoniae* with no secondary mortality was 81.81%. In all cases of our series, *K. pneumoniae* mediastinitis was secondary to thoracic-wall contamination and related to cutaneous colonization in patients with prolonged ICU stay. Signs of mediastinitis appeared within 2 weeks after surgery, with an average onset time of 10.5 days. The onset and clinical course of infection are usually acute, and sepsis is the most serious complication. Almost all patients experienced fever, tachycardia, chest pain or sternal instability, signs of sternal wound infection and purulent discharge from the mediastinal area. Nevertheless, our patient presented an insidious onset without fever, pain, chest drainage or sternal instability. Only when fulminant sepsis developed, signs of leucocytosis and haemodynamic instability appeared.

Some previous reports have described a solid sternum associated with a multiresistant *K. pneumoniae* infection but with the presence of wound drainage [5]. In those cases, haematogenous dissemination of *K. pneumoniae* appeared in patients with a longer ICU stay and was secondary to respiratory nosocomial infection. Our patient presented only moderate hypoxaemia, without fever or thoracic radiological anomalies, until fulminant sepsis appearance and ICU re-admission. The primary focus of infection was a right inferior lobe pneumonia, but typical clinical symptoms of mediastinitis (sternal instability and signs of sternal wound infection) were absent due to dissemination by communication between the mediastinum and the right pleural cavity, accidentally opened during the primary first surgical procedure, which contributed to direct bacterial dissemination and a fulminant sepsis without antibiotic treatment. The aggressiveness of this infection was related to the pauci-symptomatic clinical presentation, which increased the difficulty of reaching an earlier diagnosis and beginning an aggressive treatment. None of the preoperative risk factors constituted a direct cause for postoperative fulminant sepsis. A review of the English-written literature revealed no other cases of direct dissemination from the pleura to the mediastinum in a respiratory infection due to extended-spectrum beta-lactamase-producing *K. pneumoniae*.

The role of extended-spectrum beta-lactamase-producing *K. pneumoniae* in postoperative mediastinitis is increasing. The aggressiveness of sepsis due to direct dissemination from the pleural cavity to the mediastinum leads to a higher risk of mortality.

**Conflict of interest:** none declared.

**REFERENCES**


**eComment. Cardiac tamponade as a manifestation of acute mediastinitis**

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Valenzuela and colleagues [1] report a case of a 53-year-old woman with an intragenic right coronary artery dissection, who underwent an urgent off-pump coronary artery bypass grafting to the right coronary system. One week later, she was operated on for a compressive purulent pericardial effusion without signs of wound infection. Unfortunately, she died because of therapy-refractory multiorgan failure. This report highlights the difficulty in making a timely diagnosis of this particular condition.

Post-sternotomy mediastinitis is an ominous complication carrying a high risk of in-hospital mortality. An abundance of risk factors for the development of anterior mediastinitis has been identified and classified as preoperative (morbid obesity, osteoporosis, chronic obstructive pulmonary disease, diabetes, Staphylococcus-positive nasal swab, mediastinal radiotherapy and immunosuppressive intake), intraoperative (break in sterility, paramedian sternotomy, bone fracture with sternal retractor, technical mistakes in sternal closure, bilateral harvesting of thoracic arteries and prolonged operative time) and postoperative factors (prolonged time on ventilator and acute delirium syndrome) [2–4]. Despite regular use of prophylactic intravenous antibiotics and preventive measures, post-sternotomy mediastinitis occurs in a significant number of patients undergoing cardiac surgery.

The Centers for Disease Control definition for surveillance of surgical site infections identifies three categories of surgical site infection [5] superficial incisional surgical site infection, deep incisional surgical site infection and organ/space surgical site infection. According to the preceding definition, the in-hospital acquired infection of the retrosternal/mediastinal space of this patient without incisional site purulent drainage is not considered as a surgical site infection. The aggressiveness of the infection in this case resembles the course of an untreated descending necrotizing mediastinitis arising from odontogenic or cervico-facial infection. Another similarity is the dissemination of the infection from pleural space to the pericardial space through the opening of the right pleura. Infection in the setting of descending mediastinitis may spread across fascial planes, thus cross-contaminating into the anterior, middle, and posterior mediastinum and even pleural spaces.

As outlined in this case, mediastinitis remains a rare but devastating complication of open heart surgery. While prompt diagnosis and emergent surgical intervention are crucial for better outcome, the presenting symptoms can be easily confused with more common problems such as cardiac tamponade. Therefore, a high index of suspicion for mediastinitis is crucial for survival even in the absence of surgical site infection.

**Conflict of interest:** none declared.

**References**


